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SKIN TEMPERATURE FEEDBACK WITH AUTOGENIC TRAINING AND HOME PRACTICE IN THE TREATMENT OF CHILDHOOD MIGRAINE HEADACHES: A CONTROLLED GROUP OUTCOME STUDY

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SKIN TEMPERATURE FEEDBACK WITH AUTOGENIC TRAINING AND HOME PRACTICE IN THE TREATMENT OF CHILDHOOD MIGRAINE HEADACHES: A CONTROLLED GROUP OUTCOME STUDY

A Dissertation
Submitted to the Graduate Faculty of the Louisiana State University and Agricultural and Mechanical College in partial fulfillment of the requirements for the degree of Doctor of Philosophy in The Department of Psychology

by
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ABSTRACT

Childhood migraine is a prevalent disorder seen in pediatric practice. Preliminary uncontrolled reports evaluating skin temperature biofeedback with autogenic training have suggested that it may be a useful intervention for childhood migraine. The present study used a controlled group outcome design to further evaluate the effectiveness of skin temperature feedback with autogenic training and home practice.

Subjects were 28 children, ages 7 to 16, 14 males and 14 females. After receiving a physician's diagnosis of migraine a second diagnosis of migraine was made by the experimenter. Children were matched by baseline headache intensity, sex and age and then randomly assigned to either a waiting-list control or treatment group.

Six dependent measures of headache activity were obtained from weekly headache records that the child and parents kept during baseline, treatment and follow-up. Assessment of ability to increase skin temperature without feedback was made at pre- and post-treatment sessions.

The waiting-list control group attended 2 attention placebo sessions during the baseline period and one at the end of treatment. The treatment group participated in two
pre-treatment measurement sessions and 10 treatment sessions with home practice. Nine sessions included analogue skin temperature feedback and self-control phases. The tenth consisted of self-control only.

Results of a 2x3 analysis of variance with one repeated measure found the treated group was improved significantly on headache index, frequency, duration, highest headache intensity rating and average peak headache intensity rating at the end of treatment as compared to the waiting-list control group. At the one-month follow-up these headache variables were still significantly improved for the treated group, and their medication index was also significantly reduced as compared to the waiting-list control group.

Analyses of the skin temperature data showed the treated group significantly increased skin temperature without feedback at the end of treatment as compared to pre-treatment performance, but scores were not significantly different from the waiting-list control group's scores.

The study is the first controlled experimental demonstration that skin temperature biofeedback with autogenic training and home practice is an effective treatment for childhood migraine.
INTRODUCTION

Migraine headaches are one of the most common disorders seen in pediatric practice, with an incidence of approximately 50 cases per 1000 children or 4-5 percent incidence of children between ages 7 and 15 years (Bille, 1967; Brown, 1977). The prevalence of migraine increases with age and is rare under 2 years of age. Before puberty, the prevalence of migraine is equivalent for males and females. After puberty, the ratio changes with more women reported than men as migraneurs (Waters & O'Connor, 1975; Thompson, 1980).

Williamson (1981) has noted that although migraine headaches have generally been considered a clinical problem of interest to the medical profession, in recent years more physicians and psychologists think of most headaches to be of psychological origin. Behaviorally oriented psychologists have begun to successfully apply behavioral principles to the assessment and treatment of headaches. They have also contributed theories concerning the development and maintenance of migraine headache. There is an extensive literature concerning etiology and treatment of headaches with adults, especially migraneurs. However there are relatively few studies of headaches in children (Brown,
1977; Thompson, 1980). Thus much of the information on headache symptomology, pathophysiology and treatment presented in this paper is based on research with adults.

Clinical Symptomology

Migraine headache, as proposed by the Ad Hoc Committee of the classification of headaches (1962), has two diagnostic categories, classic and common. In the classic form the headache consists of three phases. During the first phase, prodromal symptoms are usually reported. Prodromal symptoms usually occur between 10 and 30 minutes before onset of the headache. Common prodromes are scotomata, flashing lights, fortification spectra, abdominal pain, vertigo and parathesias of the face or hands (Williamson, 1981; Prensky & Sommer, 1979). Many nonspecific changes may occur in the prodromal phase. In children, specifically, increases in bedwetting, nightmares, somnabulism and sleep disturbances may precede the headache (Thompson, 1980).

During the second phase, the migraine headache occurs. The headache phase is characterized by the onset of throbbing or pulsating, unilateral pain. The pain occurs most often in the temporal, orbital, supra-orbital or occipital cranial regions. Head pain is usually accompanied by nausea, photophobia, and constipation or diarrhea. Local edema often results around the affected area of the head. Edema exacerbates the pain and may leave the area sensitive after the headache has ended. In the final phase, post-headache, most children report a feeling of exhaustion.
Some are very talkative and occasionally euphoric. Those who retain fluid during an attack may experience polyuria (Thompson, 1980).

Common migraine headache is similar to classic migraine except pain is not well localized and often has a bilateral location. Prodromal symptoms are not experienced and headaches tend to be somewhat longer (Adams, Feuerstein & Fowler, 1980).

Congden and Forsythe (1977) have noted that no one definition of childhood migraine has gained universal acceptance. Prensky and Sommer (1979) have enumerated criteria that they believe are suitable for diagnosis in children. The criteria suggested by Prensky and Sommer are often used for studies of children with migraine headaches (Congden & Forsythe, 1979; Jay & Tomasi, 1981). Their criteria are similar to those suggested by the Ad Hoc Committee on the Classification of Headache (1962). Prensky and Sommer specify that the headaches must be separated by symptom-free periods and insist that at least three of the following six symptoms occur: 1) nausea and vomiting, 2) unilateral pain or hemicrania, 3) pulsating pain, 4) relief after rest, 5) visual, sensory or motor prodromes and 6) history of migraine in immediate family. Using the six symptoms as criteria, both common and classic migraine types are included. Prensky and Sommer's diagnostic criteria were used in defining the subject population for the present study.
The frequency of migraine occurrence may vary widely from individual to individual, but at least one episode per month has often been reported for children (Bille, 1967). There is evidence that migraine headache episodes are generally shorter in children, usually lasting an hour or more, but rarely longer than 12 hours (Vahlquist & Hackzell, 1949; Bille, 1967).

Pathophysiology

Neurological, biochemical and vascular mechanisms have been implicated in the pathophysiology of migraine headaches. It appears that migraine patients may be characterized by greater reactivity of autonomic responses, especially with increased cephalic vasoactivity. A variety of biochemical mechanisms have been proposed to explain the increased vasoactivity that has been demonstrated in cerebral and systemic blood vessels. No one theory explains all the phenomena of migraine, but considerable advances have been made during the past 20 years. Research has been difficult in the area of migraine because it is a transient functional disorder that leaves no permanent structural change after the headache. Furthermore, animal experimentation is not readily applicable to the problem (Diamond & Dalessio, 1978; Walshe, 1969).

Neurological Pain Pathways

Neurological pain pathways for pain sensitive areas of the head have been established (Diamond & Dalessio, 1978). Structures included are the arteries, muscles, scalp and
skin of the head. For pain in the frontal, temporal or parietal areas, the trigeminal nerve is thought to be involved. The glossopharyngeal and vagal nerves are involved in pain of the occipital region.

**Hormonal Theories**

Migraine in females frequently begins at menarche, improves during pregnancy, is aggravated by oral contraceptives and disappears with menopause (Whitty, Hockady & Whitty, 1966). In males, migraine frequently improves during adolescence. Observations such as these imply involvement of sexual hormones, e.g., estrogen, progesterone and perhaps prolactin (Wainscott, ref. note 1). Brown (1977) suggests normal hormonal changes influence the occurrence of headaches in the generally predisposed individual. Dennerstein, Toby, Burrows and Hyman (1970) report migraine frequently increases with decreasing serum estrogen levels. Graham (1981) hypothesizes that histamine as well as antihistamines are increased as a result of hormones during pregnancy. And alternative hypothesis to a direct effect is offered by Stein (1980) who suggests that hormones may act indirectly influencing the metabolism of vasodilating substances such as serotonin. There are several theories describing the role hormones may play in causing, predisposing or diminishing migraine. No one theory has been adequately researched, thus it is difficult to draw conclusions on the biochemical influence hormones have on the production of migraine headaches.
Biochemical Theories

Vasoactive amines have been implicated in the pathogenesis of migraine (Thompson, 1980). Some patients report the onset of headache after ingestion of cheese or chocolate, suggesting an effect of tyramine and plenylethylamine, respectively, on blood vessels sensitive to these substances (Harrington & Harper, 1967). Experimental studies in which migraine patients have been given tyramine have produced conflicting results (Brown, 1977).

Another biochemical theory suggests that changes in platelet aggregation and the blood clotting systems play a role in the migraine attack (Appenzeller, 1969). Platelet aggregation is increased during the prodrome and decreased during the headache. Serotonin, a vasoconstrictor, induces platelet aggregation and platelet serotonin is rapidly metabolized, resulting in vasodilation of scalp vessels secondary to withdrawal from its vasotonic effects (Lance, Anthony, & Hinterberger, 1969; Freidman, 1978). One problem with this theory is that serotonin could be secondary to changes in platelet aggregation, because the serotonin that is released is already contained in the platelets (Diamond & Dalessio, 1978). The serotonin hypothesis also fails to explain the unilaterality and periodicity of migraine. There is limited experimental support for this theory. Studies on the action of serotonin on intra- and extracranial blood flow in humans and animal research have been conflictual (Adams et al., 1980). Two studies using migraine
patients found that intra-cranial injection of serotonin had little effect on the calibar of intra-cranial arteries (Lance, Anthony & Gonski, 1967). Lance et al. did find vasoconstriction of the external carotid with injections of serotonin. Studies investigating the effects of intra-carotid injection of serotonin with animals reported results conflicting with the Lance et al. studies. In the animal studies, constriction of both intra- and extra-cranial arteries resulted with injection of serotonin (Welsh, Haski, & Meyer, 1973; Welsh, Spira, Knowles, & Lance, 1974).

**Hemodynamic Theories**

Hemodynamic studies support Wolff's (1963) classic theory of migraine. Wolff describes three phases of migraine characterized by vascular changes. In the first phase, pre-headache, constriction of intra- and extra-cranial arteries occurs. This reduction of blood supply was thought to cause prodromal symptoms. Most hemodynamic studies have reported that during the pre-headache phase, intra-cranial and extra-cranial vasoconstriction occurs. Skinhoj (1973) reported approximately 20-50% reduction of intra-cranial blood flow that is usually most pronounced in the cortical areas associated with prodromal symptoms of classic migraine patients. Presently, medical researchers have not firmly established the reasons for the absence of prodromes in common migraine. Recent evidence indicates common and classical migraine headache may differ due to degree of vasospasm or differing metabolic demands of the brain (O'Brien, 1971).
Wolff contended that the headache phase was caused by vasodilation of the internal and external carotids. Headache pain results from increased tension within or about pain sensitive arterial walls due to the increased blood flow after vasodilation. Hemodynamic studies have found small increases of intra-cranial blood flow and profound extra-cranial vasodilation during the headache phase (Skinhoj, 1973).

During the post-headache phase the vascular systems, according to Wolff, return to preheadache conditions. Edmeads (1977) found that although blood supply did decrease during the preheadache phase and increased during the headache phase, vascular changes did not correlate precisely with occurrence of symptoms features. Diamond and Delassio (1978) suggest Wolff's theory gives a good description of vascular changes but is inadequate in explaining the etiology of migraine.

**Unified Theory of Migraine**

Biochemical, neurological and vascular theories to date do not give a comprehensive explanation of the phenomenon of common and classical migraine. What is important to note is extra-cranial vasodilation does not invariably produce headache. Thus an adequate theory must be able to explain the simultaneous vascular and humoral changes that result in a sterile inflammation and a unilateral vasospasm of the intra-cranial and extra-cranial arteries. Diamond and Dalessio (1978) have formulated a unified theory of
migraine which presently is the best integrated theory of
the pathophysiology of migraine.

Diamond and Dalessio contend that migraine must be
understood in terms of three parallel response systems,
vascular, biochemical and subjective/behavioral. Unlike
Wolff and others they discuss only two phases, pre-headache
and headache.

Diamond and Dalessio adopt Oleson's (1972) theory of
cerebral blood supply control mechanisms to explain the
vascular changes during migraine attacks. Oleson proposed
two vascular systems which control blood flow within the
cerebrum. The first system, the "innervated cerebral
vascular system" consists of pial and large arteries at the
base of the brain. Adrenergic nerve fibers innervate this
system. The "innervated cerebral vascular system" is
responsive to external or non-local influences. Diamond
and Dalessio suggest that an external event, particularly
a stressful one, can cause vasoconstriction of the arteries
of this system.

The second system is called the "non-innervated cere­
bral system." It consists of parenchymal vessels which
mostly respond to local metabolic needs of brain tissues.
This second system is unresponsive to external events and
non-local influences because it is not innervated by
adrenergic nerve fibers.

Diamond and Dalessio postulate that during the pre­
headache phase, the innervated cerebral vascular system
constricts creating a reduction in blood flow resulting in hypoxia and cerebral acidosis. Classic migraine patients may experience focal neurological symptoms or prodromes at the subjective/behavioral level. In response to these vascular changes the non-innervated cerebral vascular system will vasodilate to meet the metabolic needs of local brain tissue. The large extra-cranial arteries will also become dilated to increase the intra-cranial blood flow if sustained intracranial vasodilation is required. Wolff (1963) and Dalessio (1972) suggest that a defective neurogenic mechanism, an abnormal extracranial vasomotor response, may be characteristic of individuals susceptible to migraine. This "over-reaction" to intra-cranial vasoconstriction results in excessive vasodilation of extra-cranial arteries. Massive vasodilation of extra-cranial arteries results in release of histamine and peptide kinins as well as mechanical stimulation of free nerve endings. During the headache phase, extreme vasodilation and liberation of histamine and peptide kinins result in sterile inflammation and local edema and the behavioral experience of pulsating pain.

This theory stresses the dysregulation of cephalic blood flow rather than serotonin, histamine and other vaso-active substances. Diamond and Dalessio's position is quite a contrast to other theories discussed such as Appenzeller's (1969) and Wolff's (1963). No one theory is supported more than the other by research in the field. However, Diamond and Dalessio present the best integration of existing data
pertaining to migraine pathophysiology. What is important about the unified theory of migraine is that although they do not elaborate on environmental, behavioral and cognitive factors, Diamond and Dalessio do recognize that these factors may play a role in the development of migraine.

Treatment of Migraine Headache

Adult Studies

Although this particular study was concerned with children who have migraine headaches, a brief review of treatment approaches for adult migraineurs is in order, especially since treatment research concerning children is scant. Traditional medical procedures can be classified as abortive, prophylactic, palliative or surgical. Abortive and prophylactic treatments can be effective in alleviating migraine headaches. However, abortive or prophylactic medication may result in serious side effects. For instance, with an abortive medication such as ergotamine tartrate, nausea, vomiting, diarrhea, drowsiness and cramping may result. Development of tolerance to medications used as a preventive measure, e.g., ergotamine and methysergide, may occur and create withdrawal symptoms, (Lucas & Falkowski, 1973). Palliative treatment, usually narcotic medications, may be helpful but varies on an individual basis and abuse of narcotics is possible. Surgical interventions have not proven to be adequately beneficial to warrant permanent structural damage (Adams et al., 1980). Because of the harmful short and long term side-effects
and variable success of medical approaches, behavior therapy has developed a variety of behavioral interventions for the treatment of migraine headaches.

Four behavioral interventions have been found to be more effective than no treatment control conditions. These treatment procedures are skin temperature biofeedback with autogenic training, relaxation training, cephalic vasomotor biofeedback and behavioral self-management. No one treatment has been shown to be more effective than the others. Controlled outcome studies have reported that between 40% and 100% of adult patients have improved after treatment.

Skin temperature biofeedback with autogenic training. Skin temperature biofeedback with autogenic training was one of the first behavioral interventions to be studied. Skin temperature biofeedback involves instructing the patients to raise their skin temperature, usually of their index finger or hand. Feedback can be in the form of visual or auditory signals, which change as a function of changes in skin temperature. Within each session, after a predetermined time, feedback is often withheld and the patient is asked to continue to raise his or her skin temperature. This treatment phase is usually called self-control. Skin temperature feedback is usually combined with autogenic training. This component is used to help patients warm their hands and relax further. Autogenic training consists of having the patient imagine various sensations and instructing how to relax muscles. For instance, the patient is instructed
to imagine his or her hand becoming heavy and warm. Once the patient achieves the feeling of warmth and heaviness, he or she then concentrates on breathing and heart rate, imagining that heart rate and breathing are regular and calm.

Several early reports indicated that skin temperature feedback was effective with migraine headaches (Sargent, Green, & Walters, 1972; Mitch, McGardy, & Iannone, 1976; Solback & Sargent, 1977; Sovac, Kunzel, Sternback, & Dalessio, 1978). Early studies were promising but outcome data were subjective and they lacked experimental control groups. More recently, a controlled group outcome study with one year follow-up data has been reported (Blanchard, Theobald, Williamson, Silver, & Brown, 1978; Silver, Blanchard, Williamson, Theobald, & Brown, 1979). Skin temperature biofeedback with autogenic training was found to be superior to a waiting-list control that monitored headache activity. In addition, at the end of treatment 54% of the subjects were either headache free or much improved (as defined by 50% reduction of headache frequency or intensity). At a three-month follow-up success was 40% and this improvement was maintained at one year follow-up.

Several studies using skin temperature biofeedback without autogenic training have shown skin temperature feedback to reduce headache activity (Johnson & Turin, 1975; Turin & Johnson, 1976; Wickramasekera, 1973; Reading & Mohr, 1976). One controlled group outcome study by Mullinix,
Norton, Hack, and Fishman (1978) compared one group receiving true skin temperature feedback to one receiving false skin temperature feedback. The true feedback group increased temperature better than the false feedback group, but there were no differences between the groups on headache measures. Headache activity for both groups was decreased. Thus it is not clear whether improvements were due to experimental or placebo effects (Williamson, 1981).

Psychophysiological explanations for the effectiveness of skin temperature biofeedback have been of two kinds. Sargent et al. (1972, 1973) first explained the phenomenon in terms of modifying peripheral and cephalic vasomotor responses. Initially he thought that as peripheral vasodilation from hand warming occurred that cephalic vasoconstriction would occur. Recent studies do not support Sargent's hemodynamic theory (Price & Tursky, 1976; Sovack et al., 1978). Results of recent skin temperature feedback studies showed for most cases cephalic vasodilation occurred with peripheral vasodilation during hand warming. However, for some of the subjects in the Sovack et al. study, cephalic vasoconstriction occurred. Sovack et al. hypothesized that skin temperature feedback was effective because it produced a general decrease of sympathetic arousal.

Cinciripini, Williamson and Epstein (1981) have provided an alternate explanation in which they hypothesize skin temperature training procedures produce effects counteracting the intra-cranial vasoconstriction of the preheadache
phase. Counteracting the initial vasoconstriction would reduce the vasodilation of the rebound effect of the headache phase. Support for either of the two theories of the physiological mechanisms underlying the effectiveness of skin temperature feedback have been minimal. Further research is warranted before either of these models can be accepted or rejected.

Relaxation training. Relaxation training has been used for the treatment of migraine headache in six studies (Lutker, 1971; Hay & Madden, 1971; Paulley & Haskell, 1975; Warner & Lance, 1975; Benson, Klemchuk, & Graham, 1974; Blanchard et al., 1978). With the exception of Benson et al.'s study, shortened versions of Jacobson's (1938) progressive relaxation procedure have been employed. Exercises consist of instructing the patient to tighten then relax various muscle groups of the body. Between exercises, suggestions of heaviness, warmth and looseness are given to the patient. As patients become more able to relax their muscles, muscle groups become combined and the number of muscle groups is reduced. As treatment comes to an end, relaxation by recall for use in the natural environment is faded in. Patients are often given tapes of the relaxation procedure to assist in practice at home.

Five of the six studies mentioned lack proper experimental control groups and objective outcome data. The sixth (Blanchard et al., 1978) was a controlled group outcome study which evaluated progressive muscle relaxation
and compared it to autogenic feedback and a waiting-list control group. Blanchard et al. found relaxation training to be significantly more effective than no treatment and equivalent in effectiveness to autogenic feedback. At the end of treatment 88% of the subjects were much improved or headache free. At a 3-month follow-up the success rate was 56% and treatment gains were maintained at one-year follow-up (Silver et al., 1979). At this time, relaxation training appears to be an effective treatment for migraine headache. Blanchard et al.'s study also indicates that relaxation training and skin temperature feedback with autogenic training have similar effects on headache activity. Silver and Blanchard (1978) suggest that these interventions may be operating via the same psychophysiological mechanisms, reduced sympathetic arousal.

Cephalic vasomotor biofeedback. Cephalic vasomotor biofeedback for training in vasoconstriction of extracranial arteries would seem to be a reasonable intervention since migraine headache is caused by vasodilation of extracranial arteries and vasoconstrictive medication such as ergotamine tartrate can alleviate head pain. Studies of cephalic vasomotor biofeedback have used similar methodology. For the majority of the studies only cephalic vasomotor feedback (CVM) was given. In order to record CVM, sensors for the vasomotor response are usually placed on the right zygomaticofacial branch of the superficial temporal artery. A few studies have used electromyographic (EMG) feedback
along with CVM feedback or have given sessions of EMG feedback interspersed between sessions of CVM feedback. EMG feedback gives feedback of local muscle tension. The frontalis muscle region is usually the site used for EMG feedback. During a feedback session the patient is provided with contingent binary feedback.

Six studies examining the effects of cephalic vasomotor biofeedback have reported success with migraine headaches (Feuerstein, Adams, & Beiman, 1976; Sturgis, Tollison, & Adams, 1978; Feuerstein & Adams, 1977; Friar & Beatty, 1976; Bild & Adams, 1980; Cohen, McArthur, & Rickles, 1980). Three of the studies were multiple-baseline designs and two were controlled group outcome studies. The first controlled group outcome study compared a placebo condition to cephalic vasomotor biofeedback (Friar & Beatty, 1976). Results found cephalic vasomotor biofeedback to be superior to the placebo conditions. The second controlled group outcome study compared cephalic vasomotor response feedback, EMG biofeedback and a waiting-list control (Bild & Adams, 1980). EMG biofeedback reduced headache frequency and duration by 50% for at least 50% of the treated subjects, which was not significantly different from the control group. Cephalic vasomotor biofeedback was more effective than the control procedure and was successful in reducing headache frequency and duration by at least 50% for 86% of the treated subjects. Cephalic vasomotor feedback produced greater changes than EMG feedback but these differences were not significant.
Cohen et al. (1980) compared four biofeedback treatments for headache. The four feedback modalities were forehead skin temperature, frontalis EMG, alpha waves and vasomotor response of the temporal scalp arteries. A group comparison found that all patients demonstrated a significant reduction in number of headaches per week with no change in intensity, disability or length of headaches. Cephalic vasomotor feedback was as effective as skin temperature, EMG and alpha wave feedback.

Cephalic vasomotor biofeedback, as evidenced from the controlled group studies, appears to be a viable and more effective treatment for migraine headache patients than no treatment. Also cephalic vasomotor feedback is as successful as skin temperature feedback, EMG feedback and relaxation training in the treatment of migraine headaches.

It is interesting to note relaxation training and skin temperature feedback procedures, both thought to produce cephalic vasodilation, have equal success rates to cephalic vasomotor feedback which produces the opposite effect of vasoconstriction. It is possible that psychophysiological effects of cephalic vasomotor feedback are different from relaxation training and skin temperature feedback. Cephalic vasomotor feedback may be effective in aborting or reducing head pain when vasodilation occurs. Relaxation training and skin temperature feedback, on the other hand, may be effective in reducing or aborting head pain by vasodilation during the preheadache phase or by reducing general sympathetic arousal (Williamson, 1981).
Cohen et al. (1980) in their comparison of four biofeedback procedures, including skin temperature feedback and cephalic vasomotor feedback recorded physiological data throughout the study. Psychophysiological changes noted were consistently small and unrelated to headache outcome. Cohen et al. conclude the effectiveness of the biofeedback procedures were similar and they attribute the success to a nonspecific one such as a relaxation phenomenon or cognitive restructuring of perceived self-control. Thus cephalic vasomotor feedback, although seemingly producing a potent vasoconstriction response effect, may be successful because of its relaxation effect.

In summary, biofeedback and relaxation approaches to migraine headaches appear to be equally efficacious with adult migraine subjects. Although only one or two controlled group outcome studies have been reported for each intervention, results are promising for behavioral approaches to migraine. Replication of the controlled group outcome studies is in order. Component analysis of treatment interventions would be informative, especially for autogenic feedback. Further studies including placebo conditions and false-feedback versus true-feedback needs to be conducted. Physiological mechanisms influenced by feedback and relaxation procedures are not well understood and basic research on this issue is needed.

**Self-management procedures.** A fourth type of intervention that has been reported to be successful with migraine
patients is a comprehensive behavioral self-management package (Mitchell & Mitchell, 1971). Mitchell and White (1977) used a dismantling design to assess the contributions of various elements of the program which included self-monitoring of headache activity, relaxation, self-desensitization and self-management skills, such as thought-stopping and assertion training. Self-monitoring of headache activity had no effect on headache pain. Relaxation training and self-desensitization resulted in 50% reduction of headache frequency with 70.4% of the subjects so improved. The addition of other self-management skills produced even further reduction of headache activity for 100% of the treated subjects. Improvements were maintained at a 3-month follow-up. The results of the self-management program are better than those reported for other behavioral interventions. More research is needed in this area, including replication and component analysis, as well as comparison to biofeedback techniques and relaxation alone.

Child Studies

Traditional treatment of children with migraine headaches has followed one of two approaches. The first and most common treatment method is via drugs as a prophylactic, abortive or palliative agent. Brown (1977) has recommended that drug therapy on a regular basis should be reserved for those children who have frequent and severe attacks that seriously interfere with normal functioning. For nausea or vomiting, prochlorperazine (Stemetil) has been found to be
helpful. For severe migraine, preparations of ergotamine tartrate (Cafergot, Migril) are often used to abort the headache. Mild to moderate headaches can usually be managed by having the child rest and take aspirin or acetaminopen. Friedman (1967) has stressed that drug treatment of headache symptoms requires the utmost prudence, particularly for children, because their response to drugs is predictable only to a limited extent from adult experience. He emphasizes not only the possible toxic effects from continuous use of medications but also adverse psychological consequences of emphasizing only the alleviation of migraine headache symptoms. Drug therapy may provide significant alleviation of migraine headaches in some cases but because of side effects, particularly in long-term use, there is a need for other nonpharmacological treatments of migraine.

The second traditional type of treatment for childhood migraine involves play techniques. These techniques are usually based upon psychodynamic theories. Although play techniques have been reported to be successful for alleviating headaches (Adams, 1967), no objective or systematic studies have been reported. Therefore, the development of alternate, nonpharmacological interventions is needed for such a common, long-lasting and often debilitating disorder as migraines in children.

As discussed in the previous section, several behavioral interventions have been found to alleviate migraine headaches
in adults. It seems reasonable to suggest that these same interventions may be successful for childhood migraine. Only a few behavioral interventions, i.e., skin temperature biofeedback, cephalic vasomotor biofeedback and contingency management, have been studied with children migraneurs.

Four studies have been reported using skin temperature biofeedback with autogenic training and home practice. Pepper and Grossman (Ref. note 2) reported upon the successful treatment of two girls, ages 9 and 13, with migraine headaches. Their study lacked proper experimental control and objective outcome data, but results indicated that this intervention may be useful. An uncontrolled group study (Diamond & Franklin, 1975) tested the efficacy of skin temperature and EMG biofeedback with autogenic training and home practice with children who had common migraine. During a 30-month period, 32 children, ages 9 through 18, were treated and results indicated a decrease in frequency and severity of migraines in 26 of the cases. Three children experienced either a decrease in frequency or severity of headache but not both, 2 children showed no response, and one was "lost to follow-up" (p. 191). No control group was available and no systematic recording of headache variables or follow-up data were reported. Andrasik, Blanchard, Edlund and Rosenblum (in press) presented two case studies of children migraneurs using skin temperature feedback and autogenic training. On visual inspection of graphs of headache activity (intensity, frequency and headache free
days) improvements are noticeable. Both headache sufferers achieved improvement rates of 57% at follow-up on all measures of headache activity.

Labbe' and Williamson (Ref. note 3) utilized a multiple-baseline across subjects design to evaluate temperature biofeedback with autogenic training for the treatment of migraine headache in three children, one girl (age 9) and two boys (ages 12 and 13). Baseline recording of headache for subjects 1, 2 and 3 were taken for 6, 7 and 8 weeks respectively. Each child participated in ten treatment sessions which consisted of skin temperature biofeedback and self-control of skin temperature. Mean skin temperature across sessions showed that all three subjects increased their finger temperature by an average of .57° Celsius during feedback and self-control phases. Self-report of headache frequency duration, intensity and dosage of medication were recorded throughout the study. Results indicated that the average headache rating for the week (headache index) was reduced after treatment. More molecular analyses of frequency, intensity, duration and medication indices found that these variables were also reduced after treatment and were maintained during follow-up at one month. Thus four studies using either single case or single group experimental methodology reported using skin temperature biofeedback for the treatment of childhood migraine. While these findings are promising, they suggest controlled group outcome research with this intervention is warranted.
in order to rigorously evaluate its efficacy.

Ramsden, Friedman and Williamson (Ref. note 4) applied contingency management procedures in the treatment of a 6-year-old girl who had been diagnosed as having migraine headaches. Careful behavioral assessment suggested this case was best conceptualized as an operant pain case since headache reports occurred under only a few stimulus conditions, e.g. work situations, and consistently produced sympathy and relief from responsibilities. A multiple-baseline across settings design was employed, first in school and then at home. A substantial reduction in head pain reports over the 18 weeks of the study was found and the effects of the contingency management procedure were maintained at a 10-month follow-up. Ramsden et al.'s study is interesting because it suggests that environmental consequences may be an important factor in headache reports, especially for children.

Only one case study using cephalic vasomotor biofeedback in the treatment of migraine in an adolescent has been reported. Feuerstein and Adams (1977) reported 4 case studies using EMG feedback and cephalic vasomotor feedback, one of which was a fifteen-year-old girl who had migraine headaches. Headache frequency and duration was not altered by EMG feedback but both frequency and duration were significantly reduced during cephalic vasomotor feedback. Their subject was able to reduce EMG during EMG feedback and reduce blood volume pulse during cephalic vasomotor feedback.
In summary, no large-scale controlled outcome study of behavioral interventions with children and adolescents who have migraine headaches has been reported. Skin temperature feedback with children has been examined more than other interventions, and even for this technique further research is necessary to establish its efficacy for children who have migraine headaches. Shortcomings of studies to date include small number of subjects, lack of random assignment of children to groups, no control group, no daily monitoring of headache activity and lack of specified treatment plan.
PROBLEM

Childhood migraine is a prevalent disorder seen in pediatric practice. Migraine headaches can interfere with a child's home and school life to the point that treatment of this problem is often sought. Traditional medical treatments are available but serious side-effects may result, especially with long-term usage. Behavioral approaches to the treatment of migraine in adults have been successful, a review of the literature indicating that relaxation training, skin temperature biofeedback with autogenic training, cephalic vasomotor feedback with EMG feedback and self-management procedures are more effective than no treatment for adult migraneurs.

Reports of behavioral approaches on the treatment of childhood migraine have been rare. Cephalic vasomotor and EMG biofeedback, contingency management and skin temperature feedback with autogenic training have been reported to be useful interventions. Studies to date have consisted mostly of single-case reports and one uncontrolled group study. Of the interventions evaluated, skin temperature feedback with autogenic training has been investigated most frequently, though only one published and three unpublished studies have been completed. Reports are interesting and promising but
no firm statements can be made concerning the efficacy of skin temperature feedback with children migraneurs from the uncontrolled investigations. Studies to date lacked random assignment to conditions, had no control groups, used small numbers of subjects and did not use objective outcome data. Positive aspects of the studies have been well-defined characteristics of subjects, assessment of situational patterning and clear descriptions of procedures. The clinical significance of such a problem as migraine headaches in children and the lack of relevant outcome data for behavioral treatments with this population indicate research in this area is needed.

The present study was designed to evaluate the effectiveness of a behavioral treatment with children migraneurs. Skin temperature biofeedback with autogenic training and home practice was the intervention studied. Skin temperature feedback with autogenic training was chosen because it is a comprehensive package, has been successful with adult migraneurs and preliminary reports indicate that it may be effective with children. Markman and Gottman (1978) suggest when a new intervention is to be tested the first step is to examine the effectiveness of the program as a whole and if it is shown to be useful, future studies can be employed to dismantle the program.

The design of the present study attempted to overcome some of the problems which existed with studies to date. First, the study was a controlled group outcome study. A
waiting-list control group was compared to the treatment group. The control group was asked to monitor headache activity throughout the study and received two attention-placebo sessions in which baseline measures of skin temperature were collected. Second, subjects were randomly assigned to the treatment or waiting-list control group. Third, objective outcome data of headache activity were employed, similar to measures used in the Labbe' and Williamson (Ref. note 3) study. All children recorded headache activity during baseline, treatment and follow-up phases of the study. Fourth, besides headache activity, self-control of skin temperature was measured before and after treatment of the experimental group and the control group. Skin temperature assessment was done to examine the ability to increase skin temperature after treatment for the experimental group as compared to baseline and to the waiting-list control. Fifth, maintenance of treatment effects was assessed one month after treatment for the experimental group and was also compared to the waiting-list control at follow-up. Sixth, a careful pre-experimental headache assessment was made and only those children whose headaches had no clear situational patterning were included in the study.

Two basic hypotheses were tested.

**Hypothesis 1.** Those children receiving skin temperature biofeedback with autogenic training will improve significantly at the end of treatment and follow-up, as
compared to a waiting-list control group, on dependent measures of headache activity.

Hypothesis 2. Children receiving skin temperature feedback will learn to increase skin temperature about .5° Celsius after treatment as compared to the waiting-list control who will not show an increase in skin temperature when asked to do so.
METHOD

Subjects

Children were referred by pediatricians in the community and solicited through an article in the newspaper. Upon referral, the child and at least one parent were interviewed. The interview was structured using the Biographical Information Sheet and included behavioral assessment for situational patterning of the child's headache and the completion of the Headache Questionnaire by both the child and parent. The Biographical Information Sheet and the Headache Questionnaire were adapted from a previous questionnaire used by Cinciripini et al. (1981) and Blanchard et al. (1978) in studies of headaches. Parents and children were asked to sign a medical consent form. See appendices A through D for interview materials, medical and subject consent forms.

Thirty children were interviewed who meet the criteria of migraine headache. Twenty-eight of the children attended the first pre-treatment desensitization session. The children who attended the first session were matched on age, sex and baseline headache index and then randomly assigned to either a treatment group or waiting-list control group. Ages of the children ranged from 7 to 16, mean age=10.82. Fourteen of the subjects were female and 14 were male.
Criteria for inclusion. To be included in the study the child had to have received a secondary diagnosis of vascular or migraine headache by a physician and report at least 2 migraine headaches per month and also meet three of the following six criteria:
1) headaches are predominantly one-sided
2) headaches are usually accompanied by nausea or vomiting
3) relief after rest
4) positive family history for migraine headaches
5) pulsating or throbbing pain
6) visual, sensory or motor prodromes.

Dependent Measures

The method for computing headache data developed by Blanchard et al. (1978) was employed in this study. Subjects were given headache booklets in which they recorded four times per day (breakfast, lunch, dinner and bedtime) the intensity of the headache on a scale from 0 to 5. The ratings were described as follow:

0 No headache.
1 Very mild headache, aware of it only when attending to it.
2 Mild headache, could be ignored at times.
3 Moderate headache, pain is noticeably present.
4 Severe headache, difficult to concentrate, can do undemanding tasks.
5 Extremely intense headache, incapacitated.

Subjects were asked to record during the baseline phase
the type and quantity of all medications they took for headaches, as well as where and what happened after they reported the headaches to an adult. For the treatment and follow-up phases the children were not asked to record where and what happened after they reported the headache to an adult. Parents were asked to aid the children in recording their headaches and not to begin use of new medications for the headache.

Clinical variables. Data from the headache records were used to generate weekly scores on several clinically meaningful variables. The five scores are as follows:

1) Headache per week--number of discrete daily headaches per week. To be scored as a headache, there had to be a rating of zero before and after each headache for that day.

2) Headache index--average headache rating for the week. The headache index was calculated by summing all of the ratings for a week and then by dividing them by 28 or the number of rating intervals for that week.

3) Highest headache intensity per week--the single highest headache rating for the week.

4) Average peak headache intensity per week--average of the highest headache ratings for each discrete headache experienced each week.

5) Headache duration--average length of headaches of intensity 2 or greater.

6) Medication index--the medication index was computed
by multiplying the number of pills taken by the potency rating. A scale adapted from Sargent et al. (1973) for rating potency of medication was employed (see Appendix E).

Data on the place, time and the consequences of reporting headaches were reviewed and analyzed for situational patterning of headaches. For all the children, no obvious patterns for operant pain were discerned.

**Apparatus**

All experimental sessions were conducted in a two-room laboratory with a one-way mirror. The subject was separated from the experimenter and physiological recording equipment in a semi-sound proof room. The fingertip temperature of each subject was measured using a temperature thermistor and a Analogue-to-Digital Converter, Med Associates (ANL-90). The temperature was monitored from the volar surface of the most distal phalange of the left index finger. Temperature responses were reported in degrees Celsius. Automatic counting of Celsius degrees of the fingertip temperature was accomplished using a Med Associates printout counter. Fingertip temperature biofeedback was provided using a Med Associates feedback volt meter with a full scale meter deflection of 7cm. Feedback sensitivity was manipulated by calibrating the temperature channel so that a 3.0° change produced a full scale deflection of the feedback needle. Also, auditory feedback was provided using a Med Associates voltage controlled audio oscillator and amplifier (ANL-910). The children were given temperature bands (Biotic-Band II) for
home practice. The temperature band was used to indicate finger temperature. It was made of thermochromic liquid crystal, and wrapped around the finger. It has a range of 20° Fahrenheit divided into 2 degree intervals which are indicated on the band by printed numbers.

Procedure

A controlled group outcome design was used with each child being assigned to either the treatment group or the waiting-list control group. The length of baseline for both groups was four weeks. During the baseline phase, subjects were requested to fill out daily headache records. Both groups were asked to attend two pre-treatment sessions and one post-treatment sessions. The first pre-treatment session was designed to desensitize the children to the laboratory. It involved attaching a thermistor to the subject for recording fingertip skin temperature. Temperature response was recorded during all sessions. The subject was asked to sit for 15 minutes without any instructions, then to relax for 15 minutes as best he or she could. For the second pre-treatment and post-treatment sessions, the subject was asked to sit quietly for 15 minutes, then asked to try to raise finger skin temperature as best he or she could.

The waiting-list control group was then instructed to keep headache records during the next two months (the treatment phase for the experimental group) and for one month after this phase. Two months after the treatment of the
For the experimental group, each child participated in 10 treatment sessions lasting about 40 minutes each. These sessions were spaced across a treatment period of 7 weeks, two sessions per week for the first three weeks, one per week for the last four weeks. The first 9 treatment sessions consisted of 2 phases. The first phase lasted 15 minutes and the child was given no instructions except to sit quietly. The first 10 minutes of phase 1 was provided to allow the child to habituate to the situation and to adjust the physiological recording equipment. The final 5 minutes of phase 1 was used as baseline. The second phase consisted of 3 minutes of self-control of skin temperature in the absence of feedback, 15 minutes of temperature feedback and 3 additional minutes of self-control of skin temperature in the absence of feedback. The tenth treatment session consisted of 10 minutes of adaptation, 5 minutes of baseline and 15 minutes of self-control of skin temperature.

For the first treatment session, the operation of the skin temperature feedback system was explained to the child and he/she was given the expectancy that learning to warm the hands is easy to do and will lead to improvement of headaches. Subjects were given autogenic training instructions indicating how to imagine their hands becoming warm as well as how to relax. The instructions were as follows:
"Body reactions can be produced by your brain through thoughts. For example, when you think you are scared of something you may notice that your heart begins to beat faster. Another example would be if you are worried about doing well on a test or competition you may feel funny in your stomach. We are going to train you to produce changes in the temperature in the skin of your hand. If you learn how to do this your headaches will become better. To do this, sit in a comfortable position. You can think of particular images--warm mittens, fireplaces, etc. (explore individual variations and preferences here). Think about these things and let the muscles in your body relax.

"You will notice that when you begin warming your skin temperature the needle on the meter will move to the right and a sound from this speaker will become higher. (The experimenter will demonstrate feedback devices by placing the thermistor first on a cool surface, for decreasing temperature, then by blowing on the thermistor, for increasing temperature.) This lets you know you are increasing your skin temperature and should continue to do so.

"Don't try too hard. If you don't do it right away, relax and try to think of warm things and soon your hand will be getting warm."

For two of the children autogenic instructions did not produce the desired temperature increase. They were then instructed to focus on a certain sensation in their fingers, such as warmth and heaviness in the hand, fingers and arm.
The experimenter stayed in the room with each child and continued verbal shaping until she/he was able to produce increases in skin temperature.

Instructions about biofeedback and autogenic training were repeated during the first 4 sessions. The children were then briefly reminded of the tasks during the remainder of the sessions.

Each child was instructed to practice the autogenic exercises and hand warming for about 10 minutes twice daily at home and to keep a record of home practice in the headache booklet. At the fourth treatment session each child was given temperature bands and was instructed to use it during the remainder of the treatment phase and one-month follow-up when practicing at home. Instructions were as follows:

"To help your headaches go away, and not come back, you must practice at least twice a day for about 10 minutes. Do the same thing that you do when you are here. You can use the temperature band to let you know when your finger is getting warmer. Remember it is your responsibility to practice and it is very important to do so."

In order to increase compliance with headache self-monitoring, all children were given a gold star for each headache booklet they turned in. When a child collected 11 stars they were rewarded with a small present valued at about $1.50. When follow-up records were completed and sent in, the child's name was placed in a lottery to win $30.00.
RESULTS

Data Analysis

**Headache variables.** Dependent measures of headache activity were computed as described in the procedure section. The six variables or scores of headache activity were: headaches per week, headache index, highest headache intensity per week, average peak headache intensity per week, average length of headache of intensity of 2 or greater and medication index. The data from the 4 weeks of baseline, last 2 weeks of treatment and 4 weeks of follow-up were used. An average score for each phase was computed for each headache variable.

Data analyses examined differences in headache activity within groups over baseline, treatment and follow-up, as well as differences between the treatment and waiting-list control group at each phase. A 2x3 analysis of variance (ANOVA) with one repeated measure was employed to test differences between the treatment and control groups on the six variables at baseline, treatment and follow-up.

Post-hoc analyses of simple effects included one-way ANOVAs and the Newman-Keuls statistic. ANOVAs were employed to determine whether groups were significantly different at baseline, treatment and follow-up phases for all headache.
variables. Newman-Keuls tests were used for individual group comparisons of headache data across phases, i.e., baseline, treatment and follow-up. An alpha level of p < .05 was used for all statistical analyses.

**Skin temperature data.** The ability to increase skin temperature was examined for the control and treatment groups by determining the average skin temperature during the two phases of the pre-treatment and post-treatment measurement sessions: 5 minutes of baseline and 15 minutes of temperature self-control. The skin temperature for each phase was the average of 10 second interval recordings during each phase. Difference scores were determined for each subject by subtracting the baseline from the self-control average. Difference scores were determined for the pre-treatment and post-treatment sessions. A two-way analysis of variance with one repeated measure was used to analyze the difference in skin temperature change between the two groups, as a function of the treatment. Due to a temporary malfunctioning of the heating system, which created an unusually cold experimental environment, three children from each group were excluded in the analyses.

**Reliability Checks**

Computation of headache data was independently checked by a second student, using a 20% sample that was randomly chosen from the headache records. One-hundred percent agreement was obtained for frequency of headaches, highest intensity per week and medication index, 98% agreement was
obtained for headache index and average peak intensity per week and 93% agreement was obtained for duration of headaches.

Computation of the skin temperature data was independently checked by a second person and 100% agreement was obtained.

**Headache Data**

Results of the analyses of each headache variable will be discussed separately and then summarized in a section describing the clinical significance of the findings. Skin temperature data will also be presented in a separate section.

**Headache Index.** The results of the analyses indicate that the groups did not differ on headache index during the baseline phase. At the end of treatment, the treated group differed significantly from the waiting-list control, indicating substantial improvement as a function of treatment. The improved headache index scores of the treated group were maintained at the one-month follow-up. Figure 1 displays changes across treatment phases of the mean headache index for the waiting-list control group and treatment groups. Refer to Table 1 for a summary of the statistical analyses. As shown in Figure 1, headache index for the treated group was substantially reduced as a function of the skin temperature biofeedback, where as the headache index of the control group did not improve over the treatment or follow-up periods. Post-hoc comparisons of the treated group's headache index at baseline, treatment and follow-up demonstrated
Figure 1. Mean headache index scores per week for both skin temperature feedback and waiting-list control groups at baseline, treatment and one-month follow-up phases.
scores were significantly lower than baseline value at the end of treatment and at one-month follow-up. No difference between the end of treatment and follow-up headache index was found for the treated group, indicating the treatment effect was maintained. Headache index for the control group remained stable across all three treatment phases.

Table 1

Summary Table of Analyses of Variance for Headache Index

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<th>MS</th>
<th>F</th>
<th>p</th>
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<td>-</td>
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<tr>
<td>Condition</td>
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<td>2.54</td>
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<tr>
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<td>.28</td>
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<td>-</td>
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<td>Within Subjects</td>
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<td>-</td>
<td>-</td>
<td>-</td>
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<tr>
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</tr>
<tr>
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<td>.23</td>
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</tr>
<tr>
<td>Error w</td>
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<td>52</td>
<td>.03</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Total</td>
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<td>83</td>
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<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Headache frequency. The results of the analyses indicate that the groups did not differ on headache frequency during the baseline phase. The treated group improved significantly at the end of treatment as compared to the waiting-list control group. The improved headache frequency of the treated group was maintained at the one-month follow-up. Figure 2 displays changes across treatment phases of the mean headache frequency scores of the waiting-list control and treatment groups. Refer to Table 2 for a summary of the statistical analyses.

As shown in Figure 2, headache frequency for the treated group was substantially reduced as a function of
Figure 2. Mean headache frequency per week for both skin temperature feedback and waiting-list control groups at baseline, treatment and one-month follow-up phases.
the skin temperature biofeedback, where as the headache frequency of the control group did not improve over the treatment or follow-up periods. Post-hoc comparisons of the treated group at each phase of the study demonstrated scores at the end of treatment and at one-month follow-up were significantly lower than baseline values. No difference between the end of treatment and follow-up was found for the treated group indicating the treatment effect was maintained. In contrast, headache frequency for the control group did not change during baseline, treatment or follow-up.

Table 2
Summary Table of Analyses of Variance for Headache Frequency

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<td>26.86</td>
<td>.48</td>
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<td>50.50</td>
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</tr>
<tr>
<td>Within Subjects</td>
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<td>-</td>
<td>-</td>
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<td>Trials</td>
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<td>TrialxCondition</td>
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<td>6.45</td>
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<td>Total</td>
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<td>83</td>
<td>-</td>
<td>-</td>
<td>-</td>
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</table>

Headache Duration. The results of the analyses indicate that the groups did not differ on headache duration during the baseline phase. The treated group improved and differed significantly in comparison to the waiting-list control group. Improved headache duration of the treated group was maintained at the one-month follow-up. Changes across treatment phases of the mean headache duration
scores of the waiting-list control and treatment groups are displayed in Figure 3. Refer to Table 3 below for a summary of the statistical analyses.

Table 3

<table>
<thead>
<tr>
<th>Source</th>
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<tr>
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<tr>
<td>Total</td>
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<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

As shown in Figure 3 headache duration for the treated group was substantially shortened as a function of the skin temperature biofeedback; in contrast the headache duration of the control group did not improve over the treatment or follow-up periods. Post-hoc comparisons of the treated group at baseline, treatment and follow-up indicated a significant reduction of headache duration at the end of treatment and at one-month follow-up. No difference between the end of treatment and follow-up headache duration was found for the treated group, indicating the treatment effect was maintained. Headache duration for the control group did not change across the treatment phases.

Highest Intensity Rating for the week. The results of the analyses indicated the groups were not different on their highest headache intensity rating during the baseline phase.
Figure 3. Mean headache duration per week for both skin temperature feedback and waiting-list control groups at baseline treatment and one-month follow-up phases.
The treated group improved significantly during treatment as compared to the waiting-list control. The improved intensity ratings of the treated group were maintained at the one-month follow-up. Figure 4 displays changes across treatment phases of the mean highest intensity ratings of the waiting-list control and treatment groups. Table 4 summarizes the statistical analyses.

Table 4
Summary Table of Analyses of Variance for Highest Intensity Rating

<table>
<thead>
<tr>
<th>Source</th>
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<th>df</th>
<th>MS</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between Subjects</td>
<td>115.31</td>
<td>27</td>
<td></td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Condition</td>
<td>48.96</td>
<td>1</td>
<td>48.96</td>
<td>19.20</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Error b</td>
<td>66.35</td>
<td>26</td>
<td>2.55</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Within Subjects</td>
<td>67.96</td>
<td>56</td>
<td></td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Trials</td>
<td>21.76</td>
<td>2</td>
<td>10.88</td>
<td>22.67</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>TrialxCondition</td>
<td>21.44</td>
<td>2</td>
<td>10.72</td>
<td>22.23</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Error w</td>
<td>24.76</td>
<td>52</td>
<td>.48</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Total</td>
<td>183.27</td>
<td>83</td>
<td></td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

As shown in Figure 4, highest intensity rating for the control group did not improve. For the treated group, highest intensity rating was substantially reduced as a function of the skin temperature biofeedback. Post-hoc comparisons of the treated group's highest intensity rating at baseline, treatment and follow-up demonstrated scores were significantly lower than baseline values at the end of treatment and at one-month follow-up. No difference between the end of treatment and follow-up highest intensity ratings was found for the treated group. In contrast, highest intensity ratings for the waiting-list control group remained stable across the treatment phases.
Figure 4. Mean highest intensity rating per week for both skin temperature feedback and waiting-list control groups at baseline, treatment and one-month follow-up phases.
Average Peak Headache Intensity Rating. Analyses of the average peak intensity rating found no group differences at baseline. The treated group differed reliably from the waiting-list control group at the end of treatment. The improved average peak intensity ratings of the treated group were maintained at the one-month follow-up. Figure 5 displays changes across treatment phases of the mean average peak headache intensity ratings of the waiting-list control and treatment groups. Statistical analyses are summarized in Table 5.

<table>
<thead>
<tr>
<th>Source</th>
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<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between Subjects</td>
<td>85.37</td>
<td>27</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Condition</td>
<td>41.39</td>
<td>1</td>
<td>41.39</td>
<td>24.49</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Error b</td>
<td>43.98</td>
<td>26</td>
<td>1.69</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Within Subjects</td>
<td>52.91</td>
<td>56</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Trials</td>
<td>11.84</td>
<td>2</td>
<td>5.92</td>
<td>11.17</td>
<td>&lt; .005</td>
</tr>
<tr>
<td>TrialxCondition</td>
<td>13.57</td>
<td>2</td>
<td>6.79</td>
<td>12.81</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Error w</td>
<td>27.50</td>
<td>52</td>
<td>.53</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Total</td>
<td>138.28</td>
<td>83</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

As shown in Figure 5, average peak intensity for the treated group was reduced as a function of treatment whereas the average peak intensity rating of the control group did not improve over the treatment or follow-up periods. Post-hoc comparisons of the treated group's average peak intensity ratings at each phase of the study indicated scores were significantly lower than baseline values at the end of treatment and at one-month follow-up. No
Figure 5. Mean average peak intensity per week for both skin temperature feedback and waiting-list control groups at baseline, treatment and one-month follow-up phases.
difference between the end of treatment and follow-up average peak intensity ratings was found for the treated group, demonstrating the treatment effect was maintained. Average peak intensity ratings for the control group did not differ across treatment phases.

Medication index. Analyses of the medication index indicate the groups did not differ significantly during the baseline phase or at the end of treatment. The groups did differ significantly at the one-month follow-up phase. Refer to Table 6 for a summary of the statistical analyses.

Table 6
Summary Table of Analyses of Variance for Medication Index

<table>
<thead>
<tr>
<th>Source</th>
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</thead>
<tbody>
<tr>
<td>Between Subjects</td>
<td>4878.71</td>
<td>27</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Condition</td>
<td>88.05</td>
<td>1</td>
<td>88.05</td>
<td>.48</td>
<td>NS</td>
</tr>
<tr>
<td>Error b</td>
<td>4790.66</td>
<td>26</td>
<td>184.26</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Within Subjects</td>
<td>2025.49</td>
<td>56</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Trials</td>
<td>298.55</td>
<td>2</td>
<td>149.27</td>
<td>4.84</td>
<td>&lt;.025</td>
</tr>
<tr>
<td>TrialxCondition</td>
<td>121.54</td>
<td>2</td>
<td>60.77</td>
<td>1.97</td>
<td>&lt;.200</td>
</tr>
<tr>
<td>Error w</td>
<td>1605.40</td>
<td>52</td>
<td>30.87</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Total</td>
<td>6904.20</td>
<td>83</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Figure 6 displays changes across treatment phases of the mean medication index of the waiting-list control and treatment groups. From a visual inspection of the medication index means for the two groups, it is clear that even though the treated group's medication index was higher than the waiting-list control at baseline it was much lower than the control group's medication index at the end of treatment. Post-hoc analyses of the data for each group across trials
Figure 6. Mean medication index per week for both skin temperature feedback and waiting-list control groups at baseline, treatment and one-month follow-up phases.
were made because of the noticeable difference in the treated group's medication use and because all other headache variables for the treated group showed a similar pattern of improvement at treatment and follow-up. Post-hoc comparisons of the treated group's medication index at baseline, treatment and follow-up demonstrated medication index was significantly lower than baseline values at the end of treatment and at one-month follow-up. No difference between the end of treatment and follow-up medication index was found for the treated group. In contrast, medication index for the control group failed to change significantly throughout the study. Thus, although the groups were not significantly different at the end of treatment, the treated children still improved substantially in their medication usage as compared to their baseline behavior. This improved medication index was significantly different from the waiting-list group at the one-month follow-up.

Clinical Significance of the Results

The clinical significance of the results for the individual subjects can be presented in terms of the percentage of subjects that were symptom free, improved or not improved at the end of treatment and follow-up. "Symptom free" was defined as a mean score of zero on any given headache variable. "Improved" was defined as half the baseline score for the headache variables, frequency, headache index, duration and medication index. For the headache variables highest intensity rating and average peak intensity rating,
"improved" was defined as an average rating of 3 or less. "Not improved" was defined as greater than half of the baseline score for frequency, headache index, duration and medication index and as a rating of 4 or higher for highest intensity rating and average peak intensity rating.

Table 7 summarizes the percentage of subjects who were symptom free, improved or not improved at the end of treatment and follow-up phases for the treated and waiting-list control groups.

For the treated group greater than 90% of the subjects were either improved or symptom free on headache index, highest intensity rating and average peak intensity rating at the end of treatment and at follow-up. In contrast, none of the children in the control group were symptom free and 14% or less were improved at the end of treatment and follow-up on these same dependent variables.

Frequency of headaches for 72% of the treated group was improved or symptom free. The percent improved or symptom free increased to 93% at the one-month follow-up. In comparison, 93% of the waiting-list control group were rated as unimproved at the end of treatment and at the one-month follow-up.

The duration of headache score was defined as the average length of headaches of intensity of 2 or greater. Thus, of the 50% of the children included in the symptom free percentage at the end of treatment, some children were still experiencing very mild headaches. At the one-month
Table 7.

Percent Symptom Free, Improved or Not Improved

<table>
<thead>
<tr>
<th>Headache Variable</th>
<th>Symptom Free</th>
<th>Improved*</th>
<th>Not Improved</th>
<th>Symptom Free</th>
<th>Follow-Up</th>
<th>Not Improved</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Txt. WLC</td>
<td>Txt. WLC</td>
<td>Txt. WLC</td>
<td>Txt. WLC</td>
<td>Txt. WLC</td>
<td>Txt. WLC</td>
</tr>
<tr>
<td>Headache Index</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>36% 0%</td>
<td>57% 7%</td>
<td>7% 93%</td>
<td>21% 0%</td>
<td>72% 14%</td>
<td>7% 86%</td>
</tr>
<tr>
<td>Frequency</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>36% 0%</td>
<td>36% 7%</td>
<td>28% 93%</td>
<td>21% 0%</td>
<td>72% 7%</td>
<td>7% 93%</td>
</tr>
<tr>
<td>Highest Intensity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>36% 0%</td>
<td>64% 7%</td>
<td>0% 93%</td>
<td>21% 0%</td>
<td>79% 14%</td>
<td>0% 86%</td>
</tr>
<tr>
<td>Average Peak</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>36% 0%</td>
<td>64% 7%</td>
<td>0% 93%</td>
<td>21% 0%</td>
<td>79% 29%</td>
<td>0% 71%</td>
</tr>
<tr>
<td>Duration</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>50% 0%</td>
<td>21% 7%</td>
<td>29% 93%</td>
<td>21% 0%</td>
<td>50% 14%</td>
<td>29% 86%</td>
</tr>
<tr>
<td>Medication Index</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>79% 36%</td>
<td>21% 14%</td>
<td>0% 50%</td>
<td>79% 21%</td>
<td>21% 7%</td>
<td>0% 72%</td>
</tr>
</tbody>
</table>

* Improved scores for frequency, headache index, duration and medication index were rated as improved if they were 1/2 the baseline value. For highest intensity and average peak intensity at score of 3 or less was rated as improved.
follow-up, only 21% were symptom free. However, if improved children were included, 71% experienced relief in headache duration. For the waiting-list control group, 93% of the children's headache duration scores were rated as unimproved at the end of treatment and 86% were unimproved at follow-up.

It is interesting to note a large percentage of both groups improved on medication index, as 79% of the treated group and 36% of the waiting-list control group had discontinued use of medication at the end of treatment. However, 22% of the waiting-list control children who had improved at the end of treatment returned to baseline medication use at follow-up. None of the treated children returned to baseline medication use at the one-month follow-up.

**Skin Temperature Data**

Statistical analyses of the skin temperature data showed that groups differed, though not significantly, at some phase of treatment at the .1 level. Refer to Table 8 for a summary table of the analyses.

<table>
<thead>
<tr>
<th>Source</th>
<th>SS</th>
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<th>MS</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between Subjects</td>
<td>21.92</td>
<td>20</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Condition</td>
<td>.66</td>
<td>1</td>
<td>.661</td>
<td>.59</td>
<td>NS</td>
</tr>
<tr>
<td>Error b</td>
<td>21.26</td>
<td>19</td>
<td>1.120</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Within Subjects</td>
<td>12.87</td>
<td>21</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Trials</td>
<td>.57</td>
<td>1</td>
<td>.574</td>
<td>1.03</td>
<td>-</td>
</tr>
<tr>
<td>TrialxCondition</td>
<td>1.68</td>
<td>1</td>
<td>1.680</td>
<td>3.01</td>
<td>&lt;.1</td>
</tr>
<tr>
<td>Error w</td>
<td>10.62</td>
<td>19</td>
<td>.560</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Total</td>
<td>34.79</td>
<td>41</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>
Figure 7 displays the means at pre- and post-treatment sessions for the two groups. To determine whether the groups were comparable at baseline a one-way ANOVA was performed. The groups did not differ significantly; $F(1,19)=2.90, p > .05$. Inspection of the mean skin temperature difference scores at baseline showed that the waiting-list control group increased their skin temperature by .48 degrees Celsius at the pre-treatment session whereas the treated group's mean skin temperature difference score was decreased by .18. Comparison of the groups at the post-treatment sessions was also nonsignificant; $F(1,20)=.14, p > .05$.

Further analyses comparing each group's mean skin temperature difference scores at pre- and post-treatment sessions were made. The group receiving feedback was able to raise their skin temperature without feedback significantly ($p < .01$) at the end of treatment as compared to pre-treatment performance. The waiting-list control group's pre-treatment mean skin temperature difference score was not significantly increased as compared to post-treatment performance. Also, their mean skin temperature difference score was slightly lower at the post-treatment session.
Figure 7. Mean skin temperature difference scores for the skin temperature feedback group and the waiting-list control group at pre- and post-treatment sessions.

THE HEADACHE DATA SUGGEST THAT SKIN TEMPERATURE BIOFEEDBACK IMPROVES HEADACHE IN ALL PARAMETERS. HEADACHE INDEX, THE MOST GENERAL HEADACHE VARIABLE, WAS SIGNIFICANTLY DECREASED AT THE END OF TREATMENT AND AT FOLLOW-UP FOR THE TREATED GROUP. CHILDREN RECEIVING SKIN TEMPERATURE FEEDBACK ALSO EXPERIENCED A STATISTICALLY SIGNIFICANT REDUCTION IN
the pain level of their headaches as both highest headache intensity rating and average peak headache intensity rating were significantly reduced at the end of treatment and at the one-month follow-up. The time length of headaches was also significantly reduced for the treated group as compared to the waiting-list control at the end of treatment and effects were maintained at the one-month follow-up. Results of the reduction in intensity, duration or severity of the headaches are similar to results evaluating the effectiveness of skin temperature biofeedback with autogenic training with adult migraineurs (Blanchard et al., 1978).

The number of headaches per week was significantly reduced at the end of treatment and at the one-month follow-up, although the difference between groups for frequency of headaches was not as great as the reduction of the severity of headaches. Blanchard et al. did not find a significant decrease in frequency of adult headaches with skin temperature biofeedback. However, the few child studies that have reported using skin temperature biofeedback with autogenic training have all reported both a decrease in frequency and intensity of headaches (Diamond & Franklin, 1975; Andrasik et al., in press; Labbe' & Williamson, ref. note 1; Pepper & Grossman, ref. note 2). Thus the finding of a significant reduction in number of headaches per week as well as a decreased intensity of headache is consistent with previous child studies in the area.

Both groups reduced medication usage at the end of
treatment and their medication scores were not significantly different as compared to each other. Although the groups were not different at the end of treatment, the treated group's medication index score was significantly different at the end of treatment as compared to their baseline usage. The waiting-list control group's medication index was reduced but not to a statistically significant degree. The waiting-list control group's medication usage returned to baseline level at follow-up. In comparison, the treated group's reduced medication usage was maintained at follow-up. Results of a decrease in medication usage is also similar to results of adult studies and preliminary studies with children in the treatment of migraine headaches.

Results of the study were not only statistically significant but also clinically relevant. Examination of percent symptom free or improved suggests that most of the children in the treated group experienced improvement of their migraine headaches. At the end of treatment only 7% of the waiting-list control group had spontaneously improved on headache index, highest headache intensity rating, average peak headache intensity rating, frequency and duration. At follow-up 14% of the waiting-list control spontaneously improved on headache index, highest headache intensity rating and duration of headache, 7% continued to be improved for frequency of headaches and 29% experienced improvement of average peak headache intensity rating. The modest degree of spontaneous improvement for the
waiting-list control subjects are consistent with the general treatment outcome literature of studies using no-treatment control groups (Garfield & Bergin, 1978).

The treated group showed the greatest improvement on the variables concerning intensity of headaches, with 100% of the children either improved or symptom free at the end of treatment and a one-month follow-up. At the one-month follow-up both headache index and frequency were greatly improved (93%) whereas only 71% of the treated children experienced a reduction in duration of headache.

One clinical anecdote may be useful for understanding how some children were able to control headaches. Prior to treatment, one of the treated children experienced parathesias of the left side of his body during the pre-headache phase and extreme pain (usually rated as 5) during the headache phase. The child required numerous visits to the emergency room where he usually received injections of demerol. Following treatment, the child successfully employed the hand warming technique to abort the pre-headache (prodromal) phase, consequently the headache phase did not occur. He attempted to increase his skin temperature in his hand for about 20 minutes whenever he felt the onset of parathesias, which usually began in his left toes. Several other children reported, with great excitement, similar successes in aborting or greatly decreasing head pain by using the hand warming procedure during the prodromal phase.

Children reported enjoying the treatment program. It
is interesting to note that throughout the treatment sessions only 5 out of 140 scheduled treatment sessions had to be rescheduled. High attendance rate is one indication that the children found the sessions to be rewarding and worthwhile.

One potential problem with the results of this study is the use of self-report data. Some may criticize the reliability of children reporting their own pain behavior. During the study, parents and children were often reminded of the importance of accurate and current recordings of headaches. Parents were asked to check daily on the child's headache booklet. Headache booklets were turned in on a weekly basis and checked by the experimenter; if headaches occurred, the experimenter discussed the headache with the child and explored how the child attempted to resolve the pain, e.g., hand warming, medication, sleep. Children received a gold star for each booklet turned in and rarely did a child forget to do so. Prompt response in turning in booklets and experimenter's observation of parent's and child's discussion of headache activity suggest the self-report data was probably fairly reliable.

In summary, analyses of the headache data, observations by the experimenter and reports by the parents of the children's interest and home practice indicate the children were able to successfully utilize the treatment procedure at the clinic as well as in the home. Results of the present study are similar to results of studies using skin temperature
biofeedback in the treatment of migraine with adults. The present study also extends the findings of the Diamond and Franklin (1975) single group study and several case studies (Andrasik et al., in press; Labbe' & Williamson, ref. note 3; Pepper & Grossman, ref. note 2) in that a controlled group outcome design was used to evaluate headache activity and medication intake. Because the study used a controlled group outcome design, it provides support for the external validity of the technique as an effective treatment of childhood migraine headaches. Of the psychological techniques available today, skin temperature biofeedback with autogenic training has been the most widely researched treatment approach for childhood migraineurs. Given the results of the present study, one can reasonably conclude that it is a very effective treatment for childhood migraine. Replication of the present study is important for further support of the effectiveness of the skin temperature biofeedback with autogenic training in the treatment of childhood migraine. Future studies should focus on component analysis of the treatment program to elucidate which components of the program are necessary for reducing headache activity.

The skin temperature data indicate that the children in the treated group were able to significantly increase their finger temperature at the end of treatment as compared to their pre-treatment performance. Prior to treatment the treated group decreased their average baseline
skin temperature by an average of .18 degrees Celsius during the self-control phase. At the end of treatment the children were able to increase their skin temperature by an average of .48 degrees Celsius with no feedback, actually increasing their skin temperature by .66° Celsius as compared to their performance at pre-treatment. Their ability to increase their skin temperature with no feedback at the end of treatment supports the initial hypothesis that treated children would be able to increase their skin temperature by about .50° Celsius at the end of skin temperature biofeedback training.

The finding that the waiting-list control group were able to increase their skin temperature by .47° Celsius prior to treatment was unexpected. They increased skin temperature at the end of treatment but not significantly so as compared to baseline performance. The results of the skin temperature data raise the interesting and controversial question of how is the ability to increase skin temperature related to headache outcome. Researchers are becoming more concerned with the question of the psychophysiological basis of therapeutic benefits of biofeedback and other behavioral interventions and several interpretations have been put forth (Elmore & Tursky, 1981; Cincirpini et al., 1981; Williamson, 1981). Given the number of subjects receiving feedback training and the limited use of psychophysiological assessment the present study can not adequately address the issue of the
psychophysiological basis of the therapeutic effects of skin temperature biofeedback with autogenic training, although some speculation can be put forth.

One hypothesis is that treatment was effective because by using the self-control phase at the home and the clinic, whether or not they actually produced skin temperature changes, a relaxation response was induced. This relaxation response, by decreasing general sympathetic arousal on a daily basis, influenced headache activity. Support for a nonspecific effect, such as relaxation, to be the basis of therapeutic benefit is the Mullinix et al. (1978) study in which false feedback also resulted in decreased headache activity as well as studies comparing and finding similarities in the effectiveness of relaxation training and skin temperature feedback (Williamson, 198). Recent biochemical studies report findings that strengthen the position that biofeedback influences sympathetic adrenomedullary activation by reducing sympathetic tone (Mathew, Weinman, & Largen, 1982). Reduction in plasma catecholamines and platelet monoamine oxidase are thought to occur with biofeedback assisted relaxation. A second interpretation is that skin temperature biofeedback with autogenic training may be successful because of the effect it has on cephalic vasomotor response, particularly during the pre-headache phase. The children who used the technique when they thought a headache would soon occur could often abort or reduce headache pain. This observation suggests the
children may have learned to reduce cephalic vasoconstriction of the pre-headache phase which led to a reduction of the "rebound" vasodilation of the headache phase. Skin temperature biofeedback, then, may have a palliative effect by decreasing general sympathetic arousal and/or a direct effect by reducing the "rebound" vasodilation of the headache phase. It may be the case that the underlying biochemical changes for both of these explanations is the same.

In conclusion, the skin temperature data, although not differentiating between groups at the post-treatment session, did show the treated group was able to significantly increase their skin temperature at the end of treatment as compared to pre-treatment performance. Their ability to increase their finger skin temperature on the average of .48° Celsius is consistent with adult research. There are no normative data on children's hand warming ability with or without feedback with which to compare the results of these skin temperature data. A study investigating normative data on children's hand warming ability is needed. Further studies investigating skin temperature feedback with children should replicate the procedure used to evaluate the ability to increase skin temperature in the absence of feedback, before and after treatment. To shed light on the psychophysiological phenomenon associated with treatment outcome, further studies should include more responses in their psychophysiological assessment, as well as biochemical assessment.
Summary

The present study attempted to address two hypotheses. One hypothesis stated children receiving skin temperature feedback would be able to raise their skin temperature by .5° Celsius and this would be significantly different from the waiting-list control group. Another hypothesis stated children migraineurs receiving skin temperature biofeedback with autogenic training and home practice would significantly improve on selected measures of headache activity as compared to a waiting-list control group.

The first hypothesis was supported in that children receiving skin temperature feedback training were able to significantly increase their finger skin temperature at the end of treatment as compared to baseline performance. There were no significant differences in the waiting-list control group's performance at pre- and post-treatment sessions. Firm conclusions can not be made regarding the skin temperature data as the waiting-list control group did increase their temperature at pre- and post-treatment sessions and their skin temperature difference scores were not significantly different from the treated group at the post-treatment session.

The second hypothesis was strongly supported. The treatment was successful as the treated group did significantly improve on all six headache variables and the waiting-list control did not. Thus skin temperature biofeedback with autogenic training and home practice appears to be an
effective behavioral intervention for the treatment of childhood migraine. The present study's contribution to clinical psychological research is that it is the first controlled experimental demonstration of the effectiveness of skin temperature biofeedback with autogenic training for childhood migraine.
REFERENCE NOTES


REFERENCES


O'Brien, M. D. Cerebral blood flow changes in the migraine headache. Headache, 1971, 10, 139-143.


Appendix A

Biographical Information

Name: ______________________ Date: ________________
Age: ______________________ Sex: __________________
Birthdate: __________________ Race: __________________
Grade: ______________________ School: _______________
Address: __________________ Phone Number: ________

1. Headaches are a problem for me. Yes ____ No ____
2. I take medications for relief of head pain. Yes ____
   No ____
3. I have had headache problems since the age of ____.
4. I have approximately ____ headaches per month.
5. I have been to the doctor for care of my headaches.
   Yes ____ No ____
6. If yes, his diagnosis was ________________________.
7. What, if any, medications has he prescribed for you?
   ________________________________
8. Have you had any of the following:
   eye problem __________
   ear problem __________
   dental problem _________
   sinus problem __________
   head injury _____________
   seizures ________
   other neurological problems _________
   If you have had any of the above, how have they been
   related to your headache?
9. Have your headaches changed in frequency, duration or
   intensity during
   GIRLS-Menstruation   Increase ____ Decrease ____
   BOYS-Beginning of puberty Increase ____ Decrease ____
10. I have been under stress which may be related to my headache. Yes _____ No _____

11. Do you smoke? _____ How much? _____

12. Have you ever suffered from car sickness? Yes _____ No _____

13. Have you ever suffered from high blood pressure? Yes _____ No _____

14. Do you suffer from high blood pressure? Yes _____ No _____

15. Do you have a parent who suffers from headaches? Yes _____ No _____

16. Does any other family member suffer from headache? Yes _____ No _____

17. Is there a seasonal pattern to your headache? Yes _____ No _____
   If yes, when ___________________________________________

18. Do you have difficulty sleeping? Yes _____ No _____
   If yes, please describe your difficulty. ____________________
   ____________________
   ____________________

19. Do you wet the bed at night after a headache? Yes _____ No _____

20. Are you involved in extra curricular activities? Yes _____ No _____
   What are they? ____________________
   ____________________
   ____________________

21. What kinds of grades do you make? ____________________
   Do your headaches interfere with school or homework? Yes _____ No _____

Notes or Comments:
Does your headache occur

<table>
<thead>
<tr>
<th></th>
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<th>SOMETIMES</th>
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<tr>
<td>in school</td>
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<tr>
<td>home</td>
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<td>other</td>
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Do headaches occur during the

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<tr>
<td>morning</td>
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<td>afternoon</td>
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<tr>
<td>sleep</td>
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<td>all day</td>
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<td>weekdays</td>
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<td>weekends</td>
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<td>certain days</td>
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Do headaches occur when the following people are present

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<tr>
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<tr>
<td>parents</td>
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<tr>
<td>siblings</td>
<td></td>
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<tr>
<td>friends</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>teachers</td>
<td></td>
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<td></td>
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<tr>
<td>other</td>
<td></td>
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</table>

When at home
What is the child doing before a headache?

<table>
<thead>
<tr>
<th></th>
<th>NEVER</th>
<th>RARELY</th>
<th>SOMETIMES</th>
<th>USUALLY</th>
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</tbody>
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Notes or Comments:
What happens when a headache is reported?

<table>
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<tr>
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</thead>
<tbody>
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<td>medication is given</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>rest</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>like to be alone</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>special requests</td>
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<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
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<tr>
<td>special treats</td>
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<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>stays home from school</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>does not do housework</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>does not do homework</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
</tbody>
</table>

When at school

Are there certain classes the child reports headaches in?

<table>
<thead>
<tr>
<th>Class</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
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<td>1</td>
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<td>3</td>
<td>4</td>
<td>5</td>
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</tbody>
</table>

What happens when the child reports a headache?

<table>
<thead>
<tr>
<th>Action</th>
<th>1</th>
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<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
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<td>rest</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>medication</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>goes home</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>does not do school work</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
</tbody>
</table>

Notes or Comments:
For the next two questions please refer to the figures below.

1. When I get a headache, the most severe pain occurs in area(s)

2. When I get a headache, I experience pain in area(s):

   only 1
   only 2
   only 3
   only 4
   only 5
   only 6
   only 1 & 2
   only 1 & 4
   only 1 & 6
   only 1, 4 & 6
   only 2 & 3
   only 2 & 5
   only 2, 3 & 5
   only 3 & 4
   only 1, 2, 3 & 4
   only 1, 2, 3, 4, 5 & 6
Appendix B

HEADACHE QUESTIONNAIRE

Name __________________________ Date __________________

DIRECTIONS: The following statements describe symptoms which occur with different types of headaches. Read each statement carefully and then circle the answer which is most correct for you. The 5 possible answers are defined as follows: Always (occurs without exception), Sometimes (occurs approximately half the time), Rarely (occurs only once in a great while) Never (absolutely does not occur and has not ever occurred).

1. I awaken with a headache. 1 2 3 4 5
2. My headache lasts less than 1 hour. 1 2 3 4 5
3. My headache lasts from 1 to 4 hours. 1 2 3 4 5
4. My headache lasts from 4 to 8 hours. 1 2 3 4 5
5. My headache lasts from 8 to 10 hours. 1 2 3 4 5
6. My headache lasts from 10 to 24 hours. 1 2 3 4 5
7. My headache lasts for more than 24 hours. 1 2 3 4 5
8. I have a headache most of the time. 1 2 3 4 5
9. Before or during a headache I have blind spots in visual field. 1 2 3 4 5
10. Before or during a headache I see stars or flashing lights. 1 2 3 4 5
11. Before or during a headache I have double vision or blurry vision. 1 2 3 4 5
12. Before or during a headache bright lights bother me. 1 2 3 4 5
<table>
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<tr>
<th></th>
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<th>NEVER</th>
<th>RARELY</th>
<th>SOMETIMES</th>
<th>USUALLY</th>
<th>ALWAYS</th>
</tr>
</thead>
<tbody>
<tr>
<td>13.</td>
<td>Before or during a headache loud noise bothers me.</td>
<td>1 2</td>
<td>3 4</td>
<td>5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>14.</td>
<td>Before or during a headache I become dizzy.</td>
<td>1 2</td>
<td>3 4</td>
<td>5</td>
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</tr>
<tr>
<td>15.</td>
<td>Before or during a headache parts of my body, eye, hand, mouth, tongue, are numb.</td>
<td>1 2</td>
<td>3 4</td>
<td>5</td>
<td></td>
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<tr>
<td>16.</td>
<td>I take a prescribed medication on a daily basis in order to control headaches.</td>
<td>1 2</td>
<td>3 4</td>
<td>5</td>
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<tr>
<td>17.</td>
<td>My headache begins during the night while sleeping.</td>
<td>1 2</td>
<td>3 4</td>
<td>5</td>
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<tr>
<td>18.</td>
<td>My headache starts after drinking coffee.</td>
<td>1 2</td>
<td>3 4</td>
<td>5</td>
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<tr>
<td>19.</td>
<td>My headache starts after drinking alcoholic beverages.</td>
<td>1 2</td>
<td>3 4</td>
<td>5</td>
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<tr>
<td>20.</td>
<td>I experience car or motion sickness.</td>
<td>1 2</td>
<td>3 4</td>
<td>5</td>
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<tr>
<td>21.</td>
<td>My headache improves after a period of rest.</td>
<td>1 2</td>
<td>3 4</td>
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<tr>
<td>22.</td>
<td>My headache begins after eating certain kinds of food like nuts, hot dogs or chocolate.</td>
<td>1 2</td>
<td>3 4</td>
<td>5</td>
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<tr>
<td>23.</td>
<td>I have sudden attacks of headache.</td>
<td>1 2</td>
<td>3 4</td>
<td>5</td>
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<tr>
<td>24.</td>
<td>My headache is worst at the end of the working day.</td>
<td>1 2</td>
<td>3 4</td>
<td>5</td>
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<tr>
<td>25.</td>
<td>My headache is throbbing or pulsating.</td>
<td>1 2</td>
<td>3 4</td>
<td>5</td>
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<tr>
<td>26.</td>
<td>My headache feels like a tightness or an external pressure (band-like or cap-like).</td>
<td>1 2</td>
<td>3 4</td>
<td>5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>27.</td>
<td>My headache begins on the left-hand side of my head.</td>
<td>1 2</td>
<td>3 4</td>
<td>5</td>
<td></td>
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<td></td>
<td></td>
<td>NEVER</td>
<td>RARELY</td>
<td>SOMETIMES</td>
<td>USUALLY</td>
<td>ALWAYS</td>
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<tr>
<td>28.</td>
<td>My headache begins on the right-hand side of my head.</td>
<td>1 2 3 4 5</td>
<td></td>
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<tr>
<td>29.</td>
<td>My headache begins in my neck, shoulders or the back of my head.</td>
<td>1 2 3 4 5</td>
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<tr>
<td>30.</td>
<td>I have nausea and vomiting with my headaches.</td>
<td>1 2 3 4 5</td>
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<tr>
<td>31.</td>
<td>My headache gets worse if I cough, strain, or lift objects.</td>
<td>1 2 3 4 5</td>
<td></td>
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<tr>
<td>32.</td>
<td>My headache is better if I can loosen up my neck muscles.</td>
<td>1 2 3 4 5</td>
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<tr>
<td>33.</td>
<td>Aspirin, Anacin, Bufferin, Excedrin, BC, Alka Seltzer, or other non-prescription pain medications relieve my headaches.</td>
<td>1 2 3 4 5</td>
<td></td>
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<tr>
<td>34.</td>
<td>I take a prescribed medication to prevent a full blown attack of a headache.</td>
<td>1 2 3 4 5</td>
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<td>35.</td>
<td>My headache begins when I am relaxing or enjoying myself.</td>
<td>1 2 3 4 5</td>
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</table>
Dr. Donald Williamson, Elise Labbe', M.A. and their associates are conducting a study to determine if temperature biofeedback and autogenic training will have the effect of alleviating the pain of migraine headaches in children. If you agree to let your child participate in this study, the following requirements will be expected of you and your child.

First you and your child will be interviewed concerning the nature of your child's headaches, e.g., symptoms and possible causes. The amount of stress in your child's life and other problems or situations that may be related to the headache will be examined. Based upon information obtained in the interview, we will ask some persons to participate in a study to evaluate the extent to which skin temperature biofeedback procedure reduces headache pain.

The persons who accept our invitation to participate in the study will be expected to cooperate in the manner described below: First, each child must obtain written permission from his/her pediatrician. For the first four weeks, you will schedule two meetings with one of the members of our staff at a time that is mutually agreeable. During these meetings physiological measures of facial and arm muscle tension, finger skin temperature, cephalic blood flow, heart rate, and galvanic skin response will be recorded. These measurements will involve placing sensors on the child's arms, legs, and head to detect the various physiological responses. None of these procedures involve methods that would be painful or would produce discomfort. During the first session the child will sit quietly and will be asked to relax. During the second session the child will first be asked to sit quietly and then will attempt to raise his/her skin temperature.

During this initial month of the study and during the remainder of the study, children will be expected to record a rating of their head pain four times daily, i.e., breakfast, lunch, dinner, and bedtime. After the initial four weeks of the study, all children will be randomly assigned either to a group being treated immediately or to a waiting list group. For the waiting list group, children will be asked to wait for 8 weeks before receiving treatment and will meet three times with our staff to report and discuss
headache activity. For the group that receives treatment immediately, children will be asked to attend two sessions each week for three weeks and then one session per week for the next four weeks. A total of ten treatment sessions will be required. In these sessions, a trained therapist will assist the child in learning skin temperature biofeedback. Children will also be asked to practice what they learn at home. Participants will be expected to attend three follow-up sessions at 1, 3 and 6 months after the end of therapy.

At any time during the study, you may discuss your child's progress and ask any questions you may have about the assessment or therapy procedures. Also, you may withdraw your child from the study at any time.

I have read the above information. I believe I understand the study sufficiently to participate. Any questions I had have been answered to my satisfaction. I agree to participate in the study as indicated by signature below.

Signed, Parent ______________________
Child ______________________
Date ______________________

Witness ______________________
Appendix D

Medical Consent Form

From: Dr. ________________________________
Date: ________________________________

I have examined the patient ____________________________
and can see no medical reason that he/she should be unable
to participate in a study to evaluate the effects of
skin temperature biofeedback and relaxation upon migraine
headaches. Therefore the patient has my permission to
participate in the experiment. By giving my permission, I
understand that I am not necessarily endorsing this type
of therapy for headaches and I understand that I am not
legally responsible for the conduct of the therapeutic
methods or the outcome of treatment. If you have any
questions regarding this study, please feel free to contact
Dr. Donald A. Williamson at the LSU Psychology Department.
(Telephone: 388-8745)

My diagnosis of this patient's headache is: ____________

Signature _________________________
# Appendix E

## Potency Rating of Medication on a Seven-point Scale

### 1 (Over the counter drugs)
- APC
- Alka Seltzer
- Anacin
- Aspirin
- Bufferin
- Ceclar
- Comtrex
- Cope
- Datril
- Empirin
- Excedrin
- Midrin
- Nervine
- Norgesic
- Parafon
- Percogesics
- Persistin
- Phenaphen
- Robaxisal
- Sinutab
- Sudafed
- Synalgos
- Tylenol 1 & 2
- Vanquish

### 2 (Sedatives)
- Darvon
- Dilantin
- Fiorinal
- Fiorinal
- Librium
- Mepergan (fortis)
- Periactn
- Phenergan
- Seconal
- Triavil
- Valium
- Vistaril

### 3 (Vasoconstrictors)
- Bellegal
- Cafergot (Cafregon)
- Ergotrate
- Ergostat
- Gynergen

### 4 (Analgesics)
- Codeine
- or other medication with codeine
- examples: Emperin, Mepergan,
- Percogesics, Phenergan and
- Tylenol 3's & 4's.
- Laratine
- Ponstel
- Talwin

### 5 Demerol

### 6 Dilaudid

### 7 Morphine
Elise E. Labbe' was born in New Orleans, Louisiana, February, 1956 and is now married with two children.
Ms. Labbe' completed her B.A., magna cum laude, in psychology with a minor in mathematics at Loyola University in New Orleans. She obtained her M.A. in 1980 while completing the doctoral program in clinical psychology at Louisiana State University. Minoring in philosophy, Ms. Labbe' also passed specialty exams in child clinical and behavior therapy. Most of her publications have been in the area of health psychology and behavioral medicine. She is an active participant in the Association for the Advancement of Behavior Therapy, Southeastern Psychological Association and Psi Chi. Internship requirements were completed in June 1983, in medical psychology at the Department of Clinical Psychology, University of Florida, Medical Center. Ms. Labbe' will begin her professional career as an assistant professor in the Department of Psychology, Virginia Polytechnic Institute and University.
EXAMINATION AND THESIS REPORT

Candidate: Elise E. Labbe'

Major Field: Psychology

Title of Thesis: Skin Temperature Feedback with Autogenic Training and Home Practice in the Treatment of Childhood Migraine Headaches: A Controlled Group Outcome Study

Approved:

Donald A. Williamson
Major Professor and Chairman

William Coyle
Dean of the Graduate School

EXAMINING COMMITTEE:

[Signatures]

Date of Examination:

June 28, 1982