EFFECTS OF THERMAL FEEDBACK TRAINING AND PREHEADACHE CUE IDENTIFICATION ON MIGRAINE MANAGEMENT

BY

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A Dissertation Presented to the Faculty of Graduate Studies

University of Manitoba

In Partial Fulfillment of the Requirements for the

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Department of Psychology

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Abstract

Thermal feedback training is beginning to be adopted clinically for management of migraine headaches though mechanisms of resulting migraine improvement are unclear. The relative importance of specific and placebo effects of feedback training were investigated in this study. Three groups of migraineurs solicited through undergraduate courses were taught to either increase or stabilize skin temperature using analogue visual feedback. Twenty females and nine males participated, ranging in age from 17 to 57. A two-level procedure factor was crossed with the training factor to compare the relative effectiveness of each on control of temperature without feedback. In addition to feedback training migraineurs in one of the two increase groups were trained to identify preheadache cues. Experimenter contact time was equated for remaining migraineurs by individual "headache history" sessions. All migraineurs were instructed in the recording of headache information, and self-reported data on migraine frequency, duration, intensity and medication were compared between groups. Results showed significant differences between groups on mean skin temperature increase without feedback, with no difference between procedures. Multivariate analysis of headache data, though confounded by the failure of one increase group to demonstrate significant increases, suggested that all migraineurs improved. There were no differences between increase and stabilize groups on

the multivariate package of migraine measures. However regression analyses of each measure indicated that skin temperature was negatively correlated with two of the four measures. The effect of preheadache cue identification was not significant. This effect was inadequately evaluated due to overlap between the training period and collection of "posttraining" measures. The importance of both specific and placebo effects of feedback training for migraine are discussed.

Acknowledgements

At times during those 17 months, contingent reinforcement seemed too infrequent to maintain my efforts. Retrospectively, the process of conducting this research from conception to final defense has allowed me to integrate on my own terms our mythical scientist- practitioner ideal. Some stages of the process are memorable: the interminable obsessing and preparatory reading, the painstaking writing and rewriting of the proposal, the relief of a small crowd at the proposal oral, the headaches of finding enough migraineurs to fill all cells of the design, the desperation of instrument failure, the expressed gratitude of migraineurs from both experimental and placebo conditions, the frustration of reliance on the computer, more writing and nail biting, editing, committee work, and a last-minute run to the downtown post office to airmail a copy to my external who was vacationing in Norway.

I am grateful for the guidance of my committee, particularly David Martin and John Schallow who offered their time and encouragement before the process formally began. Dave later proved an effective committee chairman, while John was a willing resource and critic. Roger Wilson gave freely of his time and advice in spite of the clinical nature of the research. Derek Jehu, who

joined the committee at the eleventh hour, contributed willingly and tactfully. I am grateful to my external examiner, Johann Stoyva, who made the supreme sacrifice of vacation time in Europe to read and react to this research.

I also would like to thank four people who were not on my committee but who contributed to the process at some of my more troubled moments. J. G. Eales of Zoology provided me with a chart recorder after two other instruments proved inadequate (and coincidentally, chaired my final oral helpfully by limiting discussion to the prescribed 90 minutes). Lorne Sexton made several valuable suggestions, the most helpful of which allowed me to limit the design to three groups instead of four. Les Bell provided technical assistance with instrumentation, and repaired the feedback unit, thus preventing lengthy delay. My final thanks are extended to Deborah Baldwin, who helped in data collection and edited all versions of the proposal, results and discussion with adroitness. Her constant unconditional support and encouragement were crucial to my completing this research.

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Skin temperature biofeedback training is receiving widespread attention as a nonmedical treatment for migraine headaches. Previous treatment for migraine relief has not been consistently successful. Sargent, Green and Walters (1972, 1973) claim that thermal feedback training gives migraineurs control over vascular changes which lead to migraine head pain, without the undesirable side effects of pharmacological treatment. However, treatment successes claimed for thermal feedback may be due to placebo effects. Insofar as thermal feedback training is effective, it is important to identify migraineurs most likely to benefit from it, and ways to improve effectiveness of the procedure.

Migraine refers to a group of related symptom complexes of which headache is the most common complaint. Migraine headache is a pulsating head pain accompanied by other disturbances mediated by the autonomic nervous system. Estimates of migraine incidence in adults range from 5 to 10 per cent, making migraine "one of the most common psychosomatic disorders" (Sacks, 1970). Frequency and duration of headache vary widely across migraineurs, but extreme intensity of pain is uniformly reported. According to Wolff (1963), characteristic features of migraine usually include unilateral onset of head pain, scalp tenderness, nausea and irritability. Often other members of the family of the migraine sufferer

have similar headaches.

Physiological concomitants of migraine were investigated by Graham and Wolff (1938) who implicated abnormal vascular changes in extracranial arteries as the mechanism of head pain. Schumacher and Wolff (1941) described the biphasic vascular changes accompanying migraine. They demonstrated that preheadache disturbances occur with occlusive vasoconstriction of intracranial arteries, and that headache resulted from dilation of extracranial arteries. The possible cause for vascular lability was considered by these investigators to be neurogenic.

At present, sympathetic control of vasomotor responses is considered to mediate migraine symptoms (Dalessio, 1972). Exacerbations of cranial vascular lability may be initiated by any stimulus which has a vascular effect. The identification of migraine precipitants and knowledge of the mechanism do not account for the cause of the migraine syndrome. Reports of familial incidence have led many physicians to consider migraine an inherited disorder. Waters (1971 a) cites strong evidence that although familial incidence is high, migraine may not be genetically transmitted. Thus, although the mechanisms of migraine have been investigated, the etiology of migraine is still unknown.

Medical treatment for migraine headache has been with vasoconstrictors, analgesics and tranquilizers. Vasoconstrictors tend to be more effective than placebo, with analgesics and tranquilizers having about the same success rate as

placebo (Friedman and Merritt, 1957). Alternatives to medical treatment have included traditional psychotherapy, autogenic training, hypnosis and a wide range of behavioral procedures.

Since Miller's (1969) demonstrations of operant control of autonomic functioning with animals, growing consideration has been given to applications of biofeedback in training humans to control disordered autonomic functioning. Sargent, Green and Walters (1972) combined autogenic training (Schultz and Luthe, 1969) with feedback of skin temperature to explore their combined effect on migraine headache. Skin temperature was used as a measure of blood flow which could be obtained nonintrusively. The authors reported improvement of migraine for about 70% of their patients and concluded that the procedure merited further investigation. They cautioned that "the placebo factor was not evaluated", though they acknowledged that they were indeed working in the area of conscious suggestion to bring about physiological change.

Andreychuk and Skriver (1975) compared the effects of three different training procedures on migraineurs' report of their headaches: skin temperature feedback, alpha feedback, and autohypnosis. All groups showed significant reductions on a composite index of headache measures, with degree of success reported in the aforementioned order. Differences between groups were not significant. The

authors assessed hypnotic suggestibility for each subject and found that the skin temperature feedback group had the highest average suggestibility, while the autohypnosis group had the lowest. The authors concluded that the particular biofeedback treatments were not necessarily the relevant variables in producing these effects.

Friar and Beatty (1976) used a different feedback mode, plethysmography, to explore the contentious issue of specificity of the effects of biofeedback training for migraine relief. They found significantly greater improvement in the group trained to constrict extracranial vessels than in their placebo group, which was trained to constrict finger vessels. However the group considered to be receiving a placebo treatment may have actually learned a procedure with headache-exacerbating effects.

Purpose

The present study investigated two possible treatment components in the skin temperature feedback training procedure first described by the Sargent studies (1972, 1973). Past failure to assess the possible placebo effect of that procedure has left its presumed mechanisms in doubt while retarding its acceptance as a method of treatment. In this study the placebo control group was trained to stabilize skin temperature. Procedure and apparatus were identical to the active treatment group, but stabilizing skin temperature was considered to be without specific effect for

migraine. The first hypothesis was that migraineurs trained by biofeedback with instructions to raise finger temperature would experience significantly greater migraine relief than migraineurs trained with instructions to stabilize finger temperature. Relief was measured in terms of four variables: headache frequency, duration, intensity, and amount and type of medication used.

The second component investigated was preheadache cue training. It has been suggested that interventions which affect blood flow may be successful only during a preheadache phase or early after the onset of actual head pain (Friedman, 1968; Turin and Johnson, 1976). Identification of preheadache cues might facilitate early recognition of an impending migraine and thus improve effectiveness of the feedback-trained response. The second hypothesis was that, of two groups of migraineurs trained by biofeedback with instructions to raise finger temperature, the group taught to identify preheadache cues would experience significantly greater migraine relief than the group with no cue training.

Skin temperature control, both with and without feedback, was also investigated. Demonstration of control of skin temperature as instructed is important for determining the specific effects of biofeedback training. An additional analysis was performed to examine the effects of feedback instructions and of various migraineur characteristics on skin temperature control. Similar analyses examined variables which

might predict improvement in headache frequency, duration, intensity or medication use.

Method

Subjects

Thirty-two migraineurs were recruited through introductory Psychology and Sociology classes at the University of Manitoba. They ranged in age from 18 to 56 with a mean age of 25. All subjects were required to have their physicians sign a form (Appendix A) indicating that they suffered from migraine and that no other vascular or central nervous system involvement was suspected. All subjects were later interviewed and diagnosed by the experimenter. Two subjects were excluded because the experimenter disagreed with the original diagnosis of migraine. A third subject lost all her self-recorded data after training, so her results could not be included in the analysis of posttraining headache data. The remaining twenty-nine migraineurs included twenty females and nine males. They expected a mean of three migraine headaches per month before training. Migraineurs were not required to discontinue use of medication of any kind. Twenty-one of twenty-nine migraineurs received some academic credit for participating in the investigation.

Equipment

Skin temperature feedback was provided for all subjects with a Biofeedback Technology (BFT) 301 skin temperature trainer with thermistor attached to dominant index finger on

the palmar side. Migraineurs received analogue visual feedback by a meter which was not visible during the adjustment and no-feedback periods. Information about skin temperature was recorded for each training session using a Gelman Servoscribe potentiometric recorder. Each biofeedback training session was conducted in a small, sound-attenuated room containing a reclining chair and the feedback apparatus. The room temperature was maintained at approximately 70°F (21°C) for each training session for all migraineurs.

Procedure

Data recording. All migraineurs were instructed in careful recording of headache data. Data were charted for three weeks prior to training, during six weeks of training and for three weeks following training. The headache record (Appendix B) included the date, time of onset, duration, maximum intensity of headache rated on a five-point scale and an account of all medication taken. The basic four dependent headache measures of frequency, duration, intensity, and medication used were derived from this self-recorded data file. Migraineurs were also requested to note when they used what they had learned in training with a headache. Other information collected included symptoms accompanying head pain, migraineur's location at onset of headache plus appraisal of each headache as migraine or some other type. The group receiving preheadache cue training was later asked to keep a daily record (Appendix C) of additional information

about potential precursors to migraine. They recorded possible physical or emotional preheadache warnings and possible dietary, stressor, sleep pattern or hormonal triggers as suggested by the Migraine Foundation of Canada (Note 1).

Assignment to training conditions. Migraineurs were matched in triads as closely as possible for age and for sex. From each triad, two migraineurs were assigned randomly to receive temperature increase instructions and the third to receive temperature stabilize instructions. In order to minimize experimenter bias, no distinction was made among migraineurs assigned to receive instructions to increase, until following the fourth training session. At that time ten of these migraineurs were assigned to the "increase plus" condition and received preheadache cue training. Each group was comprised of ten migraineurs except the one receiving instructions to increase without preheadache cue training (the "increase" group), which was comprised of nine.

Training. Each migraineur attended a total of seven forty-five to fifty minute sessions, of which five were biofeedback training. The first session was a group introduction to data recording and a description of the training program. During this meeting, migraineurs completed a questionnaire (Appendix D) about their migraines and Rotter's locus of control scale (Rotter, 1966). After the collection of pretraining headache data, migraineurs received individual biofeedback training in five weekly sessions. Between the

fourth and fifth training sessions, the experimenter met with each subject for a diagnostic interview. The experimenter classified each migraineur's headaches as common or classic. Migraineurs also related information about other psychosomatic disorders, family headache incidence and cardiovascular disorder histories.

During the diagnostic interview, migraineurs in the "increase plus" group briefly discussed their migraine histories. The remainder of the interview was devoted to pre-headache cue identification. The experimenter explained that increased awareness of possible warning signs would facilitate earlier intervention, and that intervention is believed most effective during a critical period early in the course of a migraine. After presenting this rationale, the experimenter asked the migraineur if he/she was aware of any such cues. After discussing the migraineur's impressions about possible cues, the experimenter gave the migraineur a copy of the cue training record (Appendix C). Each type of cue was discussed and examples given. The migraineur was then requested to keep a daily record of possible cues for four weeks.

Two separate feedback procedures were used. Fourteen subjects were trained to control temperature using a format of a twenty-minute adjustment period followed by twenty-five minutes of feedback training. This format, adopted from Turin and Johnson (1976), was followed for the first four

feedback sessions. The final session consisted of a twenty-minute adjustment period, a fifteen-minute no-feedback period and a ten-minute final feedback period. Some migraineurs trained in this original procedure showed a decline in temperature control performance from feedback periods to the no-feedback period of the final session. Because of this decline, a modified training procedure was instituted to promote optimal performance without feedback. Performance without feedback was considered crucial because migraineurs would not have access to feedback during migraines.

The remaining fifteen migraineurs were trained with the modified procedure. All five sessions were restructured so as to include a no-feedback period. These migraineurs received a twenty-minute adjustment period, ten minutes without feedback with instructions to control temperature and fifteen-minutes with feedback training for the first four sessions. The fifth session was identical in format to the fifth session for migraineurs trained with the original procedure. As a result of the modification of procedure, a two-level procedure factor was used in analyses of data.

After the first feedback training session all migraineurs were asked to practice at home what they had learned in training. The importance of daily practice for a fifteen-minute period was emphasized. During this time migraineurs were instructed to try to regulate finger temperature as prescribed in the training, doing whatever seemed effective

during training sessions with biofeedback. The rationale presented for this homework was to promote transfer of training beyond the training setting and to facilitate control under more adverse circumstances. Responses to a post-training questionnaire (Appendix E) indicated that number of homework practice sessions per week ranged from two to ten with a mean of about 4.5.

Instructions. All migraineurs received brief instructions before each training session. Instructions all implied that appropriate application of the trained temperature control response would have a beneficial effect on pain-producing scalp arteries. The two groups of migraineurs learning to increase their finger temperature received identical instructions as follows:

Instructions to Temperature Increase Groups

> After twenty minutes to allow your body to adjust to this environment, you will receive instant feedback about your finger temperature. The control of finger temperature influences blood flow through vessels in body extremities. Research indicates that increasing blood flow through finger vessels will favorably effect pain-producing scalp vessels. It is expected that increasing finger temperature will produce this favorable effect. Please remain in the chair without moving your (dominant) hand. After the twenty-minute adjustment period, try to increase your finger temperature.

The placebo group received a rationale for the potential effectiveness of stabilizing skin temperature. Instructions presented to migraineurs in this group were as follows:

Instructions to Temperature Stabilize Group

> After twenty minutes to allow your body to adjust to this environment, you will receive instant feedback about your finger temperature. The control of finger temperature influences blood flow through vessels in body extremities. Research suggests that migraine is due to instability of blood vessels in the scalp. It is expected that maintaining a steady finger temperature will stabilize scalp vessels. Please remain in the chair without moving your (dominant) hand. After the twenty-minute adjustment period, try to stabilize your finger temperature.

No prior instructions or suggestions were offered by the experimenter regarding strategies to be used in attempting to control finger temperature. Migraineurs were left to their own devices to learn how to control finger temperature as in other studies with normals (Keefe, 1975; Alberstein, 1977) and migrainous subjects (Wickramaskera, 1973; Turin and Johnson, 1976).

Subject-Experimenter Contact

Effort was made to minimize differences in subject-experimenter contact across groups. After the introductory group session, all five training sessions and the diagnostic interview were individual appointments. Experimenter contact time was constant across groups trained with the original procedure and across groups trained with the modified procedure. All migraineurs were greeted by the experimenter, told to affix the heat-sensitive thermistor to their finger, and then were read the appropriate instructions. The experimenter then left the room, and returned only at the end of the adjustment period to tell migraineurs to begin to

try to control finger temperature. The experimenter left the room again and returned after the end of the allotted time. Interchange with the experimenter was not encouraged with migraineurs trained under the original procedure.

More contact with the experimenter occurred for migraineurs trained with the modified procedure. The experimenter returned at the end of each biofeedback training session and showed each migraineur the charted record of his or her finger temperature during the session. Encouragement and praise were offered when evidence of success in controlling temperature as instructed was apparent, or when migraineurs spoke of homework or attempts to actually control headache. These discussions were limited to ten minutes maximum duration.

Results

Results reported here include several ancillary analyses, multivariate hypotheses tested, and several exploratory analyses of temperature control and migraine measures. Ancillary analyses include a rating of reliability of the diagnosis of two migraine subclassifications, and two analyses of group temperature changes. Multivariate contrasts of migraine measures adjusted for certain pretraining differences are used to test formal hypotheses regarding the effects of increasing skin temperature and of preheadache cue training. Inconclusive results are explored using multiple regression analyses of individual migraine measures. An additional multiple regression analysis is used to explore predictors of skin temperature increase.

Diagnostic Reliability

Agreement across professionals diagnosing migraine is considered of importance in treatment. A reliability coefficient can not be reported for the diagnosis of migraine in this study. The experimenter formed a diagnostic opinion only for subjects who already had been positively diagnosed by a physician. Individuals not diagnosed as migrainous by their physicians were not included in the study. The experimenter agreed with the physicians' diagnosis of migraine for 30 of 32 subjects diagnosed. This represented a 94% agreement rate.

The subclassification of migraine may account for some

variance in migraine management. The identification of pre-headache cues may augment the therapeutic effects of medication (Friedman, 1968) or of a biofeedback-trained response (Turin and Johnson, 1976). The subclassification of classic migraine is based on the occurrence of a neurogenic aura preceding the headache. This aura may be used as a valuable cue for headache.

All physicians diagnosing the headaches of prospective subjects in this experiment were requested to classify their migraines as "classic", "common" or "other". Twenty-five of the twenty-nine attending physicians whose patients were included in the study specified common or classic migraine. Two physicians specified another, and one left the question blank. The final physician noted that he didn't know the difference between the common and classic subclassifications.

Reliability of the common-classic distinction was determined using the experimenter's blinded diagnosis of each migraineur included in the study, for comparison with subclassification by each physician. Agreement on the basis of chance alone was expected to be 50%. Compared subclassifications resulted in 52% agreement, yielding a phi coefficient of .014 (see Table 1). Because of the low level of diagnostic agreement, the subclassification of migraine was not used as a variable in this study.

Table 1

Diagnostic Reliability for
Distinguishing Common vs. Classic Migraine

		By experimenter: Classic	Common	Not Specified	
By physicians:	Classic	3	4	0	7
	Common	8	10	0	18
	Not Specified	1	3	0	4
		12	17	0	29

* $\emptyset$ = .014, not significant.

Temperature Control

Demonstration of ability to control skin temperature as instructed to each migraineur was essential to the claim that changes in headache measures were due to vasomotor control. It was considered necessary to demonstrate acquisition of temperature control without feedback as well as with feedback. Results of two separate training by procedure factorial analyses of covariance are reported on the effects of temperature control with feedback (session 4) and without (session 5). The covariate used in each analysis was the adjustment period temperature recorded just prior to instructions to begin to control temperature on each respective session (baseline temperature). This covariate was used to adjust for the effect of possible differences in initial temperature across cells. Temperature change was measured as the largest increase over baseline temperature recorded during the control period after Mullinix, Norton, Hack and Fishman (1978), and Reading and Mohr (1976). In cases where there was no increase, the largest decrease was used as the measure of temperature change.

With feedback. Temperature changes for session four were used to analyze control of skin temperature with feedback. Session four was the last session before a no-feedback period was introduced for migraineurs trained with the original procedure. The novel no-feedback period of the fifth session could have produced anxiety, influencing temperatures

recorded during the feedback period of the final session. Four migraineurs in the original procedure did mention that they perceived the no-feedback period of the final session as a test. Temperature change during the fourth session feedback period was chosen for analysis to avoid this possible confound. The duration of the feedback period varied slightly between original and modified procedures. Changes during a twenty-five-minute feedback period were recorded with the original procedure and during a twenty-minute feedback period with the modified procedure.

There was no significant variation across cells on the covariate, baseline temperature (see Table 2). However small differences were partialed out of all measures of temperature change used in the analyses. Differences between original and modified procedures on control of skin temperature change resulted in a nonsignificant *F*. The effect of trained control with skin temperature feedback contributed to observed differences across groups receiving different instructions for direction of temperature control. The interaction between effects attributable to feedback training and procedure was nonsignificant. The percentage of total variability accounted for by the feedback training factor (eta; Hays, 1963) was 25%. The percentage of total variance accounted for by the procedure factor was 3%.

Simple post hoc one-tailed *t* tests were used to examine the differences between groups which accounted for the

Table 2

Analysis of Covariance of Session Four Temperature Change (with feedback) from Baseline

Source of variation	df	Mean squares	F.	p.
Covariate				
Baseline temperature	1	0.161	0.061	.808
Main effects				
Training	2	11.041	4.174	.029
Procedure	1	2.643	0.999	.328
Interaction				
Training x procedure	2	2.585	0.977	.392
Residual	22	2.645		
Total	28			

Table 2a

Post Hoc Comparisons of Covariate-Adjusted Group Means for the Training Factor

Comparison	t(22)	p< (one tailed)
I' - I	1.486	.10
I' - S	2.887	.005
I - S	1.325	.10
½(I+I') - S	2.095	.025

Note. I' = "increase plus" group.
I = "increase" group.
S = "stabilize" group.

significant main effect of skin temperature training (see Table 2a). The largest difference between groups on mean temperature increase was between the "stabilize" group and the temperature increase group which later received preheadache cue identification instruction ("increase plus" group). The resulting significant *t* test suggests that the "increase plus" group learned to raise temperature significantly higher than the group instructed to stabilize skin temperature. Differences between means of the other two group pairings were nonsignificant. The "increase plus" group had not received the preheadache cue identification instruction by feedback session four. The two increase groups were expected to demonstrate comparable magnitudes of increase of skin temperature over the "stabilize" group. But the difference between the two increase group means was not as close as expected.

Typical records of skin temperature change during the fourth session can be described for those who increased, those who could not increase, those who stabilized, and those who could not stabilize. All four records show some intrasession variability for the adjustment period, but with temperatures stabilizing after about twelve to fifteen minutes. Migraineurs who increased their temperature did so within the first ten minutes of the feedback period, and maintained the increase for not more than ten minutes. Three of 19 migraineurs instructed to increase did not

during session four. These migraineurs showed variability above and below baseline levels, with changes of largest magnitude being decreases. The largest decrease recorded was less than 2^{o} Fahrenheit. Migraineurs who effectively stabilized skin temperature showed almost no variability from their baselines. Migraineurs who were instructed to stabilize but who did not tended to show much variability, with largest increase from baseline being less than 3^{o} Fahrenheit.

Without feedback. Demonstration of control of skin temperature as instructed without feedback is crucial to inferences about transfer of ability to control temperature beyond the training setting. For this reason a second factorial ANCOVA was performed using temperature change (as described above) for the no-feedback period of the fifth session as dependent variable. The final session was chosen because all groups had a no-feedback period lasting fifteen minutes.

The summary table for this analysis (Table 3) shows greater variability within all sources of variance, except the interaction, than in the analysis of session four temperature changes. Differences between group baseline temperatures were nonsignificant but all further computations were adjusted for baseline temperature to provide more stringent tests. The difference between procedures in average temperature increase was larger than in session four, but

Table 3

Analysis of Covariance of Session Five Temperature Change (without feedback) from Baseline

Source of variation	df	Mean squares	F.	p.
Covariate				
Baseline temperature	1	13.244	2.065	.165
Main effects				
Training	2	27.560	4.298	.027
Procedure	1	7.508	1.171	.291
Interaction				
Training x procedure	2	1.493	0.233	.794
Residual	22	6.412		
Total	28			

Table 3a

Post Hoc Comparisons of Covariate-Adjusted Group Means for the Training Factor

Comparison	t(22)	p< (one tailed)
I' - I	2.683	.01
I' - S	2.331	.025
I - S	-0.860	.20
$\frac{1}{2}$(I+I') - S	0.941	.20

Note. I' = "increase plus" group
I = "increase" group
S = "stabilize" group

nonsignificant. The effect of training temperature control as measured without feedback was significantly different across groups receiving different instructions for direction of temperature control. The interaction between the training and procedure factors was not significant. The percentage of total variability accounted for by the training factor was again 25%, while the corresponding percentage accounted for by the procedure factor was 4%.

Simple post hoc one-tailed t tests were used again to examine the differences between groups which accounted for the significant main effect of skin temperature training (see Table 3a). The difference between average temperature increase of the "increase plus" and the "stabilize" group was significant. This indicated that the "increase plus" group demonstrated a significant mean increase relative to the "stabilize" group. The difference between the "increase" group and the "stabilize" group means was not significant while the difference between the two increase group means was. This suggested that migraineurs in the "increase plus" group demonstrated significantly greater increases in skin temperature than the "increase" group.

The "increase" group showed a mean covariate-adjusted increase of $-.11^{\circ}F$ in the no-feedback period of session five. This represented a turnabout from the average increase of $1.78^{\circ}F$ demonstrated in the feedback period of session four. The average performances of the "stabilize" group and the

"increase plus" group were, if anything, better than during session four. The stabilize group had adjusted mean increases of .80°F in session four and 0°F in session five. The "increase plus" group demonstrated adjusted mean increases of 2.70°F during session four and 2.80°F in session five.

Inspection of the raw temperature change data for the "increase" group indicates that two migraineurs performed quite differently between session four and session five. These migraineurs had recorded increases of 2 and 3 degrees during session four with feedback, but had not been able to increase temperature during session five. Both spontaneously cited an inability to concentrate in explaining their persistent decreases in skin temperature during session five. These unexpected turnabouts from session four account largely for the smaller mean temperature change of the "increase" group for session five. This lowered mean increase may account for the difference between the two increase groups for session five and the nonsignificance of the difference between the "increase" and the "stabilize" groups.

Results of the analysis of covariance for the no-feedback period of session five have significant implications for the analysis of the headache measures. Observed changes in migraine measures between groups may be attributed to the presumed specific effect of controlled temperature increase only for those groups which demonstrated significant increases without feedback. Results of the *t* tests performed between

training groups suggest that the "increase plus" group learned how to initiate temperature increases during a no-feedback period significantly better than did the other two groups. If migraine can be controlled by trained increase in finger temperature, the "increase plus" group should demonstrate more improvement on posttraining headache measures than the "stabilize" group or the "increase" group.

Multivariate Analyses of Self-Report Migraine Measures

In order to examine the pattern of change of dependent migraine measures from pretraining to posttraining, two separate multivariate analyses were performed. The first analysis was conducted to examine the significance of multivariate change from pre to posttraining. The second analysis was conducted to test formal multivariate hypotheses about differences between groups as measured during the posttraining period.

Multivariate analyses were performed because the migraine measures were considered part of a package. Each measure assessed a different dimension of migraine complaint. The package of four measures was considered a better index of change than any individual measure. It included frequency measured over a three-week period, duration in hours of each migraine recorded, intensity rated on a five-point scale, and medication weighted on a three-point scale. The medication scale (Medina, Diamond, and Franklin, 1976) assigns a weight of three to ergot derivatives, two to narcotics and

one to analgesics. Both multivariate analyses used the same two by three factorial design used to analyze temperature control.

Pre-post change. The first multivariate analysis was performed to evaluate global change across all groups from pre to posttraining. Although there are problems with analyzing change scores (Kenny, 1975), the use of standardized change scores or covariate-adjusted posttraining measures was not considered appropriate. Subtracting pre scores standardized around the vector of pretraining means from post scores standardized around the vector of posttraining means would always result in no differences because of the property of standardized scores. Analysis of covariance can not effectively test change from pre to posttraining. The analysis of covariance is intended for testing group differences.

The null hypothesis tested with this analysis of change scores was that change from pre to posttraining on the package of migraine measures for all migraineurs was zero. This test of the vectors of grand change score means yielded a significant multivariate F. This F indicates that significant changes occurred from pretraining to posttraining on some combination of the migraine measures. Inspection of the univariate F's for each of the four dependent measures indicated large F statistics for both change in frequency and in duration (see Table 4). Univariate tests of change

Table 4

Multivariate Analysis of
Pre-Post Migraine Measure Change Scores

Test of the Vector of Grand Means

Source of variation	dF	F	p<	Step-down F	p<	Standardized discriminant function weight
Multivariate	(4,20)	13.8767	.0001			
Univariate						
Frequency	(1,23)	36.9439	.0001	36.9439	.0001	-.9420
Duration	(1,23)	14.2207	.0010	6.2294	.0206	-.4930
Intensity	(1,23)	6.5589	.0175	0.7621	.3926	-.5536
Medication	(1,23)	4.1447	.0535	1.7878	.1962	.4977

Table 5

Observed Cell Means for Pre-Post Migraine Measures Change

Cell[a]	Identification	Frequency	Duration	Intensity	Medic.
1	stabilize, original proc.	3.40	1.46	1.40	0.54
2	stabilize, modified proc.	2.00	3.76	0.57	0.84
3	increase, original proc.	1.50	1.40	0.13	0.00
4	increase, modified proc.	1.20	3.17	-0.60	0.96
5	increase plus, original	2.80	4.40	2.18	0.26
6	increase plus, modified	0.20	5.70	1.80	3.20

[a] $n = 5$ for each cell, except $n = 4$ for cell 3.

in intensity and in medication use were also both significant.

The specific package of change measures which contributed significantly to the significant multivariate test was determined by examination of the step-down F statistics. These F's test the significance of the loss to the multivariate package of measures when a given measure is removed from the package. These statistics indicate that both measures of change in frequency and duration contributed to the significant multivariate F. Tests of the contribution to the significant multivariate package of intensity and medication were not significant. Standardized discriminant weightings for each change measure indicated that change in frequency contributed most to the significant multivariate test, and that the other three change measures were approximately equally weighted.

Inspection of observed cell mean change measures showed improvement in frequency and in duration (see Table 5) across all cells. Evidence for improvement in average intensity and medication use was noted in five of six cells. Thus, from a total of 24 cell by migraine change measure combinations (6 x 4), 22 showed at least some improvement from pre to posttraining.

Group contrasts. A second multivariate analysis was conducted to test the primary hypotheses regarding differential effect of training between groups, using posttraining reports on all migraine measures adjusted for pretraining

differences. In order to use a set of covariates a test of homogeneity of variance-covariance matrices between cells must be conducted. If the null hypothesis of no differences in variance-covariance structure between cells is rejected, the use of that package of covariates is inappropriate.

Unfortunately, cell size was too small to conduct this test of homogeneity of variance-covariance matrices using all four pretraining measures as covariates. Components for a reduced package of pretraining measures to be used as covariates were sought which would correlate with each measure omitted and would not violate the assumption of homogeneity of variance-covariance matrices. Pretraining measures of migraine frequency and medication used for relief were chosen for this package of covariates to adjust for pretraining differences between groups. Pretraining intensity was excluded because differences between group means on this measure were small. Pretraining migraine duration was excluded because this variable represented the largest threat to violate the assumption of homogeneity of variance-covariance matrices.

A stepwise regression analysis of pretraining frequency and medication as predictors for posttraining duration supported their use as a pretraining covariate composite. The large step-down F (2,21) of 10.2017 ($p=.0009$) indicated that the covariate composite accounted for a significant amount of variation in the posttraining measure of migraine

duration. The test of homogeneity of variance-covariance matrices within cells for the covariate composite resulted in a $\underline{F}$ (40, 32.1904) of 1.0452 with p = .4527. The value of the covariates in accounting for variance in the analysis of posttraining variables was affirmed by a test of no association between covariates and posttraining measures which yielded a $\underline{F}$ (8,36) of 5.2936 with p = .003. Results of these three tests suggest that the use of the covariate composite was statistically appropriate, and that the composite accounted for a significant amount of variance in the dependent measures. A canonical correlation indicated that almost 23% of the variation of posttraining measures was accounted for by the two pretraining covariates.

Hypotheses about differential improvement on self-report of migraine measures were tested by planned multivariate contrasts of posttraining measures adjusted for pretraining differences by the covariate package. Tests of differences between the two increase groups, between the "stabilize" group and the average of the two increase groups, and between the procedures were performed. The two planned contrasts reported for the training factor are orthogonal and represent tests of the two major hypotheses. The effects of preheadache cue instruction were tested by contrasting the "increase" group with the "increase plus" group. The effects of increasing digital skin temperature over placebo were tested by contrasting the "stabilize" group with the

average of the two increase groups.

The test of the interaction of procedure by training factors was not significant. This indicates that the pattern of training group differences were not significantly different across the two procedures. As a result, the variation due to the interaction was pooled with the error term for all multivariate contrasts.

The contrast of the "increase" group and the "increase plus" group yielded a nonsignificant F (see Table 6a). Only one univariate test, with duration as dependent measure, associated with the multivariate test had a reasonably large value of F (1,23) = 6.3729 with p = .0190. Inspection of covariate-adjusted group means for the two groups showed trends in the expected direction on all four dependent variables, i.e. migraineurs in the "increase plus" group reported lower posttraining measures than migraineurs in the "increase" group (see Table 7).

The contrast of the "stabilize" (placebo) group and the average of the two increase groups resulted in a nonsignificant multivariate F (see Table 6b). The multivariate F was associated with nonsignificant univariate tests for each dependent measure. The failure of the "increase" group to demonstrate a significant mean increase in temperature without feedback over the "stabilize" group confounds interpretation of this finding.

A post hoc test of equality of mean vectors between

Table 6a

Multivariate Analysis: Posttraining Migraine Measures Adjusted for Pretraining Frequency and Medication

Planned Orthogonal Contrast: Increase vs. Increase Plus

Source of variation	dF	F	p<	Step-down F	p<	Standardized discriminant function weight
Multivariate	(4,20)	2.0400	.1273			
Univariate						
Frequency	(1,23)	1.0425	.3179	1.0425	.3179	0.8574
Duration	(1,23)	6.3729	.0190	7.3148	.0130	-1.5750
Intensity	(1,23)	2.1325	.1578	0.0213	.8854	-0.1544
Medication	(1,23)	0.9956	.3288	0.1964	.6625	0.2634

Table 6b

Multivariate Analysis: Posttraining Migraine Measures Adjusted for Pretraining Frequency and Medication

Planned Orthogonal Contrast: Stabilize vs. Average of Both Increase Groups

Source of variation	dF	F	p<	Step-down F	p<	Standardized discriminant function weight
Multivariate	(4,20)	0.5927	.6720			
Univariate						
Frequency	(1,23)	0.0892	.7680	0.0892	.7680	-0.0298
Duration	(1,23)	0.0062	.9382	0.2597	.6154	0.9945
Intensity	(1,23)	1.2717	.2711	2.1173	.1605	-1.4065
Medication	(1,23)	0.0687	.7957	0.0071	.9337	0.0833

Table 6c

Multivariate Analysis: Posttraining Migraine Measures Adjusted for Pretraining Frequency and Medication

Post Hoc Contrast: Stabilize vs. Increase Plus

Source of variation	dF	F	p<	Step-down F	p<	Standardized discriminant function weight
Multivariate	(4,20)	0.6025	.6653			
Univariate						
Frequency	(1,23)	0.0723	.7904	0.0723	.7904	-0.0745
Duration	(1,23)	0.0226	.8819	0.3452	.5629	1.0671
Intensity	(1,23)	1.1749	.2897	2.0887	.1632	-1.3842
Medication	(1,23)	0.0543	.8178	0.0049	.9449	0.0686

Table 6d

Multivariate Analysis: Posttraining Migraine Measures Adjusted for Pretraining Frequency and Medication

Contrast of the Procedures: Original vs. Modified

Source of variation	dF	F	p<	Step-down F	p<	Standardized discriminant function weight
Multivariate	(4,20)	2.4251	.0818			
Univariate						
Frequency	(1,23)	1.4431	.2419	1.4431	.2419	-0.8810
Duration	(1,23)	0.0583	.8114	3.5209	.0740	1.1306
Intensity	(1,23)	3.1830	.0877	3.0575	.0950	-1.0476
Medication	(1,23)	0.0630	.8041	1.0293	.3225	0.5566

Table 7

Cell Means on Posttraining Migraine Measures Adjusted for Pretraining Frequency and Medication

Cell[a]	Training group, procedure	Frequency	Duration	Intensity	Medication
	Stabilize				
1	Original	1.00	4.29	1.11	1.06
2	Modified	1.48	3.84	2.29	0.91
	Increase				
3	Original	1.33	6.85	2.38	1.58
4	Modified	1.82	6.39	3.57	1.42
	Increase plus				
5	Original	0.89	1.46	1.32	0.87
6	Modified	1.38	1.00	2.51	0.73

[a] $n = 5$ for each cell, except $n = 4$ for cell 3.

the "stabilize" group and the "increase plus" group was conducted to reanalyze the effect of increasing skin temperature on migraine relief. The "increase plus" group was chosen for this contrast because it had demonstrated a significant mean temperature increase over the "stabilize" group, whereas the "increase" group had not. This post hoc contrast resulted in a nonsignificant F (see Table 6c). No associated univariate F statistics approached significance; nor did any step-down F statistics. The nonsignificance of this contrast suggests that the "increase plus" group did not show greater improvement on any migraine measure, although it did demonstrate significantly greater temperature increases without feedback.

The final a priori contrast tested the difference between procedures used to train control of skin temperature on the four migraine measures. The multivariate test of equality of mean vectors between the original and modified procedures resulted in a $F(4,20) = 2.4251$ with $p = .0818$ (see Table 6d). The associated univariate statistics included one F statistic near conventional significance levels. Using posttraining migraine intensity as dependent measure yielded a $F(1,23)$ of 3.1830 with $p = .0877$. Step-down F statistics indicated two variables may have contributed to the differences detected by the multivariate test. These two variables were posttraining intensity and duration. Standardized discriminant weights were used to maximize

differences between procedures on the four variables; they indicated that migraineurs trained by the original procedure reported lower intensity but longer duration of migraines than migraineurs in the modified procedure.

Multiple Regression Analysis of Temperature Control

A post hoc multiple regression analysis was conducted in an attempt to determine variables which predicted migraineurs' change in temperature during the final no-feedback period. Twelve possible predictors were taken from the pretraining and posttraining questionnaires (Appendices D and E) completed by all migraineurs. Predictors collected before training included the following: family migraine history (FAMHIST), age, sex, degree of externality based on Rotter's scale (ROTTERIE), and four self-rated variables including average number of migraines per month, number of years since migraine was first discussed with a physician (FIRSTDOC), satisfaction with migraine relief by medication (SATIS) and expectation for biofeedback to improve headaches (EXPECT). The four variables collected after training included a dichotomous variable representing assignment to stabilization or increase training groups (TRNGRP), average number of times per week temperature control was practiced (PRCTC), a self-rating of degree of relaxation during the final training session (RELAX) and a rating of frequency of perceived change in feeling of warmth while attempting to control temperature without feedback (FEELWRM). Table 8

Table 8

Variables Used in Multiple Regression Analyses

(Starred variables were collected prior to training)

Name	Description	Scaling	Use
EXPECT	Migraineur's expectation for headache relief through biofeedback.	ordinal (1-5, 5 high)	predictor
SATIS	Migraineur's reported satisfaction with headache relief with medication.	ordinal (1-4, 4 high)	predictor
FIRSTDOC	Number of years since first discussing migraine with a physician.	continuous	predictor
FAMHIST	Migraineur's report of relatives who have migraine.	dichotomous (0-1, 1-history)	predictor
AGE	Age in years of migraineur.	continuous	predictor
SEX	Sex of migraineur.	dichotomous (0-1, 1-male)	predictor
HDKSMON	Migraineur's estimate of number of migraine per month.	continuous	predictor
ROTTERIE	Number of external items selected from Rotter's measure of locus of control.	ordinal (22 maximum)	predictor
RELAX	Migraineur's rating of relaxation during the final feedback session.	ordinal (1-7, 7 least)	predictor
PRCTC	Migraineur's report of average number of times practicing temperature control per week.	continuous	predictor

Table 8 Cont'd.

Name	Description	Scaling	Use
TRNGRP	Instructions received (stabilize or increase).	dichotomous (0-1, 1 increase)	predictor
TEMPSES5	Change in temperature relative to adjustment period, during session five without feedback.	continuous	predictor and criterion
FEELWRM	Frequency of perceived change in skin temperature during attempts to control skin temperature.	ordinal (1-4, 4 consistently)	predictor
MIGFR	Report of number of migraines reported for 3 week post-training period.	continuous	criterion
PRFRQ	Report of number of migraines experienced during 3 weeks before training.	continuous	adjustment for pretraining differences
MIGDUR	Averaged report of duration of migraines reported for 3 week post-training period.	continuous (number of hours)	criterion
PRDUR	Averaged report of duration of migraines experienced during 3 weeks before training.	continuous (number of hours)	adjustment for pretraining differences
MIGIN	Averaged rating of intensity of migraines reported for 3 week post-training period.	ordinal (1-5, 5 most)	criterion
PRMIGIN	Averaged rating of intensity of migraines reported for 3 weeks before training.	ordinal (1-5, 5 most)	adjustment for pretraining differences

Table 8 Cont'd.

Name	Description	Scaling	Use
MIGMED	Averaged weighting of medication relief sought for migraines, reported for three week post-training period.	continuous	criterion
PRMED	Averaged weighting of medication relief sought for migraines, reported for three week pre-training period.	continuous	adjustment for pretraining differences

describes each scale in detail.

The order of entry of each variable into the regression on temperature change was not preestablished. Predictors were entered into the equation according to the incremental amount of variability each explained in the criterion variable over the remaining variables not yet entered. All twelve predictors were forced into the regression equation, with the first five contributing significantly to predictability of temperature change.

Three statistics were used to determine the importance of the predictors. The first was the order of entry based on the magnitude of significant "Fs-to-enter", reassessed as each predictor was added to the regression equation. The second measure of importance was the magnitude of "F-to-remove"; once a predictor entered the equation, its relative contribution to the prediction package was tested with this statistic. This statistic was also reassessed with each change in the prediction package, so F-to-remove statistics were reported only for the last equation which included all predictors with significant Fs-to-enter. Significance was arbitrarily set at the .05 level for F-to-enter and F-to-remove. The third measure of importance of a predictor was the standardized regression weight assigned to each predictor in the final regression equation. Because standardized weights were used, the weight of each predictor included could be compared with any other regardless of

scaling differences. Unless otherwise indicated, all predictors mentioned were important based on at least two of these three statistics.

Important predictors listed in order of entry into the equation were FAMHIST, TRNGRP, ROTTERIE, SEX and RELAX (see Table 9). ROTTERIE was important on entry but became less valuable as a predictor after the next two variables were entered. This decrease in importance was due to magnitude of simple correlation of ROTTERIE with RELAX ($r = -.30$). When the relaxation variable was entered on the fifth step, some of the variability explained by the locus of control measure became predictable by the relaxation variable. None of the remaining seven variables contributed significantly to the regression equation, as determined by values of F-to-enter.

The absolute value of each standardized regression weight allowed for comparison of relative importance regardless of scaling differences. The sign of the standardized weight provided additional useful information given knowledge of the scaling of each predictor (see Table 8). Both FAMHIST and TRNGRP were positively weighted: having relatives with migraine and belonging to an increase group were positively correlated with positive temperature change without feedback. The remaining three variables were negatively weighted. This indicated that internality as defined by the Rotter measure, being female, and relaxation during the

Table 9

Multiple Regression of Predictors on TEMPSES5

Predictor	Order of entry	F-to-remove	Standardized regression weight	Proportion of criterion variance explained
FAMHIST	1	9.074	.4960	.1098
TRNGRP	2	4.784	.3521	.0908
ROTTERIE	3	2.103	-.2372	.0853
SEX	4	4.832	-.3668	.0737
RELAX	5	3.020	-.2917	.0743

Using these predictors, Multiple R^2 = .4339.

final session correlated positively with increased change in skin temperature without feedback.

The multiple R of the package of five predictors with change in temperature without feedback during the final session was .6588 with a squared multiple R of .4340. This indicated that about 43% of the variance of the criterion variable was accounted for by the five predictors. The inclusion of all twelve predictors in the regression equation would have yielded a multiple R of .7352 with a squared multiple R of .5405.

Multiple Regression Analyses of Migraine Measures

Four separate post hoc regression analyses of the four posttraining migraine measures were conducted to examine patterns of prediction for each. The same set of twelve predictors described in the regression analysis of temperature control were used, plus TEMPSES5. This predictor was the measure of temperature change from baseline taken in the no-feedback period of the final training session.

Individual predictors were entered into each regression equation separately. The first predictor entered in each analysis was the pretraining measure corresponding to the criterion variable. Subsequent predictors entered accounted for variance in the criterion other than that attributable to preceding predictors. Entering the pretraining measure of the criterion first served to adjust the criterion for pretraining differences. Importance of predictors wa

determined by the same statistics used for the regression analysis of temperature control, described above.

Predictor packages varied for each analysis. Similar patterns of predictors resulted from the analyses of post-training migraine frequency, intensity, and medication. The prediction package for posttraining duration was distinct from those of the other measures, and is reported separately. It is interesting to note that pretraining frequency and duration were important predictors of their respective posttraining measures, but pretraining intensity and medication were not. The "F-to-enter" for each pretraining measure into its respective regression equation reflected this distinction. See Tables 10-13 for summaries of each regression equation.

The multiple R squared of each predictor package with its criterion was .808 for frequency, .735 for duration, .642 for intensity and .687 for medication. This statistic represented the amount of variance of the criterion measure accounted for by predictors. Thus it appears that the predictors accounted for more variance in frequency and duration than in the other two measures. However when the variance accounted for by pretraining differences is removed, amount of variance explained by other predictors was 37% for frequency, 36% for duration, 64% for intensity and 68% for medication. This suggested that frequency and duration were more resistant to change than intensity and medication.

Prediction by other predictors of each dependent measure adjusted for pretraining differences was better for migraine intensity and medication than frequency and duration.

After posttraining migraine measures were adjusted for pretraining differences, a similar package of important predictors emerged from the analyses of migraine frequency, intensity, and medication (see Tables 10, 12, 13). The first variable entered in each regression equation after the pretraining measure was FEELWRM. This predictor remained as the most important predictor for migraine frequency, and continued to be important, though less so, as additional predictors entered the equations for intensity and medication. The next predictor was RELAX. This predictor was the most important, judged on standardized regression weights, for migraine intensity and a close second for migraine medication. The third variable entered for analyses of intensity and medication was SEX. It was the most important predictor for migraine medication based on standardized regression weights.

Inspection of standardized regression weights for the above predictors shows that each weight had the same sign across the specified analyses. FEELWRM was positively weighted, indicating that migraineurs who reported frequently perceiving a change in degree of warmth in their skin temperature had more migraines of greater intensity and used more medication after training than those who did not.

Table 10

Multiple Regression of Predictors on Posttraining Migraine Frequency

Predictor	Order of entry	F-to-remove	Standardized regression weight	Proportion of criterion variance explained
PRFRQ[a]	1	17.085	.4727	.4359
FEELWRM	2	17.054	.4306	.1854
RELAX	3	5.811	-.2429	.0741
FAMHIST	4	6.331	-.2646	.0485
TRNGRP	5	2.527	-.1810	.0253

Using these predictors, Multiple R^2 = .7692

[a]Forced first entry

Table 11

Multiple Regression of Predictors on Posttraining Migraine Duration

Predictor	Order of entry	F-to-remove	Standardized regression weight	Proportion of criterion variance explained
PRDUR[a]	1	13.440	.5063	.3749
TEMPSES5	2	15.303	-.5282	.1045
SATIS	3	5.583	.3003	.0790
SEX	4	8.120	-.3848	.0453
FEELWRM	5	3.594	.2481	.0557
RELAX	6	2.750	-.2150	.0295
EXPECT	7	2.762	-.2300	.0132
AGE	8	2.473	.2424	.0328

Using these predictors, Multiple R^2 = .7349

[a]Forced first entry

Table 12

Multiple Regression of Predictors on Posttraining Migraine Intensity

Predictor	Order of entry	F-to-remove	Standardized regression weight	Proportion of criterion variance explained
PRMIGIN[a]	1	0.179	.0582	.0022
FEELWRM	2	9.078	.4045	.2004
RELAX	3	12.180	-.5203	.1525
FIRSTDOC	4	1.941	-.2128	.0912
SEX	5	2.913	-.2474	.1057
FAMHIST	6	3.465	-.2763	.0314
SATIS	7	3.425	.2693	.0584

Using these predictors, Multiple R^2 = .6418

[a]Forced first entry

Table 13

Multiple Regression of Predictors on Posttraining Migraine Medication

Predictor	Order of entry	F-to-remove	Standardized regression weight	Proportion of criterion variance explained
PRMED[a]	1	1.959	-.1932	.0031
FEELWRM	2	5.398	.3150	.1236
RELAX	3	15.763	-.5320	.1278
SEX	4	16.122	.5470	.1651
TEMPSES5	5	12.232	-.4678	.1284
ROTTERI	6	4.301	-.2858	.0915
AGE	7	3.201	.2420	.0477

Using these predictors, Multiple R^2 = .6872

[a]Forced first entry

The negative regression weight for RELAX indicated that migraineurs who reported feeling more relaxed during the final feedback session recorded higher levels of all migraine measures following training than migraineurs who reported feeling less relaxed. The negative standardized regression weight for SEX meant that women reported more intense migraines and used more medication during the posttraining period. Negative weighting of FAMHIST indicated that migraineurs who reported having a family history of migraine had fewer and less intense migraines during the posttraining period than migraineurs who did not so report.

The multiple regression analysis of migraine duration (Table 11) yielded a distinct pattern of predictors from that shown for frequency, intensity and medication. After pretraining duration, the first predictor to enter this equation was TEMPSES5, a variable which had been expected to be an important predictor for all dependent migraine measures, but which was only important for duration and, to a lesser extent, medication. In order of entry, the remaining important predictors were SATIS, SEX, FEELWRM, RELAX, EXPECT and AGE.

Positive regression weights for SATIS, FEELWRM and AGE indicate that these predictors were positively correlated with reports of longer posttraining duration. Negative regression weights, predicting reports of briefer migraine duration during the posttraining period, were associated

with the remaining important predictors. Migraineurs who increased temperature without feedback in the final training session reported migraines of decreased duration relative to other migraineurs. Males reported decreased duration of migraine relative to females during the posttraining period. Those migraineurs who reported being more relaxed during the final training session reported longer migraines during the posttraining period than other migraineurs. Migraineurs who rated higher expectation for success of biofeedback training before training began reported briefer migraines during the posttraining period.

Several predictors were notable by their absence from the package of important predictors for migraine measures. The importance of ROTTERIE was only noted in predicting posttraining medication. This predictor had a negative regression weight indicating that migraineurs scoring toward the external end on Rotter's scale reported lower use of medication during the posttraining period. TRNGRP was not a significant predictor for any migraine measure except frequency. Assignment to an increase group predicted report of lower posttraining frequency. PRCTC, which represented migraineurs' report of number of practice periods per week, was not a significant predictor for any measures. TEMPSES5 was an important predictor of migraine duration and medication, but was expected to be important for all four migraine measures.

Discussion

The present study examined the effects of skin temperature training and preheadache cue identification on management of migraine headaches. The first hypothesis tested the effect of feedback-trained finger temperature increases, as described by Sargent, Green and Walters (1972), on a package of four migraine measures. Groups which had demonstrated differences in temperature increase were not different when compared on the package of posttraining migraine measures. These results cast doubt on the importance of increasing skin temperature for migraine management by feedback training. The second hypothesis tested the incremental effect over thermal feedback training of preheadache cue identification on the same package of migraine measures. Interpretation of results of this nonsignificant test is confounded, and a more effective program for preheadache cue identification is suggested for further evaluation.

The following discussion begins with interpretation of results related to training control of skin temperature. Possible mechanisms of controlling temperature and the importance of motivation in training are considered. Control of migraine is next discussed with reference to placebo and specific effects. Finally, the results of four post hoc regression analyses of individual migraine measures are interpreted. These post hoc analyses provide additional information relevant to the first hypothesis, and to the management of migraine in general.

Control of Finger Temperature

The transfer of control of skin temperature to instances where feedback is not available is crucial for the assertion of effectiveness of the trained response in managing migraine. Early studies of thermal feedback training (Sargent et al., 1972; Andreychuk and Skriver, 1975; Wickramaskera, 1973) did not report skin temperature data to support their claims that migraineurs had learned the appropriate response. The failure to report skin temperature data weakens their conclusions about the effect of increasing skin temperature on migraine management.

In the present study, one group instructed to increase skin temperature demonstrated a decline in mean increase from a feedback period to a no-feedback period. This suggests that future studies should report temperature data collected during no-feedback periods as well as during feedback periods. Evidence that the "increase plus" group registered a significantly greater mean temperature increase over the "stabilize" group validates the comparison of these groups at posttraining to test effects of increasing skin temperature.

Mechanisms of temperature control. Information about effective mechanisms of temperature control may serve to improve control and enhance the clinical effect of training. Biofeedback theory holds that feedback of a specific physiological response trains the subject to discriminate a

specific interoceptive stimulus. Once isolated, desired changes in the stimulus may be reinforced (Miller, 1969). A major issue in feedback training of human subjects is whether changes in the target response associated with the interoceptive cue are directly or indirectly controlled by reinforcement. Those who contend that control is indirect claim that some other response is directly controlled which mediates changes in the target response (Katkin and Murray, 1968). The mechanisms by which migraineurs learned to increase their skin temperature were not experimentally examined in this study. However, the regression of predictors gathered before and after training onto temperature change provides relevant information.

One of two relevant predictors was a measure of ability to perceive changes in skin temperature while trying to control it. Presumably, this perception would be the interoceptive cue discriminated in feedback training. However, this variable was not a significant predictor of temperature change. This might be attributed to the fact that only four of twenty-nine migraineurs reported that they could consistently feel a change in temperature while trying to control the response. The self-report of relaxation was a significant predictor of increases in skin temperature. This indicates that migraineurs who report feeling relaxed also increase temperature during training. In so far as self-reported relaxation may serve as an index of reduced

somatic muscle tone, this finding suggests that somatic mediation might have been a component in migraineurs attempts to increase skin temperature.

The effect of cognitive mediation on temperature control was not evaluated. However, cognitive strategies were spontaneously described to the experimenter by most migraineurs. Strategies most often mentioned involved concentrating on warm imagery such as sun bathing at a beach, cooking near a hot oven, taking a sauna, or sitting near a hot fire. It is not known whether these strategies were correlated with temperature increase. However, it seems reasonable that subjects would mention successful strategies, and that cognitive mediation may be a component in control of skin temperature.

Motivation. The use of an operant paradigm requires that a reinforcer be contingent on demonstration of control of the target response, whether control is mediated (indirect) or not (Black, 1974). The reinforcer considered to control skin temperature was anticipation of migraine relief by appropriate temperature control. The importance of obtaining relief may vary across migraineurs. Shapiro and Schwartz (1972) reason that the more pain and suffering patients experience, the more motivated they will be to learn a biofeedback-trained response which they believe will help. It is interesting to note, therefore, that the group which had the most difficulty controlling temperature as instructed

was the "increase" group. This group also had migraines of significantly lower intensity than the other groups during the pretraining period. Intensity was considered by a pretraining poll to be the most valuable dimension of relief which might result from feedback training. The "increase" group also reported the lowest mean number of years since first contacting a physician about migraine. The "increase" group had the shortest history of migraine suffering and the least intense pain, and is considered to have been the least motivated to learn to control skin temperature.

No other reinforcement was contingent on control of skin temperature. Twenty-one migraineurs received noncontingent reinforcement by course credit for participation. The eight migraineurs not receiving credit may have been more motivated to learn temperature control; they also had a significantly higher mean expectation for training to have a beneficial effect on migraine. Of these eight migraineurs only one was in the "increase" group. This also suggests that motivation to learn to control skin temperature may have been lowest in the "increase" group.

The predictor accounting for the most variation in temperature increase over baseline was an indicator of familial migraine history. One possible interpretation of this variable which might account for this relationship, is as an index of motivation. Those migraineurs who reported having a family history of migraine may be more acutely aware of the

pain and suffering entailed. A post hoc inspection reveals that the "increase" group appears to be the least motivated based on this measure. Only two migraineurs reported a family history in the "increase" group, while eight in the "stabilize" group and seven in the "increase plus" group reported a family migraine history.

Most clinical reports of the effectiveness of skin temperature training on migraine make some implicit assumptions about training skin temperature. The first, and perhaps least tenable, is that several sessions of temperature training result in control as instructed (e.g. Andreychuk and Skriver, 1975; Blanchard, Theobald, Williamson, Silver, and Brown, 1978). The second is that, if significant control with feedback is demonstrated, this control will generalize to no-feedback conditions (e.g. Mullinix, Norton, Hack and Fishman, 1978; Turin and Johnson, 1976). Finally, all skin temperature training studies with migraineurs have assumed equal motivation to learn and use the temperature control response. Future studies should report evidence of temperature change recorded during periods with and without feedback, and should attempt to control for motivational variables.

Control of Migraine

The first step in any migraine intervention is reliable diagnosis. The high percent of agreement between physicians

and the experimenter insures that the general diagnosis of migraine was reliable. However the low level of agreement on the classic-common subclassification prevented the use of this potentially important distinction. Higher agreement on this subclassification might have been possible if the experimenter had given physicians a set of specific criteria for making the subdiagnosis. The distinction between pure migraine and migraine mixed with tension headaches was not made. According to Mitch, McGrady and Iannone (1976), this distinction may be relevant for predicting the effectiveness of skin temperature training.

Hypothesized effects. Posttraining differences between groups were tested after adjusting posttraining migraine measures for pretraining variation. Results reveal no multivariate differences between any two groups. These consistent findings, in spite of significant differences between groups similarly contrasted for temperature increase, suggest that increasing skin temperature is not more effective in controlling migraine than placebo. These findings support those of Andreychuk and Skriver (1975), Mullinix et al. (1978), and Blanchard et al. (1978).

Evidence supporting the specific effectiveness of trained temperature increases on migraine was found in two post hoc regression analyses. Session five temperature increase was a significant predictor of posttraining migraine duration and medication use. Increasing skin temperature

was correlated with lower duration and less medication. These two findings, while correlational in nature, suggest that increasing skin temperature may have a circumscribed specific effect on migraine. The finding that session five temperature increase was a significant predictor of the duration measure is supported by the significant univariate *F* for duration reported on the orthogonal contrast of the two increase groups. The "increase plus" group reported shorter mean duration after training; it also demonstrated a significantly greater mean temperature increase than the "increase" group.

The contrast of the two increase groups was intended to test the effect of preheadache cue identification. However, the failure of the "increase" group to demonstrate increases in skin temperature comparable to the "increase plus" group confounds interpretation of the effect. Preheadache cue identification was not adequately evaluated in this study. Cue identification began late in the course of the experiment in order to avoid its possible effect on learning to control skin temperature, and to minimize experimenter bias. As a result, migraineurs collected preheadache cue data during the posttraining collection of migraine measures. They had very little time to demonstrate possible effects of cue identification on migraine measures. The effect of cue identification would have been optimally tested if migraineurs had a chance to collect cue identification data and relate it to occurrence of migraine before the

posttraining period. The difference in mean posttraining duration between the two increase groups can be attributed to the difference in mean skin temperature increase rather than the effects of preheadache cue training.

Placebo effects. All groups showed significant improvement from pre to posttraining on the package of migraine measures. These results are in accord with similar findings by Andreychuk and Skriver (1975), Mullinix et al. (1978) and Blanchard et al. (1978). Each of these studies reported improvement for all treatment groups, with nonsignificant differences between placebo and experimental groups. Global improvement across all groups in the present study is not explained by the effect of increasing skin temperature.

There are several possible explanations for the improvement reported by all groups. These include learning a response the performance of which diverts attention from migraine pain, expectations for improvement, and motivational variables. Examples of trained responses which might compete with migraine include relaxation, alpha training or perhaps concentrating on performing a task which is believed to be beneficial. Expectation of beneficial results is considered to be a component of any therapeutic improvement (Shapiro, 1971), especially in the treatment of psychosomatic disorders (Lachman, 1972). Motivational variables indicative of desire to improve and willingness to take responsibility in one's own treatment are important in any biofeedback treatment.

Degree of relaxation is a factor in most feedback training procedures. The multiple regression analyses of migraine measures showed significant correlations of reported relaxation during the final session with higher levels of all migraine measures. A low reported degree of relaxation was a significant predictor of lower posttraining levels on each migraine measure. This suggests that migraineurs who are successful in managing their headaches may have learned a control procedure which included a subjective state of low relaxation.

Previous studies have reported the effects on migraine of relaxation trained by various methods. Mitchell and Mitchell (1971) and Blanchard et al. (1978) used progressive relaxation procedures to train migraineurs to relax. Both studies compared a group trained to relax with a no-treatment control. The Mitchell study found no difference in reported migraine relief, and the Blanchard study found a significant difference between the relaxation group and the no-treatment control. Wickramaskera (1973) used a single subject design for two migraineurs both trained first to reduce frontalis muscle tension with electromyogram (EMG) feedback and then to increase skin temperature with thermal feedback. Both subjects reported a slight improvement in migraine intensity during the EMG phase of treatment and significant reductions in intensity and duration during the thermal feedback phase.

None of these studies substantiated claims that migraineurs trained to relax actually did so during training. This failure to demonstrate relaxation may be due to problems in defining relaxation and measuring it. One way to measure relaxation would be to define it as reduced muscle tone, and report change in muscle action potentials. Measurement of action potentials has a reactive effect even if no feedback is provided. The subjective report of relaxation is an alternative measure of relaxation which may be easily determined with minimal reactive effect on training. Future studies of feedback training should continue to address the issue of the role of relaxation, measured objectively and subjectively, in migraine management.

Reports of migraine improvement from baseline levels have been reported for autogenic training (Shultz and Luthe, 1969), hypnosis (Graham, 1975), alpha training (Andreychuk and Skriver, 1975), desensitization (Mitchell and Mitchell, 1971), plethysmograph feedback training, and relaxation (Blanchard et al., 1978). Each of these training procedures may have provided migraineurs with a response presumed to be effective for obtaining migraine relief and which would divert attention from the pain of the migraine. The new response learned may not have had a specific effect on migraine mechanisms but may reduce perception of pain. Expectation for improvement and diversion of attention may also be effective elements for obtaining migraine relief in thermal feedback training.

Shapiro (1971) states that expectations for success of treatment have a nonspecific effect which may be neglected by clinicians. However patients' expectations for treatment effectiveness are subject to influence and may be altered to therapeutic ends. The failure of expectations for treatment success to significantly predict more than one migraine measure in the present study was due to small variation in the predictor. All migraineurs reported moderate or greater expectation for thermal feedback training to help their headaches. Although scaling and measurement of expectations are considered difficult, research in applied biofeedback training should continue to evaluate possible expectancy effects. One promising index of placebo expectations has been described by Stroebel and Glueck (1973).

Motivation to obtain relief is another nonspecific element in therapeutic improvement as well as in learning the feedback-trained response. Assumptions of equal motivation across patients may be untenable. Motivational variables are considered particularly relevant to therapies which rely on the patient assuming responsibility for his/her own training and treatment (Thorlsen and Mahoney, 1974). One index of motivation may be pretraining level of migraine measures; in this study, groups did not differ significantly in this respect. Other variables which may be related to motivation will be discussed in the following section. These include report of familial migraine history and

satisfaction with the effects of medication.

Predictors of Migraine Control

The post hoc multiple regression analyses of individual migraine measures provided useful information about prediction of success in the control of each measure with a temperature feedback training program. Two separate patterns of predictors emerged, one for migraine duration and one that was similar for the three remaining migraine measures. Two predictors were significant in predicting variation in all four measures: the reported ability to detect changes in skin temperature while trying to control it, and reported relaxation during the fifth training session. In addition, migraineur's gender predicted all measures of migraine except frequency.

The finding that reported ability to detect changes in skin temperature was a significant predictor of increased levels for each migraine measure is contradictory to what was expected. This might indicate that migraineurs who could not rely on interoceptive temperature cues were better able to control their migraines. These migraineurs presumably used other mechanisms acquired through training to control migraine.

The finding that males had lower average posttraining levels than females on all migraine measures except frequency was also unexpected. Clinical folk wisdom maintains that females are more suggestible and tend to respond better than

males to treatments with a placebo component. This correlational finding does not support those impressions. It is conceivable that males may try to endure migraines more than females by denial. This possibility might explain in part why migraineurs requesting help are predominantly female (Wilkinson, 1971).

There was a consistent relationship between reported degree of relaxation and each migraine measure. The direction of this relationship suggests that a subjective state of relaxation may not be a beneficial component in attempts to obtain migraine relief through feedback training. Perhaps a state of alert with concerted effort being put into use of a strategy to control headache might have an antagonistic effect on the migraine mechanism.

Familial migraine history was a significant predictor of posttraining migraine intensity and frequency. The standardized regression weight for this variable with both migraine measures was negative, indicating that the report by a migraineur of a family history of migraine was a positive prognostic for feedback training. The report of a family history is a strong validator of the diagnosis of migraine; these individuals might benefit more from a treatment presumed to be specific for migraine. It is also possible that the patient with a family history may have greater awareness of the degree of suffering and pain involved. This awareness may serve as motivation to avoid similar

suffering. Another possibility is that other family migraineurs might serve as models for effective control of migraine.

Four other predictors accounted significantly for variance in posttraining migraine duration. Increased skin temperature during session five predicted shorter migraine headaches and use of less medication. The significant prediction for both duration and medication is difficult to reconcile with findings of no significant multivariate differences between stabilize and increase groups. These divergent findings do suggest that seemingly conflicting results reported by previous investigators for the effectiveness of skin temperature training on migraine relief may have resulted from different measures used to gauge relief. For example, if assessment of relief were based exclusively on duration and medication, then findings might support the specific effect of skin temperature training. Other measures of migraine are perhaps less responsive to specific effects and more responsive to placebo effects. It is important to include more than measures of duration and medication alone in assessing improvement. Of the four measures used in this study, migraineurs rated intensity and frequency as the first and third most important dimensions of migraine relief.

Satisfaction with medication treatment was a significant predictor of posttraining duration and intensity. Migraineurs who were satisfied with their medication tended to report greater duration and intensity, as might be

anticipated due to lower motivation (Stroebel, 1975). Expectation for headache relief with biofeedback also was a significant predictor of posttraining migraine duration. As anticipated, migraineurs who reported higher expectations for success of training also reported migraines of shorter duration following training. Age was the final significant predictor included in the regression equation. As might be predicted from clinical impressions reported by Diamond (1975 a), older migraineurs reported longer migraine headaches. It was surprising however, that neither age nor expectation for relief predicted any other migraine measure.

Conclusion

Migraine improvement was found for all groups from pre to posttraining on all four migraine measures. This significant global improvement is interpreted as the result of a range of effects both specific and placebo. The effect of preheadache cue training was not adequately evaluated. Since it is being used by clinicians (Turin and Johnson, 1976), the effects of cue training merit further study. Generally, the effect of increasing skin temperature on migraine management was no better than placebo. However, larger skin temperature increases did predict briefer reported duration of migraine and use of less medication. Previous reports of migraine improvement attributed to placebo (Andreychuk and Skriver, 1975; Blanchard et al., 1978) or specific feedback training effects (Turin and Johnson, 1976;

Friar and Beatty, 1976) have used different dependent measures as criteria. Future evaluations of feedback effectiveness should document control of the target response in a no-feedback period, and should analyse migraine improvement on all relevant dimensions of migraine suffering. Clinical efforts to train patients to manage migraine should attempt to enhance both specific and placebo effects.

Review of the Literature

Introduction

Chronic headache is the most common complaint of patients suffering from a group of related symptom complexes diagnosed as migraine. Environmental or emotional precursors to attacks have led physicians (Birk, 1973; Stroebel, 1975) and psychologists (Miller, 1969; Lachman, 1972) to consider migraine a psychosomatic disorder. Others consider migraine to be an inherited organic disorder (Whitty, 1972) possibly precipitated by factors ranging from dietary indiscretion to climatic extremes.

The Research Group on Migraine and Headache of the World Federation of Neurology (1969) defines migraine as "a familial disorder characterized by recurrent attacks of headache widely variable in intensity, frequency and duration. Attacks are commonly unilateral and are usually associated with anorexia, nausea and vomiting. In some cases they are preceded by, or associated with neurological and mood disturbances." (p. 181) This definition has been adopted from the American Medical Association (AMA) Ad Hoc Committee on Classification of Headache (1962). The definition presents a moderate position on heredity, noting familial occurrence without specifying genetic transmission. It mentions accompanying emotional disturbances without specifying that they are possible precipitators or residual effects.

The recurrent, paroxysmal nature of migraine is widely recognized and important in differential diagnosis. Location and quality of the headache are also important in defining migraine and distinguishing it from other types of headache (Diamond, 1975a). According to Friedman (1968) Hippocrates designated unilateral headache as hemicrania from which stems our present use of the word migraine. The migraine headache is typically unilateral at onset but may become more generalized. Dalessio (1972) has noted that the sites of migraine headache are temporal, supraorbital, frontal, retrobulbar, parietal, postauricular and occipital. The headache may vary in duration from a few minutes to several weeks. Typically the headache is not severe enough to prevent sleep, which seems to be the state of optimal comfort for the patient. The quality of the headache is aching and throbbing early in its course, but it may become a steady ache with the passage of time. Intensity of the headache varies, but intensity is increased by walking, bodily effort, change in position, bright light, loud sounds and mental effort (Wolff, 1963).

Migraine symptoms secondary to the headache may precede or accompany it and suggest autonomic as well as central nervous system involvement. Prodromal symptoms are primarily visual, such as scotomata or hemianopia, and usually contralateral to the impending headache (Graham, 1966). Other prodromal symptoms may include sensory disturbances

such as unilateral paresthesias and speech disturbances. Other symptoms which may accompany the headache itself are nausea, vomiting, anorexia, constipation or diarrhea, photophobia and phonophobia, irritability or depression, vertigo, oliguria, excessive sweating and cold extremities.

Estimates of the prevalence of migraine based on percentage of migrainous patients encountered by a physician in general practice (Lennox, 1941; Diamond, 1975a) or more elaborate epidemiological studies (Waters and O'Connor, 1969; Dalsgaard-Nielsen, 1969) range from five to ten percent of the general population. Selby and Lance (1960) observe that of 500 migrainous patients seen, 60% were female, with age at onset of first attack between ten and forty years for 80% of the sample. Pearce (1971) and Wilkinson (1971) also report a slightly higher prevalence of migraine among women (66% and 64% respectively). Pearce's study supports Selby and Lance's findings for age at onset. Dalsgaard-Nielsen (1969) reports that his sample had a mean age at onset of ten years for females and fourteen years for males. Rees' (1974) epidemiological data suggest slightly higher mean ages at onset.

Various demographic and personality variables have been reported in clinical and case studies of migraine. Such efforts have sought to describe common characteristics of the migraine sufferer and have been widely accepted until hypotheses derived from impressionistic data have been

challenged by controlled study. Migraine has long been thought to be more common among the more intelligent, and among the higher social classes. These impressions were based on physicians' reports of migraine patients seeking treatment. Problems abound with the use of impressionistic data based on patient samples which may not represent the entire migraine population. Conceivably those patients who can afford treatment and who actually seek treatment are more wealthy and intelligent than those who cannot afford or who do not seek treatment.

Waters (1971a) sought to identify migraine sufferers in a sample of 1,718 adults who responded to a brief headache questionnaire mailed randomly to residents of a small city in Wales. The investigator then administered a group intelligence test to a migraine sample, two nonmigraine headache samples and a headache-free sample. Comparing pairs of groups, Waters found no evidence supporting the hypotheses that individuals with migraine had a higher mean intelligence than members of the other samples. A higher proportion of the more intelligent migraine sufferers reported consulting a physician about their headaches. Social class data was obtained by classifying stated occupation for all of the 160 men who took the intelligence test. There was no evidence of a higher proportion of individuals with migraine in the upper two classes relative to the no-headache group or the two nonmigraine headache groups.

Wilkinson (1971) reported a normal distribution across all social classes of migrainous patients seen at the City Migraine Clinic in London. Barolin (1972) in a review of psychological and demographic variables frequently attributed to migraine patients, concluded that migraine was not related to class or intelligence. The common feature among migraine patients was somatic, a constitutional factor of vaso-lability and autonomic irritability.

In 1937, Wolff described a constellation of personality characteristics which, though not specific or limited to migraine, were considered to dispose the individual to emotional reactivity which could precipitate attacks of migraine. He described a typical obsessional character: tense, driving, perfectionistic, inflexible and resentful of any alteration of plans. Feelings of resentment are infrequently expressed or resolved. According to Wolff, these attributes imposed difficulties in adaptation and a liability to react excessively to environmental demands or interpersonal problems. Fromm-Reichman (1937) described a migraine personality based on her treatment of migraine by psychoanalysis. She likewise believed the migraine sufferer to be obsessional, while considering his central conflict to involve the repression of anger. Expression of anger was alleged to produce intense feelings of guilt. Graham (1966) described the characteristics of a typical migraine patient as delicate, perfectionistic, intellectual and

overconscientious; a driving personality with a desire to please and be liked which compels him to take on more than he can do. He is unable to say no and tries to keep the peace at any price. The outward expression of emotion is taboo and "resentments are harbored in smouldering silence."

Friedman (1964) in his address as chairman to the AMA Section on Nervous and Mental Disease, declared there was no migraine personality. He then proceeded to list several characteristics, similar to those mentioned above, which he believed to apply in general to migraine patients. Henryk-Gutt and Rees (1973) performed a correlational analysis of responses to the Eysenck Personality Inventory (EPI), an abridged form of the Minnesota Multiphasic Personality Inventory (MMPI) and the Buss Durkee Hostility/ Guilt Inventory by a group of migraine sufferers and two matched control groups. They found statistically significant correlations between migraine sufferers and the following main items: an increase in the neuroticism score of the EPI; an increase in hostility scores on the Buss scale; increases in the anxiety and somatisation scores for women on the MMPI. Henryk-Gutt and Rees concluded that evidence of increased emotionality without indication of greater past or present emotional stress, when compared to controls, suggested an increased reactivity of the autonomic nervous system. Emotional stress was presented as a possible precipitating factor of the migraine attack. A follow-up

report by Rees (1974) emphasized that subjects' self-report of precipitating factors were most frequently "anxiety, overwork, and other forms of emotional reactions such as anger and resentment". During the two month observation period, fifty subjects reported 121 migraine attacks of which 64 attacks coincided with emotional stress.

Bihldorf, King and Parnes (1971) attempted to identify a migraine personality by comparing responses to their own adjective check list by a group of migraine patients with a group of tension headache patients and a no headache control group. Results showed that the groups differed significantly but migraine patients selected only four adjectives more frequently than the other two groups, of the 190 adjectives on the list. Other differences were reported between the two headache groups and the control group, but the paucity of statistically significant findings seems more important than the actual differences found in light of the expected type one error rate for 190 tests of significance.

In sum, reported evidence for the reliable identification of a migraine personality is inconclusive. If recurrent characteristic behavior patterns may be reliably identified, the ultimate purpose should be to improve diagnosis and/or treatment of migraine. Diagnosis of migraine is not difficult due to distinct physiological symptoms. Identification of recurrent precipitating factors

which may influence a characteristic emotional reactivity seems important to treatment and possibly prevention of migraine attacks. According to Bakal (1975) identification of clinically significant behavior patterns should be stressed over searching for statistically significant commonalities in character.

Heredity

Migraine sufferers commonly report that some other members of their family have similar headaches (Selby and Lance, 1960). Migraine has long been considered a familial syndrome (Dalessio, 1972) and some investigators have presented familial occurrences as evidence of its hereditary character (Pearce, 1969; Dalsgaard-Nielsen, 1965; Whitty, 1972). However, problems abound with inferences about heredity derived from patients' reports of family members with similar headaches. Diagnosis of migraine may be inconsistent at best if the physician relies on an intermediary to relay impressions about the symptoms of a third person. Granting that results gathered indirectly may provide useful information about familial occurrence, the explanation for these results may implicate heredity, environment or some combination.

Goodell, Lewontin and Wolff (1954) collected information about 119 migraine patients seen at a New York hospital on familial occurrence of migraine. Sixty-five of these patients were available at the time of study for a special

interview. Thirty-four relatives of this group were interviewed either in person or by phone in order to amplify information given by patients. The remaining 54 patients were contacted by mail and personal interviews were arranged with 25 of this group. Using information then from 59 interviews of relatives, 65 interviews with patients and correspondence with another 54 patients, the authors constructed pedigrees for 119 migraine patients making 654 indirect diagnoses. The authors found that 28.6% of those with neither parent affected had migraine, 44.2% of those with one migrainous parent had migraine and 69.2% of those with both parents affected had migraine. The authors concluded that it was reasonable to assume that migraine is due to a recessive gene with penetrance of approximately 70%.

Waters' (1971 a) epidemiological study included data on familial prevalence. Using 155 probands randomly selected from the general population, standardized headache questionnaires were administered to 519 of 669 first degree relatives over the age of 21. From these questionnaires each individual was classified as migraine, possible migraine, other headache or headache free. The families of the probands with migraine had a higher proportion of individuals with migraine than in the remaining three groups. The prevalence of migraine reported was 5% in the families of the "other headache" group, 6% in the families of the

"headache-free" group and 10% in the families of the probands with migraine. These differences were not significant.

In a subsequent comment on the familial prevalence of migraine, Waters (1971 b) criticized other studies of migraine prevalence. Prior to his study (1971 a) no studies were based on a direct assessment of the headaches of relatives of representative migraine sufferers selected randomly from the general population. Previous studies had not compared migraine family prevalence to migraine prevalence in a control group of relatives of no-headache probands selected from the same general population. Waters concluded that members of the same family may suffer from migraine but that heredity may be less important than is usually believed.

Lucas (1977) presents data from the first migraine twin study with adequate sample size, 86 monozygotic (MZ) and 75 dizygotic (DZ) pairs, to statistically investigate the importance of heredity in migraine. Migraine concordance for MZ twins was 26%; in DZ same sex twins 16%, and in DZ opposite sex twins, 13%. The difference between concordance for MZ pairs and DZ opposite sex pairs was the only statistically significant ($p < .05$) finding. The better matched comparison between MZ's and DZ same sex was not significant and environmental factors could explain the differences between MZ's and DZ opposite sex pairs. Lucas reasoned that if a strong genetic factor were operating,

concordant twins would share migraine headache characteristics and precipitants. However, in nine MZ twins and five DZ twins concordant for common migraine, no shared pattern was found for severity, laterality, time of onset, duration of attack or any of nine possible precipitants. Lucas concluded that his overall findings suggest a "much lower genetic factor" than previously thought.

Reconciliation of the importance of environmental and inherited factors has been advocated recently by Sacks (1970), Anthony and Lance (1972) and Lachman (1972). Anthony and Lance describe an inherited diathesis which they label as vascular instability. A dysfunction of control over blood vessel diameter is also indicated by studies of the mechanism of migraine attacks (Graham and Wolff, 1938; Appenzeller, Davison and Marshall, 1963). Inherited vascular instability plus the body's physiological response to certain environmental stimuli may produce the migraine attack. It is also possible that migraine represents several different disorders some of which are inherited and some of which are functional responses controlled by the environment.

Diagnosis

The AMA's classification system for headache (1962) lists fifteen different headache classifications. Under the classification of "Vascular Headaches of Migraine Type" are five different subclassifications: classic migraine; common migraine; cluster headache; "hemiplegic" migraine and "opthalmoplegic" migraine (elsewhere grouped together as complicated migraine); and "lower half" headache. The Research Group on Migraine and Headache of the World Federation of Neurology (1969) also classifies five similar subtypes of migraine but groups classical migraine and common migraine together as the conditions which are "generally accepted" within their stated definition of migraine (mentioned above). The other three subtypes are grouped together as conditions which "may fall within the category of migraine." Classical migraine is described by the AMA system as "Vascular headache with sharply defined, transient visual and other sensory or motor prodromes or both." Common migraine is described as "Vascular headache without striking prodromes and less often unilateral than (in classic migraine)."

Estimates of occurrences of classic and common migraine vary widely. Friedman (1976) states that 10% of all migraines are classic, 80% are common and the remaining three subclassifications comprise the last 10%. Dalessio (1972) offers a ratio of 9:1 for occurrence of common over

classic migraine. Wilkinson (1971) in her summary of data collected from 500 patients seen at the City Migraine Clinic in London, suggests that numbers of common and classic migraines were almost equal. These discrepant estimates of occurrence of migraine subclassifications indicate either that different populations were sampled or that reliability of the subclassifications is poor. No blinded studies of inter-rater reliability appear in the research literature.

It is necessary to differentially diagnose migraine from other disorders of known cause, the symptoms of which may mimic migraine. Diamond (1975 a) labels as traction headaches those which are symptoms with an identifiable organic cause such as brain tumor or other gross cell pathology. He emphasizes that treatment for traction headaches must be very different from typical migraine.

Another diagnostic problem which may complicate differentiation is that patients may suffer from different headaches at different times. They may tend to consider all their headaches as pains in the head and not recognize differences between headaches before and during the actual pain. Also, migraine symptoms may not recur together consistently across headaches. Vomiting may accompany one patient's migraine attack, and not be a complaint in the next attack. Finally, the prodrome which precedes classic migraine may resemble phenomena which may precede common

migraine. Friedman (1968, 1972) believes that the quality and reliability of occurrence of the classic prodrome do differ from the phenomena preceding an attack of common migraine.

According to Friedman, classic prodromes are sharply defined contralateral neurologic manifestations of a visual, sensory or motor nature. Disturbances in speech may also occur. These prodromes may appear separately or in sequence. The preheadache phenomena of common migraine may occur hours or even days before the headache. Symptoms of the common migraine prodrome may involve behavioral changes, disturbances in fluid balance and gastrointestinal complaints. The headache following these prodromata is usually longer in duration than the classic migraine and is less consistently unilateral. Although identifiable preheadache phenomena may obscure subclassification differences, there are important treatment implications which will be discussed later.

Carroll (1971) reports on the diagnosis of 200 patients referred to his migraine clinic in Guildford, England. Only 44% of the referrals were diagnosed as migraine. Twenty-nine percent were diagnosed as headaches caused by CNS pathology or insult; 12% were diagnosed as tension headache; 15% were considered to have headaches resulting from depression. Since treatment may differ greatly according to diagnosis, the author emphasizes the importance of careful classification.

Ziegler, Hassanein and Hassanein (1972) note disagreement among physicians in defining specific criteria for migraine. In order to clarify symptom constellations which might reliably be used to differentiate between types of headache, the authors report results of a principal components analysis of headache patients' responses to a headache questionnaire. Subjects were 289 headache-prone adults prescreened to rule out organicity. They answered 27 questions about their headache episodes and 12 questions about how their headaches responded to medication. Results of the principal components analysis in which axes were rotated to facilitate interpretation (not independence of factors) yielded seven factors accounting for about 90% of the variance. No single factor contained all of the symptoms considered characteristic of migraine. Classical symptoms of migraine were represented by three separate factors. The authors also noted that evidence of family history did not correlate with any of the seven derived headache factors.

Improvement of diagnostic reliability through the identification of homogenous symptom clusters is considered important for the validity of any classification (Zigler and Phillips, 1961). The validity of a diagnosis may be assessed on different dimensions. Kanfer and Saslow (1969) note three dimensions: etiology, course of illness and treatment. Since cause and course are considered

idiopathic for migraine, the most important dimension for assessing validity of the diagnosis of migraine is treatment. Before discussing treatment approaches, findings on the mechanism of migraine and inferences about possible causes will be considered.

Mechanism

In 1938, Graham and Wolff reported results of experimental intravenous injections of ergotamine tartrate to 22 subjects during 52 different attacks of migraine head pain. They noted a marked decrease in the amplitude of pulsations of the temporal or occipital branches of the external (extracranial) carotid artery. This decrease coincided with the subjects' report of decreased intensity of headache. The authors proposed that the known action of ergotamine tartrate on smooth muscle had a vasoconstrictive effect on the extracranial arteries observed. Similar observations on a group of 34 control subjects also produced a similar reduction in amplitude of arterial pulsation. The ameliorative effect on headache subjects suggest that head pain is related to vasodilation of extracranial arteries. Wolff (1963) notes that other physiological changes follow the injection of ergotamine tartrate in headache patients and normals. These include a slowing of pulse rate, a slight rise in blood pressure and increased cerebrospinal fluid pressure.

Ostfeld and Wolff (1957) in their study of migraine

prodrome, provided evidence that the mechanism of prodrome is linked to intracranial artery vasoconstriction. They demonstrated that:

1) Preheadache scotomata were transiently eliminated by inhalation of amyl nitrate or carbon dioxide, both known cerebral vasodilators;

2) Intravenous administration of levarterenol, an agent having vasoconstriction as its sole significant effect, induced scotomata;

3) Cranial artery constriction, measured by plethysmograph, occurred during the appearance of scotomata.

Skinhoj and Paulson (1969) confirmed these findings by measurement of regional blood flow and angiography of a subject during the preheadache period. They suggested that observed increase in vascular resistance is secondary to some other unknown mechanism.

General agreement is noted (Friedman, 1972; Lance, 1969; Dalessio, 1966) that intracranial vasoconstriction produces the preheadache phenomena and decreased blood flow. This is followed by a "rebound" vasodilation, increased blood flow to cranial arteries and headache. The development of characteristic head pain is due to dilation of the extracranial arteries plus a local accumulation of pain producing or pain threshold-lowering compounds within and in the vicinity of the vessel walls (Heyck, 1969). These compounds are kinins with a hormone-like vasodilator effect.

Researchers believe that they are either liberated locally by neurogenic control (Heyck, 1969; Friedman, 1966 a) or are delivered in the blood (Appenzeller, 1969; Lance, 1969). According to Dalessio (1966, 1972) a local sterile inflammatory reaction about the vessel wall with accompanying edema follows a period of vasodilation. The increased pressure on the inflamed vessel walls coupled with an apparent drop in pain threshold contribute to the sensation of pain transmitted neurally to the cortex.

Since vascular changes are implicated, closer inspection of vasomotor regulation is warranted. According to Forsyth (1974) changes in the radius of blood vessels are controlled locally by three mechanisms. Myogenic activity of the smooth muscle surrounding the vessel; vasodilator metabolites; and sympathetic innervation of the smooth muscle may each affect the radius of a vessel. The normal homeostatic mechanism at the local level autoregulates blood flow in the following way. When blood pressure or flow is decreased arteriolar smooth muscle is stretched and exerts decreased tone. Lower pressure in tissue capillaries promotes decreased filtration of fluid into the tissues, increasing blood volume, cardiac output and blood pressure. Low arterial pressure in the kidney decreases the glomerular filtration rate and the production of urine which also increases blood volume.

Remote control of vessel radius may be hormonal, which

is slower acting but has more lasting effects; or neurogenic by the baroreceptor reflex. The carotid baroreceptor reflex is considered by Forsyth "the most powerful and rapidly acting homeostatic mechanism in the cardiovascular system." Afferent nerve endings which are sensitive to distortion or stretch travel to the vasomotor center via the carotid sinus nerve to the medulla. The efferent arm of the reflex, the vagus nerve, alters heart rate which influences blood flow and pressure. Forsyth states that different tissues have different densities of sympathetic innervation as well as different combinations of receptors. Thus, although the sympathetic nervous system discharges en masse, it has different actions on different organs.

In discussing the autoregulatory mechanisms of cerebral circulation, Symon, Bull, duBoulay, Marshall and Russell (1972) suggest that the role of the sympathetic nervous system in vascular control indicates a close link between psychological influences and cerebral circulation. Stroebel (1975) explains the effects of emotional arousal on the vascular system via the sympathetic nervous system as part of the body's defence reaction to stress. Blood supply is shifted to important fight/flight organs by the sympathetic nervous system. Stroebel suggests that the body's "red alert emotional response" is adaptive in life-threatening situations, but claims that "the pressure of a Western life style" has played a trick on man. He believes that

inappropriate learned fears or bottled up anger may now also trigger the "red alert emotional response" to non-life-threatening stimuli or even to imagined stimuli.

Dalessio (1972) has summarized evidence that people subject to migraine exhibit significantly greater variability in cranial artery radii than people not subject to migraine. Such variability during headache-free phases was especially evident during life periods marred by frequent and severe attacks.

> The striking manifestations in cranial artery function characteristic of the headache attack merely punctuated the more or less continuous series of physiological changes that comprised part of the particular life adjustment of these persons. The observed modifications in cranial artery function were accompanied by mood alteration, feelings of tension, sustained effort and restlessness. The concurrence of these changes suggests that these modifications in vascular function and structure are the sequel of sustained adaptive reactions to life stress. (Dalessio, 1972, pp. 267-268).

The possibility that external stimuli initiate a chain of interdependent physiological mechanisms which result in migraine suggests that the headache is not attributable to tissue pathology. Lance (1969) rules out neuropathy.

"There is no convincing evidence at present that positive neural control of blood vessels is impaired in migraine, that migraine is caused by an abnormal neural discharge, or that operation on nerve pathways will prevent migraine." Recent migraine mechanism research has investigated biochemical and metabolic factors which might account for vascular changes producing migraine.

Substances which control vascular tone and could possibly be involved in the biochemical process of migraine include serotonin, reserpine, acetylcholine, histamine, neurokinin and bradykinin (Friedman, 1966 a). In 1972 Lance and Anthony presented a similar list including serotonin, histamine and bradykinin with the additional implication of catecholamines (adrenaline and noradrenaline), tyramine and prostaglandins. Most of these agents are considered to have an effect either insufficient to initiate headache alone or secondary to the action of serotonin.

> The fall of plasma serotonin, which we have found at the onset of the migraine attack, appears to be specific and not simply a reaction to headache, vomiting or simple stress. Migraine is precipitated when serotonin levels are lowered artificially by injection of reserpine and is ameliorated by the injection of serotonin. (Anthony, Hinterberger and Lance, 1969)

The resulting conceptualization of migraine mechanism

recognizes cerebral vasospasm at one level of explanation. The mechanism of the vasospasm itself is less certain. (Friedman, Wood, Rowan & Frezier, 1972). Tissue pathology is not directly implicated, though some authors suggest that a vascular instability may be inherited. Neurogenic biochemical factors with mutually interactive effects may control changes in the radii of various blood vessels and/or lower pain threshold. The mechanism of these biochemical factors, when and how they contribute to migraine, is also not certain (Friedman, 1976). It is possible that these factors may be influenced by some endogenous dysfunction; the ingestion of compounds similar to those occurring naturally in the body or by the body's response to some environmental stimulus.

Treatment

According to Friedman (1976) there is no wholly effective treatment, pharmacologic or psychologic, that changes the natural course of migraine. Treatment of migraine to date has been limited to symptomatic relief of headache or interval treatment intended to prevent symptom recurrence. Complete remission of all symptoms due to external intervention is not documented. Treatment of choice for the individual sufferer depends on successful diagnosis and the patient's response to alternative treatments. The effectiveness and cost to the patient is considered for each treatment available relative to

alternatives. By choosing appropriate treatment in this manner, the incremental validity of the treatment is assured. Incremental effectiveness of any treatment may be demonstrated by comparing the results of a given treatment to a no treatment control, to alternative active treatments or to a nonspecific placebo control. Demonstration of incremental effectiveness over placebo control is considered necessary to the evaluation of treatment effectiveness of psychosomatic disorders (Mason, 1971; Shapiro, 1964; Waters, 1970; Lachman, 1972).

Two broad classes of treatment will be reviewed: medical and nonmedical. Particular emphasis will be placed on thermal biofeedback, and on other modes of treatment where research reports incremental effectiveness over placebo controls. Medical treatment is considered to be any attempt to alleviate symptom severity or frequency by administration of pharmacologic agents to the body of the sufferer, or alteration of diet. Nonmedical intervention is considered to be any attempt to alleviate symptom severity or frequency by intervention which does not involve administration of such agents or diet change.

Medical Treatment

Three general classes of drugs are prescribed by physicians for treatment of migraine: drugs which affect the vascular mechanism of migraine, those which may alleviate secondary symptoms such as pain or nausea, and drugs

considered ancillary for direct symptom relief such as tranquilizers and soporifics. Since the headache is caused by dilation and distension of extracranial arteries, agents with a known vasoconstrictor effect have been administered.

According to Dalessio (1972) the administration of fluid extract of ergot has been known to terminate migraine headache for at least half a century. The known vasoconstrictive action of ergotamine tartrate is considered to be the best pharmacologic treatment for severe migraine (Sacks, 1970; Friedman, 1976; Ostfeld and Wolff, 1958). It is most effectively administered intramuscularly (Wolff, 1963) and as soon as possible after signs of headache appear (Sacks, 1970).

Edmeads (1971), a practicing neurologist, warns that all ergotamine compounds can be dangerous in oral doses beyond six mg. per day or ten mg. per week. Toxic side effects include nausea, vomiting (exacerbating existing secondary symptoms), ischemia of the extremities, cardiac arrhythmias, aching muscles, paraesthesias, angina pectoris and thrombophlebitis (Friedman, 1968).

Contraindications for the use of ergotamine compounds include infectious states, vascular diseases, coronary scleroses and a history of anginal pain, pregnancy, renal disease, and severe vitamin deficiencies (Dalessio, 1972). Since ergot also has a central stimulation effect,

habituation, dependence and withdrawal can occur (Friedman, 1968). Friedman (1964) also noted that patients on continuous ergotamine therapy may have to increase the daily dosage to secure relief, and that in so doing there is an increase in the frequency of attacks. Dalessio (1972) labels abuse of the drug ergotism. He claims that it is uncommon but, "The clinical picture of ergotism is dramatic and terrifying. First there is a vigorous vomiting, then the extremities, usually the feet, become pulseless, and swell with congestion and cyanosis. Ultimately gangrene develops. Jaundice may also occur." (p. 404).

Ergotamine tartrate has gained widespread acceptance among physicians presumably for its effectiveness in terminating attacks. Ostfeld and Wolff (1958) report that it will alleviate head pain about 75% of the time if taken early enough in the course of an attack. If taken too late to be helpful, they recommend drugs with strong analgesic effect such as codeine or demerol. Maxwell (1966) believes strongly enough in the reliable effect of ergotamine tartrate to suggest that response to the drug may be used for diagnostic purposes.

Waters (1970) as a prelude to his epidemiological study of migraine, compared response to ergotamine tartrate with placebo (lactose) in 79 women, 67 of whom were diagnosed as migrainous. This study was double blinded with a cross over design so that each subject received both

ergotamine tartrate treatment and placebo separately without knowing which pills were which. There was no evidence that ergotamine tartrate in doses of two to three mg orally was more effective than the placebo. Ergotamine tartrate, however, aggravated the attack significantly more than placebo.

This study has been criticized for the administration of homeopathic dosages and for being too short to allow all clients to respond effectively to ergotamine (eight weeks for drug treatment and eight weeks for placebo). But, results of this well controlled study suggest that response to ergotamine should not be considered for diagnostic purposes and that ergotamine tartrate may be less incrementally effective over placebo than previously assumed.

Another vasoconstrictor with diuretic and stimulant effects, caffeine, has been used for three centuries (Sacks, 1970) for migraine treatment. Sacks recommends that it be taken copiously early in the attack. Dalessio (1972) describes its usefulness as a powerful potentiator of ergotamine, to be used in combination. Friedman (1976) supports its usefulness with ergotamine because it allows the dosage of ergotamine to be reduced. Caffeine is not considered by Friedman or Dalessio to be adequate treatment for migraine if used alone.

Methysergide is also used for its vasoconstrictive effect on scalp arteries. The mechanism of this drug is

through antagonizing the serotonin effects on some organ systems while simulating serotonin in maintaining scalp artery constriction. (Anthony and Lance, 1972). Although it is not effective once the headache has begun, it is considered the most effective drug for migraine prevention, producing an overall decrease in headache frequency in 60% of patients (Friedman, 1973). However, according to Curran, Hinterberger and Lance (1967), side effects are experienced by about 40% of all patients while 10% of all patients cannot tolerate the drug. Side effects and contraindications are similar to those of ergotamine tartrate. Discontinuation of methysergide is recommended for two months of every six months of treatment to minimize possible complications (Friedman, 1968, 1976).

Agents which raise pain threshold may be effective in alleviating the secondary symptom of head pain. Salicylates; non-narcotic analgesics such as Darvon and aspirin; and narcotic analgesics such as codeine or morphine are listed by Friedman (1966 b, 1976) as potentially effective with less severe head pain or when ergotamine preparations are ineffective. These agents are not migraine specific and are used widely for general pain alleviation. Maxwell (1966) has recommended diuretics to minimize fluid retention and decrease blood volume. Diuretics are not considered effective when used alone (Friedman, 1964) but may affect severity or frequency. Anti-emetics may also be

prescribed to treat the secondary symptoms of nausea and vomiting.

The third class of drugs used for migraine treatment are tranquilizers or anti-depressants which influence the patient's mood and response to stress (Friedman, 1973). However, Dalessio (1972) criticizes the sole use of sedatives because they can be addicting, have other toxic side effects, and fail to influence any underlying basis of conflict or tension. Friedman and Merritt (1957) found tranquilizers to yield results comparable to placebo in migraine treatment.

Dietary factors which may precipitate headaches are reviewed by Dalessio (1972). He states that foods containing tyramine may trigger vasoconstriction with a subsequent rebound vasodilation of scalp arteries. Tyramine is found in significant amounts in cheese, fish, beans and dairy produce. Other dietary restrictions have been designed to have a diuretic effect, or to eliminate general vasodilation due to alcohol consumption. Dalessio concludes that:

> To be sure, the migraine subjects like any other being may feel more comfortable on this or that diet; but food, per se, either through its effect on intestinal stasis, fluid retention, or by virtue of allergic or sensitivity effects is probably of little importance, except as noted above (concerning tyramine). (1972, p. 410)

In sum, the most effective medical treatments for migraine have been with ergotamine tartrate for relief of headache and methysergide for prevention. These drugs do not effect a cure for the disorder, but may be effective in reducing severity, duration or frequency of attacks. Other nonspecific drugs may help to alleviate symptoms accompanying or produced by change in the cranial vasculature. Side effects and contraindications of the various drugs mentioned limit the number of sufferers who may find relief pharmacologically. Medical research continues to seek more effective drugs for treatment of migraine. Many physicians (Friedman, 1973; Edmeads, 1971; Wolff, 1963; Sacks, 1970) suggest that a consideration of psychological variables and treatment must accompany medical treatment to maximize effectiveness:

> Of more importance to the patient than awareness of immediate precipitating events is a broader understanding of factors in his personality and life situation that persist and precede the headache....The patient should be made to appreciate from the beginning that there is no easy road to the goal he wishes to achieve, and especially must he appreciate that anything out of a bottle can offer him no more than transient help.
> (Dalessio, 1972, p. 418)

Alternatives other than Biofeedback to Medical Treatment

Various psychoanalytic, psychotherapeutic, psychological and behavioral treatments of migraine have been reported. Most reports are case studies with inadequate controls and specificity in accounting for results. Studies reviewed will be separated into three categories: psychotherapeutic treatment (including psychoanalytic), hypnosis (including suggestion and autogenic training or self-hypnosis), and behavioral treatments. Caution is advised in comparing treatment effectiveness across reports using different diagnostic criteria for migraine and different dependent measures. Inadequate controls do not allow the reader to clearly discern the cause of observed change or to attribute treatment success to active or nonspecific (placebo) effects.

In 1937 Fromm-Reichman reported on the effects of her psychoanalysis of eight migrainous patients. She deemed five to have become "practically headache free"; two found "decided" relief as to the frequency and intensity of their attacks; and one remained unchanged. Despite many unanswered questions in this collection of case studies, it is important to note that insight therapy (possibly accompanied by nonspecific effects) emphasizing the acknowledgement and expression of anger may have a beneficial effect on migraine. Cost in terms of time and financial expense, and failure to replicate such success in a subsequent effort

reported by Dalessio (1972) rule psychoanalysis out as an incrementally valid treatment for migraine.

Hunter and Ross (1960) report observations of the effects of psychotherapy on 35 patients attending a neurological clinic who showed little or no response to drugs. A psychiatrist took a detailed history of migraine paying special attention to onset and times of exacerbation. Patients were then invited to talk about themselves freely, the psychiatrist allowing the interview to be led by the patient. When it became clear that certain events or certain persons were associated with exacerbations, the patients were encouraged to discuss these events and their emotional reactions to them either later in the same interview or as the starting point of subsequent interviews.

Results of this uncontrolled study were based on psychiatrists' assessment of patient's report of change in frequency, duration, severity of headache; incidence of secondary symptoms; diminution of drug intake and incapacity during attacks. Forty-five per cent of the patients were considered to have improved mildly to moderately and 49% markedly. The authors state that improvement was not related to new or additional drug use since no drugs were prescribed.

> Indeed, the best results were obtained in those who finally took minimal or no drugs at all, even during attacks....It appeared that excess drugs

> were making some patients worse....In some patients drug taking had become a habit, even to the point of addiction, and migrainous symptoms became mixed up with the side effects of drugs. In many patients a reduction in total drug intake resulted in drugs that had ceased to control migraine attacks regaining their earlier therapeutic effect. (Hunter and Ross, 1960)

It is again difficult to judge what was responsible for the reported success, as the authors acknowledge. Their brief description of treatment format is commendable and suggests that the association of environmental events or emotional reactions to such events with headache occurrence might be a possible active treatment factor for controlled study.

Gentry (1973) reports a successful case study using directive therapy, patterned after the work of Milton Erickson and Jay Haley. Gentry describes directive therapy as a communication-oriented approach to treatment of symptoms which postulates that psychopathology is produced by the patient's attempt to gain control of an interpersonal relationship. The relationship between therapist and patient must be controlled by the therapist so that the patient's control is averted and her (in this case a 26-year-old housewife) symptoms are not reinforced and

perpetuated. Therapy, as described by Gentry, focused upon the patient's present circumstances and the functional value of the patient's symptoms. The therapist made no effort to elicit or explore childhood experiences.

In order to assume control of the relationship, Gentry directed that the patient perform a prescribed task considered by her to be particularly unpleasant. "Giving Melody (patient) a specific task to do while having her headache placed the symptom under the therapist's control." The aversive nature of the task, working on the weekly family budget, was supposed to be adequately self-punitive as to supplant the same punitive function served by the migraine attack. Total symptom remission was reported after ten weekly one-hour sessions.

Much speculation is possible in attributing cause for success to a specific factor or set of factors. The factors reported by Gentry are described in terms of a directive therapy paradigm. But the patient-therapist relationship may have been important only because of its similarity to the patient's relationship to her husband over whom she was theoretically trying to establish control by symptom formation. In other words, the importance of establishing a transference might be postulated. It is also possible that the homework assigned Melody may have been a factor in symptom alleviation. The prescription by a doctor of some task (presumed by the patient to be therapeutic) to

do while having an attack might have a placebo effect. Or the task, since it was chosen to be aversive, might have effectively punished the patient's symptomatic behavior.

The psychotherapeutic case studies reviewed above leave many questions about relative treatment effectiveness unanswered. Fromm-Reichman (1937) suggests that insight into the patient's anger has a specific effect on migraine alleviation. The work of Hunter and Ross (1960) indicates that frequency and severity of attacks is influenced by psychological factors. Their successes were attributed to the patient's guided effort to specify the discriminative stimuli which preceded individual attacks. The Gentry (1973) case study suggests that the patient-therapist relationship, some nonspecific effects, homework concurrent with therapy, or a symptom punishment paradigm may be effective in treating the symptoms of migraine.

Schultz and Luthe (1969) describe a treatment approach which has been applied to a range of medical and psychologic disorders. Known as "autogenic training", this treatment method involves a psychophysiological orientation to the simultaneous regulation of mental and somatic functioning. The desired somatic responses are considered to be effected by "passive concentration" on preselected word phrases. The "somatic responses" to be controlled in migraine treatment are heaviness in the limbs, warmth in the extremities, heart rate, warmth in the abdomen and cooling of the

forehead. The authors report that the majority of patients treated experienced fewer headaches, and of decreased intensity. They note that periodic attacks can be intercepted before onset by initiating autogenic exercises as soon as prodromal symptoms develop.

Schultz and Luthe's impressionistic reports of success do not qualify as strong evidence favoring the treatment effectiveness of autogenic training. However, speculation about the active elements of this method has influenced subsequent treatment efforts. Possible ingredients in autogenic training which may account for migraine relief are: trained relaxation which is instituted as a response antagonistic to stress reaction; homework practice effects; learning of alternate ways to react to stress; attention placebo perhaps manifested as concern for the patient by a knowledgeable and presumably competent therapist.

Graham (1975) treated two patients with clinical migraine histories of ten and fifteen years, using autogenic training for hand warming and hypnosis. One month of baseline data was collected on frequency, duration and intensity of headaches. Each patient was treated twice weekly for five weeks. Treatment consisted of hypnosis followed by hand-warming suggestions modeled after Sargent, Green and Walter's (1973) adaptation of autogenic training. Patients were instructed in self-hypnosis during the second session and told to practice hand warming daily. During

the final session a list of premigraine cues was compiled and discussed with each patient. The importance of implementing the technique during the preheadache phase was stressed.

Results reported one month after treatment shows one patient headache free and the others' headaches reduced in frequency, duration and intensity below baseline levels. The author concludes that hand-warming training and self-hypnosis were "clearly effective" in treating migraine in these two cases. But results may also be explained in terms of learning new behaviors antagonistic to headache such as relaxation, a nonspecific practice effect, or an attention placebo. The final session in which prodromal cues were discussed may also have been an important ingredient in treatment.

Andreychuk and Skriver (1975) used a factorial design to investigate the effects of three treatments including self-hypnosis, biofeedback training for hand warming and biofeedback training for alpha enhancement. A median split of subjects' scores on a hypnotic induction profile was used to define a dimension of susceptibility to hypnosis. This crossed factor was used as a measure of patient suggestibility, considered by the investigators to be a significant subject variable in the placebo effect. The hypnosis group was considered to be a nonbiofeedback control group, and the alpha feedback was chosen as a form of biofeedback with no known relation to migraine, the effects of which

were considered nonspecific.

Thirty-three subjects kept six weeks of pretreatment headache data for establishing a baseline, continuing to record the same information during the final five weeks of treatment. Each of ten treatment sessions lasted about 45 minutes and were administered in a "pleasant, 4 x 8 foot, sound attenuating cubicle", using "very dim, indirect lighting". Results show all three groups improved significantly over baseline with hand-warming training having the greatest effect on percent improved, alpha training next and hypnosis having the least effect. Differences between the three treatment groups were not significant, but differences in symptom alleviation between the high hypnotizable and low hypnotizable groups were significant.

The investigators conclude that more suggestible subjects are apt to respond more favorably to treatment situations where they have high expectations of being helped than are less suggestible subjects. Andreychuk and Skriver attribute this difference to the placebo effect. The difference between treatment groups is explained as a statistical artifact of unbalanced assignment of high and low hypnotizable subjects across each treatment group. The treatment group exhibiting the most improvement (hand-warming biofeedback) also had the largest number of subjects with high hypnotizable scores. "In short, the particular biofeedback treatments were not necessarily the relevant

variables in producing these effects."

The investigators conclude that suggestibility, considered as placebo, was the primary variable influencing the results. However, two experimental problems are apparent with the study while an alternative explanation might serve to account for results obtained. Disproportionate assignment across three treatment cells leaves results less interpretable than if subjects were assigned to equate high hypnotizables and low hypnotizables in each treatment group. No evidence was presented to substantiate that biofeedback groups had actually learned to control hand warming and alpha production respectively. If these groups did not demonstrate acquisition of trained behaviors, a placebo effect might be a more satisfactory explanation for treatment effects. An alternative explanation for the observed effects of treatment, as the investigators acknowledge, is learning a relaxation response to stress or possibly counterconditioning stress by relaxation.

In a case study, Lutker (1971) examined the effects of relaxation with training in awareness of preheadache cues on a 22 year old migrainous student. Treatment consisted of three stages. Muscular relaxation was trained using audio taped instructions administered in four hourly sessions over two weeks plus nightly practice without tape. Next, preheadache signs were identified and the patient was instructed to relax whenever she recognized these signs.

Finally the patient was instructed to think about the relaxed feeling state briefly after recognition of preheadache signs. No headache frequency data is reported, but Lutker hypothesizes that during the third stage the relaxation response became conditioned to even the slightest recognition of preheadache symptoms. He suggests further that the relaxation response substituted for the previous headache symptom in the patient's response repertoire. The author concludes that: "The attachment of a non-harmful and socially acceptable psychophysiological response to a stimulus which previously elicited a maladaptive response would seem to be a relatively simple and possibly quite effective procedure."

Mitchell and Mitchell (1971) report the results of two migraine treatment studies using behavioral approaches. In the first study 17 volunteers from a university population were assigned to three groups: a no-treatment control; progressive relaxation training; and a combined desensitization package of simultaneous application of relaxation, desensitization and assertive therapy. Baseline data was recorded for eight weeks prior to treatment and treatment consisted of fifteen hourly sessions given at a rate of two per week. The initial session involved presentation of a standardized explanation of migraine as a psychosomatic disease with learned psychological mechanisms. At the second session all subjects completed a pretreatment test

battery designed to assess general anxiety, interpersonal anxiety and distressing behaviors related to sex, family life, vocation, religion, etc. Group-specific treatment followed. Posttreatment measures of headache symptoms and battery measures were taken at termination and at a nine week follow-up.

Results of the first study indicated a significant effect of type of treatment as measured by headache frequency and duration. Subjects in the combined desensitization group reduced frequency and duration of headaches significantly below the no-treatment group, whereas changes between relaxation and no-treatment groups were not significant. The investigators conclude that relaxation treatment failed because it did not provide subjects with any method of controlling their environments. It should be noted, however, that relaxation might provide an adequate way of controlling the subjects' response to the environment providing sufficient instruction in application of the procedure accompanies training. Obtained results in both studies may be questionable because group sizes were unequal and mean pretreatment dependent measures were different across groups. Consequently, decreases in headache measures in extreme groups from preheadache measures might be partly explained as a regression effect.

The second study assigned 19 university subjects to four groups: no treatment, desensitization, combined

desensitization with previous pharmacotherapy treatment, and combined desensitization without previous pharmacotherapy. Procedures for the no-treatment and combined desensitization groups were comparable to the first study. The desensitization group received relaxation training and systematic desensitization. All treatments were again hourly and limited to 15 sessions. Results of between-group comparisons showed that both combined desensitization groups had improved significantly over the desensitization and no-treatment groups for headache frequency. No significant differences were found between the no-treatment and desensitization groups.

The investigators consider the restricted improvement gained by using a single treatment model as evidence that single model procedures are only partly effective when applied to relatively complex behavior problems. They believe that the success of the combined desensitization groups was due to inclusion of conditioning procedure (desensitization) and re-educative and environmental manipulative procedures. Unfortunately, both studies failed to experimentally isolate separate effects of different procedures and conclusions cannot be made about the most effective package.

Mitchell and Mitchell include a theoretical discourse on migraine etiology including a rationale for use of the treatments investigated. They state:

> Migraine is a symptom representing the interactive effect of constricted overt emotional expression and chronic covert emotional over-reactivity, with its somatic concomitants, excessive sympathetic nervous system activity, manifested by hypersensitive cranial arteries....Treatment consequently took the form of 'detaching' or reducing the level of disruptive and chronic anxiety-laden emotionality associated with general and specific environmental situations. That is, the treatment aimed at increasing the migraine individual's capacity to control emotional reactivity to events perceived as stressful in his environment, thus stabilizing sympathetic nervous system activity and reducing its 'triggering' effect upon cranial arteries. (Mitchell and Mitchell, 1971)

Lambley (1976) reports the case of a 38-year-old woman who had suffered migraine attacks for 22 years. Medication had provided some relief in the past, but the patient noted a recent decrease in effectiveness. Baseline headache data on frequency and severity was recorded for one month prior to treatment. After an initial interview, the therapist hypothesized that the patient had avoided recognition and expression of anger because she had never been taught to express herself assertively and because of punishment previously afforded such expression.

The first treatment phase consisted of assertive training and behavior rehearsal "to enable her anger and related feelings to be expressed in appropriate contexts." The patient was thus taught what to do when confronted with conflict between anger and anxiety over expressing it. The next phase of therapy was designed to develop her insights into the reasons for this conflict. The psychodynamic stance together with assertive training were credited for reducing headache frequency to nil after eleven weekly treatment sessions.

This study was not controlled or systematic and conclusions about specific mechanisms of change must be considered speculative. This case study does offer some evidence for the effect of a treatment which does not directly treat the headache symptoms. Instead treatment focused on antecedent psychological contexts which may precipitate the headache, and learning more appropriate responses for use in these contexts.

Each of the behavioral treatment studies mentioned thus far has relied on patients' self-recorded headache data to chart or measure change. Recent publications (Kazdin, 1974; Thoresen and Mahoney, 1974) have suggested that self-recording or self-monitoring may have a reactive effect on subjects and be responsible, in part, for behavior change. Mitchell and White (1977) attempted to evaluate the specific effects of self-recording of headache

data and self-monitoring preheadache stress cues as treatment for migraine.

Twelve subjects from a university population were assigned to four groups: self-recording of headache frequency only; self-monitoring of preheadache stress cues only; self-monitoring in combination with training in physical and mental relaxation and self-desensitization; and all elements of the previous group plus "13 further self-change techniques." All subjects were seen on a group basis with group membership reducing from twelve to nine to six to three as the four groups completed, at different intervals, their training. Training in phases three and four consisted of one group session with audio taped instructions and assigned homework with additional audio tapes.

Results indicate that neither training in self-recording alone nor self-monitoring alone effected reductions in migraine frequency. There were no significant differences between the four groups through the first two training stages or between the self-recording and self-recording plus monitoring groups at treatment end (week 48) and at follow up (week 60). Significant reductions were noted in headache frequency only after the third phase of training, including relaxation and self-desensitization, began. The two groups having received this third phase differed significantly from the other two groups, but not from each other.

The only group receiving the fourth phase of training differed significantly from the remaining three after the final training phase began, and at follow up.

The authors conclude that neither self-recording of migraine symptoms nor self-monitoring of preheadache stress cues are sufficient to effect a substantial reduction in headache frequency. This finding seems firmly supported, but the specific effects of the other "self-change" training procedures remains questionable. Duration of training, additional homework effects, some possible effect of group meetings or placebo are not excluded as possible alternative explanations for observed changes. Mitchell has again used a "shot gun" behavioral approach to training more appropriate responses to the environment in order to avoid hypothesized migraine precipitants. After adequately assessing the effects of self-recording/self-monitoring, this study failed to gauge the effects of separate elements in training phases three and four.

The foregoing nonmedical migraine treatment studies and case reports have used patient self-reports of headache frequency as the primary dependent measure of success. Type of treatment varied from analysis, to hypnosis, to forms of behavior therapy. Most treatments relied on the identification of preheadache or discriminative stimuli to cue some trained response by the patient. These treatments included home practice of the new response(s). The Mitchell

and White (1977) study suggests that self-recorded data does not have a reactive effect, and should not be considered as an active ingredient in migraine treatment. The specific effects of discriminative stimulus identification and home practice have not been isolated. The effect of relaxation as the active ingredient in hypnosis, desensitization and by itself has not been completely resolved. The only controlled study of relaxation (Mitchell and Mitchell, 1971) suggests that it was not successful alone in reducing frequency of migraine attack.

Biofeedback

Biofeedback refers to a procedure in which a specific physiological function of the body is measured and continuously fed back to the organism, usually in the form of a visual or auditory signal. The organism is made aware of spontaneous changes in functioning which when coupled with reinforcement may facilitate an acquired control over the function. Biofeedback procedures have received wide acclaim and have been the object of many recent research efforts both basic (Kimmel, 1967; Miller, 1969) and applied (Birk, 1973; Blanchard and Young, 1974) with animal and human subjects. The focus of this section will be on research with humans which investigates biofeedback of peripheral vascular functioning, and applied research examining migraine treatment success with two types of biological feedback.

Information obtained from mechanical or electrical receptors affixed to the body proximal to the site of specific functioning brings interoceptive stimuli to awareness. These stimuli convey information about internal physiological responses which may conceivably be altered by known learning mechanisms. Shapiro and Schwartz (1972) review research on the clinical applications of various types of biofeedback with high blood pressure (essential hypertension); cardiac arrhythmias; tension headaches; Raynaud's disease; and migraine headache. Blanchard and Young (1974) refer to clinical applications of biofeedback by type of function measured by different feedback instruments. They review applications of feedback of muscle tension (electromyogram or EMG); heart rate; blood pressure; stomach acidity; cortical electrical activity (electroencephalogram or EEG); blood volume and skin temperature.

The advent of biofeedback as a nonmedical alternative to treatment of such psychosomatic disorders necessitates a novel emphasis on participation by the patient in his own treatment. The degree to which an organism can exhibit control over its own internal visceral responding suggests learned mechanisms may be voluntary. Consequently, the role of the individual patient in assuming responsibility for his own physical health becomes of paramount importance (Schwartz, 1973). Thoresen and Mahoney, (1974) discuss

four possible advantages of self-management over change traditionally administered by others. Self-control decreases the demand on professional help. Since the individual has continuous access to his own overt and covert behavior, he is best able to perceive and control it. Self-control may aid in generalization of newly acquired responses across settings thus facilitating transfer of training. Finally, important skills and information are learned which may be valuable in other contexts.

Biofeedback training of both humans and animals has raised several questions pertinent to basic research. The concept of voluntary control is considered difficult to define, especially now that body functions once considered to be controlled autonomically or involuntarily are being modified with biofeedback training (Davidson and Krippner, 1972) and the voluntary efforts of the subject. Related to this outmoded division of body functions into those which are autonomically (visceral) and those which are voluntarily (somatic) controlled, is the concept (promoted by Skinner, 1938) that autonomic functions may be conditioned classically and voluntary functions may be conditioned operantly. The question of whether observed changes in autonomic functioning were due to operant or classical conditioning also arose. These two definitional/theoretical issues have been debated for over ten years. Proponents of biofeedback procedures as operant techniques

which can be used to control autonomic functions (Kimmel, 1967, 1975; Miller, 1969; Dicara, 1970) use bidirectional conditioning designs to demonstrate their claim. Because classical conditioning may only serve to change functioning in one direction depending on the nature of the response conditioned (Miller, 1969) the demonstration of bidirectional conditioning of a given response suggests that changes in response are due to operant conditioning. Opponents base their criticisms of these demonstrations on methodological (Katkin and Murray, 1968; Katkin, Murray and Lachman, 1969) or theoretical (Schoenfeld, 1971) grounds.

Examination of the possible mechanisms by which autonomic responses are conditioned suggests that conditioning may be indirect, or mediated by other responses. Katkin and Murray (1968) suggest that target autonomic responses are not directly conditioned by operant procedures, rather somatic (voluntarily controlled) responses are conditioned operantly which in turn may effect autonomic responses. Miller (1969) summarizes operant biofeedback research efforts with animals which were designed to rule out mediation as explanation of results. Both Miller and DiCara (1970) emphasize that somatic mediation in autonomic operant conditioning research is very unlikely by demonstrating that conditioned responses are specific, or by demonstrating autonomic response conditioning while the animals' somatic motor system is paralyzed.

The issue of mediation has not been completely resolved because of failures to replicate Miller's studies using curare to paralyze experimental animals. However, Miller (Miller and Dworkin, 1975) stands by "the strongest argument" against explaining operant autonomic conditioning research as mediated, which is demonstration of autonomic response specifity by operant conditioning. He notes that such demonstrations have been replicated and expanded across different autonomic functions and laboratory animals.

The issue of mediation is very much alive in some explanations of the mechanisms of biofeedback and conditioning of autonomic responses with human subjects. However, mediation is a more complex issue with humans. Response specificity may be demonstrated to counter somatic mediation explanations of autonomic conditioning, but cognitive mediation is an additional alternative explanation confounding attempts to demonstrate direct autonomic operant conditioning in human subjects.

That some effect is produced by biofeedback procedures with humans is beyond question (Davidson and Krippner, 1972; Kimmel, 1975; Katkin and Murray, 1968; Crider, Schwartz, Shnidman, 1969; Dicara, 1973). Active ingredients in biofeedback procedures or mechanisms accounting for observed effects continue to be the source of dispute (Shapiro, 1977). Black (1974) proposes that operant conditioning of an

autonomic response can occur with mediation:

> When we say that a change in some response was operantly conditioned, what we usually mean is that the change in the response was produced by the pairing, or contingency between, response and reinforcer. If we accept this position, then a change in an autonomic response can be produced by operant conditioning whether mediation occurs or not, as long as the response-reinforcement contingency produced the change. When there is no mediation, the operant conditioning procedure changes the autonomic response directly. When there is mediation, the operant conditioning procedure changes the mediating responses which in turn change the autonomic response. That is, the operant conditioning procedure changes the autonomic response indirectly. One might be tempted to argue that the operant conditioning procedure really affected only the mediating response. But as long as the mediating and mediated responses are positively correlated, it seems more correct to say that it affected both. (Black, 1974, p. 231)

In discussing the importance of "autonomic substructure" and "cognitive superstructure" in behavior theory, Razran (1973) defines cognition as direct or inferred awareness of phenomenal experience which is always specifically related

to higher level neural action which he refers to as "neurocognition". Accordingly, he argues strongly for behavior therapy using both cognitive learning and visceral conditioning.

> Although the motor and verbal are obviously our means for affecting the environment and each other, and although cognitive learning is perhaps less universal than noncognitive, and interoceptive less prompt than exteroceptive, it is true nonetheless, that simplistic therapies in presumed eviscerated, denervated, and "decognized" human beings are too truncated to be successful in more than limited areas, and too static to be productive for long. The black box, unlike Pandora's, should be plied open. (p. 169)

Recently Lang (1974) has hypothesized that the greater cognitive capacity of human subjects and the less tightly controlled situations in which they may be tested alter the learning situation (from that of experimental animals) to such an extent that an operant conditioning paradigm may not provide an adequate description of the mechanism of human autonomic learning. He suggests the theoretical models of feedback discussed by E. A. Bilodeau (1969) and I. Bilodeau (1969) as possibly more appropriate. In human skills learning, feedback is considered more as information than reinforcement. This information provides response-

correcting properties as well as confirmation of correct responding.

In discussing this theoretical issue, Shapiro and Surwit (1976) note that the information model may be the more powerful of the two in explaining human behavior. However, information and reinforcement should not be considered to be mutually exclusive models or explanations. The importance of feedback as information seems relevant to the development of a new response, while reinforcement may better explain the maintenance of a response. Information about interoceptive stimuli may bring to awareness changes in the target function. Desired changes will be recognized and reinforcement will serve to increase the frequency of the desired changes in autonomic responding.

Conditioning of vascular changes in humans has relied on two different measures of vessel diameter change. Early efforts fed back measures of blood volume as monitored by plethysmography to subjects as information about relative vessel diameter. Recent biofeedback procedures have used measures of skin temperature as an index of relative blood supply and hence of relative vessel diameter. Both measures for biofeedback have been successful in otherwise similar procedures for conditioning vascular changes. Following is a brief review of findings relevant to these changes.

Blood Volume Feedback

In 1938, Skinner briefly reported an unsuccessful attempt in collaboration with Delabarre to condition vasoconstriction of the arm in human subjects. He cited this unsuccessful attempt as evidence that autonomic responses could not be operantly conditioned. Subsequent research has failed to support this contention. In 1957 Lisina, a Russian psychologist, reported on three series of experiments on conditioning of vasodilation. Unfortunately the location of transducers on the subjects was not reported. Measures of relative vascular change were recorded by plethysmograph. All three series used an electric shock to stimulate a vasoconstrictive reaction. When this vasoconstriction subsided, vasodilation ensued and was rewarded by termination of the shock. In the first series of subjects, no feedback or instructions were provided and no conditioning of vasodilation was observed after 80 trials with five subjects. In the second series of subjects visual feedback of vascular reactions was provided to each subject, though no information was given about the condition for shock termination. Lisina reported that after ten to fifteen trials, subjects became aware of the relationship between feedback and shock termination. After 40 to 45 trials, each subject was able to demonstrate consistent vasodilation. The third series of subjects were taught to recognize "their own skin tactile sensations" and rely on

these as indices of relative vasodilation. Experimenter's instructions facilitated this training, and subjects were able to demonstrate vasodilation using their own "feedback". Lisina lists instructions to subjects to relax and change depth of respiration as possible confounds.

In 1968, Snyder and Noble used undergraduates to investigate conditioning of digital vasoconstriction. Two experimental groups received information and reinforcement by a light which was illuminated after each vasoconstriction detected plethysmographically. A group reinforced for vascular stability and a no-reinforcement group were used as controls. Data was recorded during five sessions each lasting forty minutes. In order to demonstrate that conditioned vasoconstriction was independent of gross bodily movements, EMG data from forearm and finger was recorded in addition to heart rate and respiration. The two experimental groups differed significantly in the expected direction from the two control groups in mean number of recorded vasoconstrictions. Other measures taken suggested that operantly conditioned vasoconstriction was independent of somatic responses measured.

Stern and Pavlovski (1974) successfully replicated the Snyder and Noble (1968) study using a larger sample in three groups. The experimental group received reinforcement by a light illuminated contingently on vasoconstriction. One control group was yoked and the second partly yoked so that

reinforcement was not contingent on control subjects' vasoconstrictions. The experimental group demonstrated significantly more vasoconstrictions than both control groups at each of five sessions. Performance of the experimental group reached almost the highest level of responding during the first five minutes of the first session.

Volow and Hein (1972) used eight male college students to investigate bidirectional conditioning of vascular change in the finger. Subjects received continuous visual feedback with auditory reinforcement for plethysmographically measured vascular changes in the desired direction. Results showed that two subjects could both dilate and constrict reliably; four subjects could only constrict reliably; one subject could only dilate reliably; and one subject learned neither response. The authors conclude that individual differences in magnitude and direction of vascular conditionability explain obtained results.

Christie and Kotses (1973) investigated conditioning of vasomotor response of the temporal artery of the scalp. Eight male college students were divided into a dilation training group and a constriction training group. Vascular changes were measured plethysmographically and fed back visually to subjects. Escape from aversive white noise was used as reinforcement. Each subject participated for six thirty-minute sessions. Although magnitude of change is not reported, reliable conditioning in the appropriate

direction was demonstrated for all subjects. The authors note that "a significant amount of control" had been established during the initial three sessions.

Skin Temperature Feedback

Taub and Emurian (1973) used skin temperature feedback with an operant shaping technique to train 19 of 20 subjects to reliably increase finger temperature. Small, incremental changes in temperature were reinforced by a variable intensity feedback light. Subjects were encouraged to use thermal imagery, with the nature of the imagery left to each individual. After each session the graph of their finger temperature was shown to each subject and performance during the session was discussed. This was another source of feedback and verbal reward. The investigators report that training "to a level of unequivocal acquisition" rarely required more than four 15 minute feedback periods. The mean reported temperature change for all subjects over sessions 4, 5 and 6 was 2.2^{0}F. After initial training some subjects were asked to reverse the direction of temperature change. The four subjects trained longest demonstrated the ability to autoregulate bidirectional control during successive periods on the same day. After "sufficient training" autoregulation of finger temperature was as reliable with feedback as without. Retention of bidirectional control in the four subjects trained longest was "virtually perfect" after a follow up interval of four

to five months.

McDonagh and McGinnie (1973) used autogenic training and skin temperature feedback to increase subjects' skin temperature as recorded at the middle finger of the dominant hand. After data was collected on all fourteen student subjects, data was split into two groups. The low initial temperature group performed significantly better at the hand-warming task than the high initial temperature group. Although the specific effects of training were confounded, the investigators were able to demonstrate the importance of the law of initial values. Subjects with lower initial skin temperature will exhibit a greater ability to increase temperature than subjects with a higher initial temperature. This point is particularly relevant to skin temperature training of migrainous subjects who tend to have lower peripheral skin temperature than normals. (Ostfeld and Wolff, 1958; Dalessio, 1972)

Roberts, Kewman and MacDonald (1973) examined hypnosis and skin temperature feedback to train six student subjects to control skin temperature of one middle finger relative to the middle finger of the other hand. For each of three 50 minute training sessions, subjects were asked to alternate direction of temperature difference between fingers three times. Five of six subjects demonstrated significant control over direction of change, with 1.7^{o}C average magnitude of change after three sessions for all six subjects.

The two subjects demonstrating the greatest degree of control were studied in two subsequent sessions without skin temperature feedback. Both subjects demonstrated a continuation of reliable control over skin temperature difference between hands, while again alternating the direction of difference between hands. In discussing individual differences in acquisition, the investigators noted that different subjects may rely on different physiological processes to alter skin temperature.

Keefe (1976) used feedback of the difference between skin temperature recorded at the right index finger and midforehead to train eight male college freshmen to control magnitude of difference. Subjects were randomly divided into a group instructed to decrease difference and a group instructed to increase the difference. Twelve training sessions of fifteen minutes apiece each consisted of a five-minute baseline followed by ten minutes of visual and auditory feedback. Results indicated that all eight subjects were able to demonstrate reliable control of skin temperature differential in the appropriate direction. Furthermore, changes in absolute finger temperature correlated .87 with overall changes in differential skin temperature. This substantiated the claim that changes in skin temperature are primarily due to absolute changes in finger temperature (Sargent, Green and Walters, 1973). Keefe concluded subjects "clearly responded to feedback and instructions to

alter skin temperature difference in a specified direction."

Albertstein (1977) assigned 56 male undergraduates to one of four experimental groups. One group received visual fingertip skin temperature feedback and instructions to increase finger temperature. A second group received the same feedback with instructions to decrease finger temperature. A third group received no feedback but instructions to relax. The fourth group received visual frontalis EMG feedback and instructions to relax the forehead. EMG and skin temperature data were recorded for all over one thirty-minute baseline session and four forty-minute training sessions. Significant differences in temperature change were reported between the no-feedback group and the temperature decrease group; and in frontalis relaxation between the no-feedback group and the EMG decrease group. No reports were made regarding the effect of instructions to relax or EMG decrease in skin temperature. These comparisons suggest that normal subjects (not identified for migraine) can learn to decrease their digital skin temperature (vasoconstrict) better than they can learn to increase it in comparison with a relaxation group.

Surwit, Shapiro and Feld (1976) used monetary reward with visual skin temperature feedback from the middle finger of the nondominant hand to train vasomotor control. Subjects were respondents to a newspaper advertisement, ranging in age 18 to 30. Eight subjects each were assigned to

two different groups: one temperature increase group and one temperature decrease group. Additional data was recorded simultaneously from two plethysmographs, transducers of which were placed next to the temperature thermistor on each hand; a cardiotachometer; and a respiration strain gauge. Five training sessions of 60 minutes duration followed two 30-minute baseline sessions over a period of seven days. Reported correlations between skin temperature and blood volume recorded plethysmographically ranged from .55 to .80. These significant correlations are considered to be important validations of the use of skin temperature to measure relative blood volume. Results of skin temperature training indicated that temperature decrease and thus vasoconstriction in the nondominant middle finger was easier to learn than temperature increase and vasodilation. The investigators reasoned that, since subjects trained to increase their skin temperature showed average skin temperature near body core temperature, a ceiling effect may have prevented better performance in the increase group.

Surwit, Shapiro and Feld (1976) include the results of a second study designed to investigate this ceiling effect. A different group of eight subjects was recruited and trained under the temperature increase condition with all procedures identical to the first experiment except that the ambient temperature in the experimental room was lowered $3^{o}C$. Results of this study again showed difficulty in

vasodilation by subjects in the increase temperature group. The investigators concluded that the relative inability of their subjects to increase temperature was not due to a ceiling effect. Neither study revealed significant differences in respiration rate or heart rate between temperature increase and decrease groups. From this the investigators concluded that the observed vasomotor control was not mediated by respiratory or heart rate control.

Evidence of a ceiling effect referred to by Surwit, Shapiro and Feld (1976) was not found by changing ambient room temperature. However, the study by McDonagh and McGinnis (1973) suggests that attempts to increase temperature were unsuccessful because subjects' initial temperature approached a maximum value. Thus a ceiling effect may be explained in terms of individual differences in initial or baseline temperatures rather than differences in ambient room temperature. Migrainous patients may be better able to increase digital skin temperature than normals because they tend to have lower peripheral skin temperature (Wolff, 1963).

The foregoing studies with subjects unselected for migraine suggest that skin temperature may be used as an index of blood volume (Roberts, Kewman and MacDonald, 1973; Surwit, Shapiro and Feld, 1976; Taub, 1977). When differential feedback procedure between finger and forehead is used, the absolute changes in finger temperature account for

changes in the difference (Keefe, 1976 a; Sargent, Green, and Walters, 1972). Skin temperature feedback plus instructions to increase or decrease skin temperature influence change in the appropriate direction (Taub and Emurian, 1973; Albertstein, 1977; Keefe, 1976 a). Training of subjects with plethysmograph or skin temperature feedback is immediately effective with reliable acquisition demonstrated after four sessions or less, each less than 60 minutes duration. (Taub and Emurian, 1973; Keefe, 1976 a and b; McDonagh and McGinnis, 1973; Roberts, Kewman and MacDonald, 1973). Results of feedback training transfer to no-feedback conditions (Roberts, Kewman and MacDonald, 1973) and are maintained after brief follow-up periods (Keefe, 1976 b).

The physiological mechanism of reported changes in blood volume and skin temperature does not seem to be due to changes in heart rate or respiration (Surwit, Shapiro, and Feld, 1976); gross bodily movement (Taub, 1977); or muscle tension in the forearm or finger (Snyder and Noble, 1968; Taub and Emurian, 1976). These findings lend some support to Sargent, Green and Walters' contention (1972, 1973) that vasomotor changes may be regulated by control of sympathetic flow. Likewise, instructions to relax the whole body (Keefe, 1976 b) or the forehead (Alberstein, 1977) produced no significant effect in temperature change.

Repeated successes across varying conditions in training normals to change their skin temperature or peripheral

blood volume indicate that vasomotor responses may be conditioned. The mechanisms of these reported changes remain unclear. Experimental procedures may rely on specific or nonspecific effects to condition either directly or indirectly the autonomic responses which regulate blood flow and skin temperature.

Biofeedback Treatment of Migraine

The application of biofeedback training to treat various psychosomatic disorders stems from some implications of autonomic conditioning demonstrated with animals. Miller (1969) suggests that evidence of the operant learning of visceral responses removes the main basis for assuming that psychosomatic symptoms that involve the autonomic nervous system are fundamentally different from functional symptoms of the cerebrospinal system.

> I have emphasized the possible role of learning in producing the observed individual differences in visceral responses to stress, which in extreme cases may result in one type of psychosomatic symptom in one person and a different type in another. In fact, given social conditions under which any form of illness will be rewarded, the symptoms of the most susceptible organ will be the most likely ones to be learned. (Miller, 1969, p. 444)

Miller reasoned that a patient who is motivated to get rid of a symptom can learn to control operantly that

symptom, given appropriate feedback about his responses and reinforcement for correct responses. Specifically, Sargent, Walters and Green (1973) hypothesize that feedback for hand warming is effective in treating the migraine headache because patients learn to regulate sympathetic control of vascular changes. Hand temperature serves as an index of vasoconstriction which is presumed to be controlled by the sympathetic nervous system. Regulation of sympathetic flow can then influence vascular changes at the forehead and fingers.

In 1972 Sargent, Green and Walters reported results from a pilot study of the use of autogenic and biofeedback training as treatment for migraine. This clinical study included data collected on 75 patients either self-referred or referred by physicians to the Menninger Clinic for headache treatment. The authors emphasize that their sample was select in that their patients were typically unable to find relief by more conventional therapeutic means. A comprehensive medical diagnosis excluded from the study those with organic complications and served to distinguish migrainous patients from those suffering from tension headaches or a combination of the two. In addition to autogenic training described by Shultz and Luthe (1969), the patients were given visual feedback of the difference in temperature between mid-forehead and right index finger. Patients practiced both techniques daily at home until they demonstrated

effective voluntary control over the skin temperature differential. Patients also kept daily records of headache intensity and drugs taken. Improvement was rated by each author for the 62 patients for which adequate data was available. Global improvement ratings by each author were 90%, 80%, and 68% of all patients. The authors acknowledge that these results were the product of a pilot study uncontrolled for placebo effects.

Sargent, Walters and Green (1973) presented results of a subsequent study at the Menninger Clinic of 25 headache patients, six of whom were diagnosed with tension headache, the remaining 19 with migraine. Medical screening, autogenic and biofeedback training, home assignments and patient-recorded data were all the same as described in the 1972 report. Modifications in individual training programs were made as the study progressed. As with the 1972 report no attempt was made to systematize training or experimentally control for possible confounds or nonspecific effects. Improvement ratings were again made by each author. Unanimous agreement on improvement (criteria not reported) was noted for 63% of those patients with migraine and only 33% of those with tension headache. The authors noted that temperature regulation of the hands was responsible for changes in the differential between finger and forehead. They conclude that "...temperature regulation of the hands seems a useful adjunct in the treatment of migraine attacks.

Tension headaches may require a different type of training."

Solbach and Sargent (1977) presented a follow up evaluation of the Sargent et al pilot work done through the Menninger Clinic. The amount of time elapsed after completion of training was not uniform, but was longer than a year for all migraineurs. Participants in the pilot study were contacted by mail and were requested to complete a questionnaire. Nonrespondents were surveyed over the telephone, with a total of 56 of the 74 migraineurs who completed training being contacted. Responses to the survey are also reported by 12 of the 36 migraineurs who failed to complete training. All participants who completed the program judged their headaches as decreased in frequency, intensity, duration and use of medication but the baseline used for comparison was not reported. Program drop-outs judged their headaches as being less frequent, less intense but without change in duration, or use of medication. Reported results suggest that the training program was beneficial on follow-up as judged by participants contacted. The strong demand characteristics of a follow-up survey, the nonexperimental nature of the original study and this follow-up preclude any generalization about long-term effects of the training procedure used by the Menninger group. It is of interest to note that migraineurs from both groups reported that, in retrospect, the most helpful aspects of training were relaxation exercises and staff interest and support.

Wickramaskera (1973) reported two single case studies of a 46-year-old female and a 45-year-old male, both of whom had previously received psychotherapy and chemotherapy without positive outcome. The male patient received 16 EMG feedback sessions with instructions to practice relaxation at home daily. The female patient had completed 18 EMG feedback sessions with similar instructions to practice daily at home. Both reported a slight reduction in intensity of headache while frequency remained unchanged.

Failing adequate improvement with muscle relaxation training both patients were offered skin temperature training as an alternative treatment. Both patients had been checked medically to rule out organic complications. Both received skin temperature training in the same room used for muscle relaxation training. Thermistors were placed at mid-forehead and on the right index finger of each subject, with instructions to concentrate on warming the temperature of their hands. Patients were asked to record duration, and intensity of headaches for three weeks prior to training and for the eleven weeks of training. Recordings of skin temperature indicated that handwarming was learned rapidly. Both patients exhibited significant reductions in duration and intensity of headache, and reported at a three month follow-up that use of analgesics had been reduced.

Beasley (1976) assigned 37 female patients to four

separate treatment conditions. Each patient was requested to record her own data on headache frequency and intensity. Groups were treated as follows: one group (A) received relaxation exercises, autogenic training and finger temperature feedback; one group (B) received feedback alone; one group (C) received relaxation exercises and autogenic training with no feedback; and one group (D) served as a no-treatment control. Each patient received ten one-half-hour sessions of treatment, during which EMG and finger temperature were recorded. Group A, receiving the full treatment package, reported significant improvement in headache frequency and intensity. In the finger temperature only group, a trend toward less intense headaches was noted in comparison with groups C and D. Group C reported no decrease in headache frequency and intensity. Beasley concluded that biofeedback coupled with other procedures was a viable technique for relieving migraine pain.

Beasley's attempt at a controlled study of migraine treatment failed to systematize treatment presentation and to isolate the effects of different independent variables. Skin temperature feedback was one common ingredient in both groups exhibiting any improvement. This study also failed to study nonspecific or placebo effects. Thus alternative explanations, such as patient expectations for a novel and impressive treatment procedure, might adequately account for changes observed.

Reading and Mohr (1976) reported results of a well conceived pilot study, training six migraineurs to increase right index finger skin temperature. Migraineurs kept their own records of headache frequency, duration and intensity during baseline, training and posttraining periods. The number of training sessions was not specified, but presumably varied depending on some criterion of acquisition. Although no controls were used to assure internal validity, the authors did provide evidence for an improvement in subjects' ability to increase skin temperature concomitant with subjects' reports of decreased migraine frequency, duration and intensity. The authors took care to demonstrate acquisition of voluntary temperature control without feedback at the end of training. This provided support for the assumption that subjects actually can increase skin temperature in vivo to control headaches. Subjects continued to demonstrate control of skin temperature without feedback at one-month and two-month follow-up intervals. Reduction of frequency, duration and intensity of migraine was maintained during the two-month posttraining period.

The first biofeedback study of migraine treatment attempting to control for possible placebo effects was reported by Friar and Beatty (1976). Nineteen migraine sufferers were recruited through a college newspaper, and ranged in age from 19 to 54. Each had completed a headache

questionnaire which was reviewed by a physician who pre-selected patients for maximum diagnostic certainty of migraine. Subjects recorded their own headache data including frequency, duration and intensity for 30 days prior to training; the three-week training period, and for 30 days following training. Subjects were divided into an experimental and a control group matched for headache frequency, age and sex. Skin temperature and pulse amplitude (a measure of blood volume) were recorded for all subjects over each of nine training sessions. A temperature thermistor and a plethysmographic transducer were affixed to the scalp and the finger on the side of the body most often affected by headache during the 30-day baseline. The experimental group received feedback from the scalp plethysmograph site, while the control group received feedback from the finger measure of pulse amplitude. Both groups received visual and auditory feedback with instructions to reduce pulse amplitude, i.e., vasoconstrict. All subjects were given a positive set toward outcome initially and the experimenter encouraged all subjects throughout training.

Results indicate that experimental subjects were able to demonstrate vasoconstrictions of greater relative magnitude in scalp arteries than control subjects trained at a different site. Both groups demonstrated vasoconstrictions of roughly the same magnitude at the finger site. Between-group differences in posttraining self-reported headache

symptomatology were reported with $t(17) = 1.96$ and $p < .05$, one tailed. Both groups reported fewer headaches; while the best results, from the experimental group, demonstrated a reduction in frequency by about 47%. No significant differences were noted for between-group differences in posttraining headache intensity. The investigators did not quantify drug intake, but noted that the experimental group and control group reported a decreased use of vasoconstrictors and mild analgesics.

The investigators acknowledge that it is impossible to rule out the fact that forehead training was more convincing than hand training. But they noted that "...experimental and control subjects seemed equally convinced at the end of training that they would be able to control their headaches." The investigators conclude that operant procedures may result in "moderate alterations" in vasomotor tone at the site of reinforcement, and that learned vasomotor control may be clinically meaningful for migraine.

The Friar and Beatty study is the first reported study to effectively make uniform presentations of training to experimental and control groups. Aside from failing to report important data (such as skin temperature), a possible relaxation effect confound and antiquated statistical analyses, the issue of a possible placebo effect remains uncertain in this study. Hertzman (1959) has stated that sympathetic innervation has a constrictor effect in the

hands and feet and a dilator effect on the face and scalp. If this is true, then training the control group to constrict finger vessels may have conditioned central sympathetic innervation (as Sargent, Green and Walters, 1972, 1973 believe) which might have the paradoxical effect of dilating scalp vessels. That is, the group considered to control for nonspecific effects of biofeedback might have received active and specific countertherapeutic training. It does not seem reasonable to assume that the control conditions used were clearly lacking in specific action. Thus, the nonspecific effects of biofeedback were not effectively assessed separately from the possible specific effects of biofeedback.

Johnson and Turin (1975) reported a relatively well controlled single case study which noted the effects on headache of skin temperature feedback for hand cooling, then warming. The patient was a 27-year-old nurse with a two-year migraine history. She was instructed to record headache duration, frequency and number of pills taken for migraine over a five-week baseline period; six weeks of hand-cooling training and six weeks of hand-warming training. The patient received visual feedback from the skin temperature trainer with thermistor attached to the index finger of her dominant hand. She received two 45-minute training sessions per week and was told to practice daily at home. Results indicated that she was able to reliably

cool her finger during the first training period and reliably warm her finger during the second period. The magnitude of change was larger for warming, as would be expected for a person with cold hands (McDonagh and McGinnis, 1973). Self-report headache data indicated that although instable, frequency and duration of headache increased over baseline during the cooling period and decreased from baseline during the warming period. The average number of pills taken for headache relief increased over baseline during the cooling period and decreased from baseline during the warming period.

In 1976, Turin and Johnson reported additional results of trained finger warming and cooling for seven patients with vascular headaches, primarily migraine. Homework, data recording and training procedures were similar to Johnson and Turin (1975). After recording four to six weeks of baseline data, three patients received six weeks of finger-cooling training prior to six weeks of finger-warming training. Four other patients received only training in finger warming. All subjects were instructed to alter skin temperature in the indicated direction and to use their training at the first sign of a headache. Results again indicated that duration, frequency of headache, and number of pills taken increased with finger-cooling training and decreased with finger-warming training.

Turin and Johnson suggest that differential success

between cooling and warming training in migraine treatment demonstrate that a placebo-expectancy explanation is not tenable. This study is considered to be a strong demonstration of the effects of skin temperature training procedures, but it does present a procedure with possible specific effects to the placebo control group. Selection of patients by imprecise diagnosis calls into question a possible diagnosis by training procedure interaction which might have distorted findings of simple effects. Results are also rendered difficult to generalize due to poor definition of patient selection.

Mullinix, Norton, Hack and Fishman (1978) provided training with skin temperature feedback for two groups of six migraineurs each. Both groups participated in six thirty-minute training sessions over a two-to three-week period with additional sessions one, two and six weeks following initial training. One group was given contingent feedback with instructions to increase skin temperature, while the second group received noncontingent feedback yoked to members in the first group. The migraineurs receiving contingent feedback increased mean skin temperature significantly higher than migraineurs receiving noncontingent feedback. The authors failed to report whether measures of skin temperature used were obtained during a feedback or no-feedback period, and when during training they were obtained.

Seven of eleven migraineurs who completed the training

reported improvement on the standard four headache measures after training relative to pretraining baseline. Four migraineurs were from the contingent feedback group and three were from the noncontingent feedback group. One migraineur from each group reported no change, and one from each group reported increases on headache symptoms. Furthermore, the authors did not find any correlation between the magnitude of temperature elevation achieved and amount of improvement on headache symptoms. The authors concluded that biofeedback training seemed to be effective in treating migraineurs, but the beneficial effect probably did not depend on modification of physiological functioning. The incomplete description of procedures used, and failure to report mean temperature changes made the authors conclusions seem tenuous, though provocative.

Kewman (1977) also attempted to examine the relationship between finger temperature change and change in the standard headache variables. One group of eleven migraineurs was trained to increase finger temperature, a second group of twelve migraineurs was trained to decrease finger temperature while a third group of eleven migraineurs served as an untreated control group. Not all migraineurs in the temperature control groups learned appropriate control of finger temperature. However all three groups of migraineurs tended to report a decrease in posttraining headache measures relative to pretraining baselines.

In a post hoc analysis, migraineurs were regrouped according to a learning criterion. Migraine patients who did learn to raise finger temperature with feedback showed a reduction across all headache measures. However this group of migraineurs did not demonstrate significant headache improvement over the untreated group or the migraineurs who did not learn to raise finger temperature. Each of these three groups showed significantly greater improvement compared to the group that decreased skin temperature. Kewman concluded that finger temperature changes alone could not account for improvements on headache measures.

Considerable evidence exists to suggest that people of both sexes and across an age range of 18 to about 60 can quickly learn to control skin temperature at sites on fingers or the scalp. Recent research on biofeedback procedures with those suffering with migraine in the same population suggest that these procedures may be partly effective in treating the headache symptom of migraine. Studies thus far have used plethysmograph or skin temperature data as relative indices of blood flow and vascular change. Site of training has evolved from a combination of finger and mid-forehead to finger only (Sargent, Walters and Green, 1973). The thermistor of the skin temperature apparatus is most often attached to the index finger of the dominant hand. All subjects received visual analogue or binary feedback, with some studies using both visual and auditory

feedback. A controlled study comparing the effectiveness of analogue visual feedback versus analogue binary visual and auditory feedback indicated no difference in acquisition of control over skin temperature (Surwit, 1977).

Thorough biofeedback studies of migraine treatment have included multiple diagnostic opinions by both physicians and psychologists and have attempted to exclude subjects with any hormonal or nervous system complications. No study has requested that subjects accepted for the study discontinue any medication, although a decrease in medication is usually noted with reduction in headache frequency or intensity. All subjects in each study have been required to keep daily records during baseline, treatment and a short posttreatment period. Once treatment began, all studies have recommended home practice of training procedures either with or without home feedback units.

Dependent variables used in early studies were physician's or psychologist's assessment of improvement based on patient's self-report (Sargent, et al., 1972, 1973). Recent studies have used patient self-reports of some combination of headache variables (Wickramaskera, 1973; Johnson and Turin, 1975; Turin and Johnson, 1976; Beasley, 1976; and Friar and Beatty, 1976). All of these studies include some measure of frequency. Other dependent measures used include headache duration, intensity, and number of pills taken for headache relief.

Biofeedback procedures have been shown to be effective with feedback from a plethysmograph (Friar and Beatty, 1976) or a skin temperature register (Johnson and Turin, 1975); in combination with hypnosis (Andreychuk and Skriver, 1975) and without (Turin and Johnson, 1976); in combination with autogenic training (Sargent, Walters and Green, 1973) and without (Mullinix et al., 1978). Although relaxation of subjects during training seems to occur invariably and may even be encouraged, biofeedback training has been more effective than simple muscle relaxation procedures in migraine treatment (Wickramaskera, 1973; Beasley, 1976). It is possible that the relaxation learned with feedback of muscle action potentials differs in quality from relaxation which seems to accompany control of skin temperature. Nonspecific elements of the biofeedback training procedure may also have influenced improvements reported across the literature (Stroebel and Glueck, 1973).

Reports of interaction between experimenter and subject in biofeedback treatment studies of migraine are limited to instructions to subjects to implement what they learn in training at the first sign of headache; to remain relaxed during feedback sessions; and a brief description of desired direction of temperature change. With the exception of studies which include ancillary techniques such as autogenic training or hypnosis, no instructions are given regarding effective cognitive strategies. One study (Turin

and Johnson, 1976) included instructions to check the effectiveness of any cognitive strategy used by attending to the feedback meter.

Transfer of training from treatment setting to the subject's natural environment has not been documented formally, nor has transfer from training with feedback to practice without feedback. Subject self-reports of decreased headache frequency, duration, intensity and medication reduction have been the basis for experimenters' conclusions that transfer has been effective. The duration of treatment effects is likewise poorly documented. Two of the controlled biofeedback studies with migraine have reported follow-up data: after one month (Friar and Beatty, 1976) and three months (Wickramaskera, 1973). Both studies suggest that improvement is maintained, but long-term follow-up data is lacking (Sargent, Green and Walters, 1973).

Placebo Effects

Although considered experimentally difficult or impossible (Stroebel and Glueck, 1973) and clinically unnecessary or inefficient (Evans, 1975), the isolation of placebo effects from a new treatment procedure such as biofeedback is considered important in demonstrating its validity as an effective treatment (Birk, 1973).

> A placebo is defined as any therapy, or that component of any therapy, that is deliberately used for its nonspecific, psychologic, psychophysiologic

> effect or that is used for its presumed specific effect on a patient, symptom, or illness, but which, unknown to patient and therapist, is without specific activity for the condition being treated. A placebo, when used as a control in experimental studies, is defined as a substance or procedure that is without specific activity for the condition being evaluated. (Shapiro, 1971)

According to Shapiro (1960), the placebo effect appears to consist of three general factors. The first involves errors in methodology which result from variables other than the experimental variable determining results. This can occur when groups are compared which have not been adequately matched for all possible variables affecting outcome. The second factor is the effect of the treatment agent in influencing patients' suggestibility or expectations for treatment outcome. This factor is typically attributed to the physician in medical studies but might apply to whoever interacts with the patient or aspects of the treatment setting itself. The third factor is any uncontrolled variable in experimental research which is influenced by methodological errors described above as factor one, or the agent of treatment. Shapiro also notes that a placebo should be distinguished from its effect, as not every placebo results in a placebo effect.

Shapiro (1964, 1971) lists specific elements which

may contribute to placebo effects as patient variables, situation variables, and physician variables. Included as patient variables are suggestibility (addressed by Andreychuk and Skriver, 1975), faith or hope in treatment or therapist, expectations and motivation for treatment and other demographic and personality variables. Situation variables include staff attitudes to the study, treatment procedures or machines, and the prestige of the treatment setting. Physician variables include therapist's attitude toward the patient, the experiment and interest in results.

Leiphart (1976) examined the effects of an expectancy-based treatment on behavioral change for 84 snake-phobic students. Subjects received a series of treatment sessions comprised of an "inert combination" of procedures using either biofeedback or feedback by a therapist. Results indicated that expectancy was effective in producing behavior change. Leiphart also concluded that inert apparatus feedback (nonspecific biofeedback) was superior to feedback by a therapist in enhancing expectancy, and producing a greater degree of behavioral change.

Gibb, Stephan and Rohm (1975) provide evidence that belief alone in the effectiveness of biofeedback may influence physiological changes such as frontalis muscle relaxation and raising skin temperature. Sternbach (1964) has also shown that subjects' expectations for treatment effectiveness have an effect on autonomic responses as measured

by palmar skin resistance, electrocardiogram and plethysmograph. In Sternbach's study expectations were influenced simply by experimenter's instructions regarding the effect of white noise in reducing the pain of administered shock. Results of this study suggest that once belief in treatment effectiveness is influenced even without the treatment being applied, subjects' thoughts about the treatment may have a beneficial physiological effect.

In an excellent review on pain and control of pain, Weisenberg (1977) discussed several aspects of treatment which contribute to the placebo effect. He emphasized that suggestion by the therapist of treatment success and positive expectation for treatment outcome by the patient are important in establishing a placebo effect. Weisenberg cited research which suggested that both of these factors contribute directly to the relief of the patient's anxiety about obtaining relief from pain. Since anxiety in combination with pain augments the subjective experience of pain, the reduction of anxiety may decrease the subjective experience of pain. Weisenberg claimed that this was widely recognized by the medical profession. In practice, many physicians recommend relaxation to counter anxiety for migraineurs with head pain and some physicians prescribe tranquilizers to insure relaxation for some migraineurs.

Weisenberg also summarized the effects on pain relief of several cognitive variables. Effects of special

instructions to patients about coping with pain may be mediated by attentional factors which divert attention from pain. For example, two studies were reviewed which instructed patients to use emotive imagery while experiencing cold pressor pain. Emotive imagery was found to be effective in both studies in increasing tolerance of pain. Evidence was also cited which suggests that when subjects are taught to attribute changes in pain tolerance to their own efforts, the tolerance of pain is increased. Weisenberg also suggested that once expectations for self-control of pain are developed, anxiety about future pain may be decreased, thus decreasing subjective experience of pain in the future. Cognitive dissonance might work toward increasing pain tolerance if the patient expects that self-control of pain is possible and that medication is not necessary. The patient could reason that if he can control the pain without help, then perhaps it wasn't so intense. The final cognitive variable mentioned by Weisenberg was the patient's uncertainty about the cause, mechanism and patterns of his pain. This uncertainty correlates with anxiety and can be successfully ameliorated by a discussion of cause, mechanism and patterns with the therapist.

Biofeedback treatment for migraine may incorporate many of the elements of effective placebo treatment for pain reviewed by Weisenberg. Stroebel and Glueck (1973) consider biofeedback treatment as an ultimate placebo, a powerful

procedure without specific effect which provides the patient with a means of preventing illness or of self-cure by learning to reduce "susceptibility to pathologic levels of hyperactivation when faced with stressful life events." They emphasize that placebo effects are "inextricably interwoven" with the illness onset and recovery processes. Although considered a contaminant to experimental research, the placebo effect is considered an important clinical variable.

Stimulus Discrimination Training

Successful treatment for migraine headache, by medical and non-medical means, is predicated on the identification of preheadache signs as soon as possible to cue intervention strategies. It is suggested that interventions which have a vasomotor effect may be successful only during the preheadache phase or early after the onset of actual head pain (Friedman, 1968; Turin and Johnson, 1976). No previous medical or nonmedical treatment studies have systematically studied the effect of attempts by a therapist to promote increased awareness of preheadache signs in the patient. Hunter and Ross (1960) helped migraine patients identify early warning signs or situations in which migraine often occurred or was exacerbated, describing this process as psychotherapy. The process of identifying a cue or a constellation of cues which reliably signal headache onset for an individual may also be considered stimulus

discrimination training. Dalessio (1972) warns of the difficulty in reliable identification:

> Of extreme variability and conspicuousness are the preheadache phenomena. Some patients with migraine headache never have clearly defined prodromes. Many have feelings of mounting tension, hunger, and wakefulness, often followed by profound sleep just preceeding the attack. Still others are aware of declining energy and drive, and a few of extreme buoyancy, talkativeness and well-being just before the attack. On the other hand a small group predictably have visual and other sensory disturbances immediately before the onset of the headache. (Dalessio, 1972, p. 228)

Preheadache phenomena are known to vary across migraine sufferers but the identification of these phenomena across headaches of an individual may be more reliable. If a migraine sufferer is able to discern specific preheadache phenomena reliably, he may then initiate self-control techniques to prevent the headache sooner and with presumed greater success than other migraineurs who do not recognize their preheadache phenomena. The recognition of such phenomena could potentially be valuable to patients relying on medical or nonmedical intervention. Recognition of individual preheadache patterns might also suggest to the patient possible precipitators which could be avoided, thus

preventing future possible migraine attacks.

Possible preheadache cues or phenomena mentioned in medical and nonmedical migraine literature range widely from the classical aura (Wolff, 1963) to ingestion of certain foods (Sacks, 1970), to "life experiences" (Friedman, 1964). Difficulty in identification of an individual migraineur's preheadache phenomena may be minimized by the self-recording of possible precursors by the migraineur and discussion of preheadache cues with someone experienced in their recognition. Guidelines for training discrimination between cues available to an organism come from research in animal discriminant learning.

Sutherland and Mackintosh (1971) note two processes in animal discriminant learning. The organism must first learn to attend to relevant cues, or those which yield a successful solution to the problem. Second the organism must attach the correct response to the relevant cue. A preheadache cue may be a reliable predictor, or it may be necessary to identify a set of relevant cues in order to reliably predict headache onset. The identification of a relevant cue or compound of cues may be promoted by a guided discussion with someone experienced in their identification. The correct response to relevant cues could be any response which might prevent headache onset, such as learned skin warming. Avoidance of headache is considered to be strong motivation for preheadache cue discrimination learning.

Restle (1975) mentions three general procedures for facilitating discrimination learning. First, identify as many relevant and redundant cues as possible. Second, attempt to note irrelevant cues and identify them as such. Third, identify emphasizer stimuli which might serve to direct the organism's attention to relevant cues. Organisms must be exposed to both relevant and irrelevant cues in order to better distinguish between them.

Boles (1975) states that the effectiveness of a learning procedure depends on some intrinsic relationship between a cue and its consequence. If migraine sufferers can be made aware of this relationship between preheadache phenomena and headache onset, the discrimination of these phenomena and association of an appropriate response to them may be facilitated. The stimulus discrimination training session may be used to emphasize this relationship and to identify individual preheadache cues. Intervention with procedures learned by skin temperature feedback may then be attached or associated with these preheadache cues to prevent headache onset or reduce duration and intensity.

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Appendix A

Letter to Physicians

Dear Doctor,

The student bearing this letter has expressed an interest in participating in an experiment being conducted through the Department of Psychology at the University of Manitoba by the undersigned. We are attempting to identify student migraineurs as subjects for an investigation of an experimental treatment of migraine headache. The proposed treatment is non-medical and involves learning a specific response to be used when migraine headache occurs. Students already receiving medication for migraine will not be requested to discontinue medication.

As psychologists, we rely on the diagnosis of migraine by a physician. We request your cooperation in this research by offering a diagnostic opinion of this student's headaches. In so doing, please note if any central nervous system involvement, or other vascular dysfunction is suspected. If the student's headaches are diagnosed as migraine, please indicate whether the headaches are considered classic migraine, common migraine or some other sub-diagnosis.

Your cooperation with this research in providing a diagnostic opinion of your patient is appreciated. If you have any questions or would be interested in discussing

Appendix A (cont.)

the experiment with one of us, please write or call the number listed below.

Sincerely,

John Baldwin,M.A. David Martin,Ph.D.

109 Fletcher Aruge
Univ. of Manitoba
Winnipeg, Man. R3T 2N2

phone: 474-8169

. . . . . .

Date:

Re: Non-medical migraine treatment study.

I have diagnosed the headaches of _____

as: common migraine

classic migraine

other (please specify)

In addition, I do not suspect other vascular or central nervous system involvement.

Signed,

_____

Office address:

Office phone:

Appendix B

Headache Data Record

Name:

Indicate for each headache:

Date/ time at onset:

Migraine or other headache (M or O):

Duration (hours):

Maximum intensity (1-5; 5=most, 1=least):

Medication and amount:

Complaints accompanying headache - be specific:

Your location at onset:

Indicate use of control procedure, time started, and how long:

Remarks:

Appendix C

Preheadache Cue Record

"Increase Plus" Group Only

For each day, describe:

Warnings

- Physical
  - N/V
  - sensory disturbances
  - speech disturbances
  - cold extremities
  - low blood sugar, hunger
  - retention of water
- Emotional
  - alteration of mood
  - lonely
  - irritable
  - depressed

Triggers

- Diet
  - alcohol
  - citrus fruits
  - cheese
  - chocolate
  - fatty fried food
  - food additives (sodium nitrate, MSG)
  - nuts
- Stress
  - anxiety
  - anger
  - emotional change
  - depression
- Sleep
  - irregular patterns
  - fatigue
- Hormonal
  - menstruation
  - pill

Other

Appendix D

Pretraining Migraine Questionnaire

Name:

Age: Faculty: Major:

Occupation:
(Please also mention any part time work.)

How long have you had migraine headaches?

How long ago did you first contact a physician about your headaches?

What medication are you presently taking for migraine headaches?

How satisfied are you with the medication treatment?

Please circle one.

Very satisfied

Satisfied

Dissatisfied

Very dissatisfied

Please list age and sex and relationship to you of relatives whom you believe also have or had migraine headaches.

Please list any signs which make you aware of an impending headache, before the headache begins.

Please rate in order of importance to you the changes which you would most value resulting from headache treatment. (4 signifying greatest importance, 1 least)

headache duration

headache intensity

headache frequency

reduction in use of headache medication

Pretraining Migraine Questionnaire (cont.)

What is your concept of the possible cause of your migraine headaches?

Based on what you know about biofeedback, rate your expectation for this treatment to improve your headaches. (circle one)

1) Biofeedback training will not help my headaches.

2) probably will not help

3) might help

4) probably will help

5) will help

Appendix E

Posttraining Migraine Questionnaire

NAME:
Date of birth:
Mailing address:

1) Please rate your confidence in temperature training to have a beneficial effect when used with a migraine. Rate on a scale of 1-7, with 1 representing lowest confidence.

2)Please rate your ability to control finger temperature as instructed, using biofeedback. Rate on a scale of 1-7, with 1 representing low ability.

3) Please rate your ability to control finger temperature as instructed without biofeedback. Rate on a scale of 1-7, with 1 representing low ability.

4) Please rate your ability to control a migraine by initiating the response learned in training. Rate on a scale of 1-7, with 1 representing low ability.

5) How often are you able to control a migraine by initiating the response learned in training?
consistently frequently seldom never

6) What is your understanding of how temperature training and control may help you with migraines?

7) What strategy or strategies do you use to control finger temperature as instructed?

8) Please rate how effective each strategy seems to be in controling skin temperature when used without biofeedback. Rate on a scale of 1-7, with 1 representing ineffective.

9) Please rate the feeling of degree of warmth you usually have in your dominant hand.
hot warm moderately warm moderately cool cool cold

10) Please rate the feeling of degree of warmth you have in your dominant hand after attempting to control skin temperature as instructed.
hot warm moderately warm moderately cool cool cold

Posttraining Migraine Questionnaire (cont.)

11) How often can you subjectively perceive a change in feeling of degree of warmth while attempting to control temperature as instructed without feedback?
consistently frequently seldom never

12) Please rate your degree of relaxation during the last training session. Rate on a scale of 1-7, with 1 representing deeply relaxed.

13) How many times a week did you practice control of skin temperature as instructed?

14) Please provide the name of the doctor who diagnosed your headaches for this training procedure.

15) What was the approximate date of the above diagnosis?

16) How many other times have you seen a doctor about headaches?

17) What medication has been prescribed for your migraines?

18) How else do you seek relief from migraine?