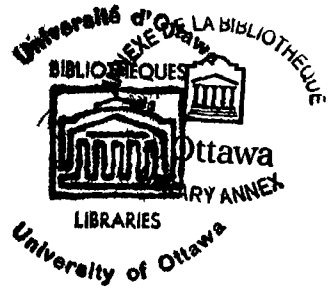
THE EFFECT OF UNILATERAL TEMPORAL ARTERY FEEDBACK ON REPORTED HEADPAIN IN MIGRAINE SUFFERERS

by Robert A. Allen

Thesis presented to the School of Graduate Studies of the University of Ottawa as partial fulfillment of the requirements for the degree of Master of Arts



Ottawa, Canada, 1977

ACKNOWLEDGMENTS

This thesis was prepared under the supervision of William Barry, Ph.D., of the Faculty of Psychology at the University of Ottawa.

Special thanks are due to Gary K. Mills, Ph.D., Edwin Achorn, and J. P. Dionne for their continued interest and helpful criticism throughout this study.

Appreciation is due to Robert Nelson, M.D., and David Grimes, M.D., for their diagnosis and patient referral, and to the Psychiatric Department of Queensway-Carleton Hospital for the use of their biofeedback training equipment.

Finally, a personal thank you to my wife JoAnn Allen for her continued encouragement and support throughout this study.

CURRICULUM STUDIORUM

Robert A. Allen was born in Stockton, California, on July 24, 1952. He earned the degree of Bachelor of Arts from DePauw University, Greencastle, Indiana, in 1975.

ABSTRACT

Migraine headache pain has been shown to be related to the size of the artery in the affected area (Graham & Wolff, 1938). Pain, therefore, can be reduced through the use of drugs such as ergotamine tartrate, a smooth muscle constrictor. The rationale for many nonchemical biofeedback studies has evolved from these earlier chemical studies. Due to the multilevel effect such drugs may have on headpain, it is not specifically known what extent nonchemical vasoconstriction plays in pain reduction. The purpose of the present study is to further discriminate the relationship between reported headpain in migraine sufferers and the operant control of superficial temporal artery size.

Sixteen classic migraine headache sufferers participated in this study. The analyses revealed that both dilative and constrictive control of superficial temporal artery size could be achieved. Eight of these subjects were able to adequately anticipate the beginning of headache in order to come to the laboratory and be observed during the first hour of headpain. Dilative and constrictive control of superficial temporal artery size was maintained during headache. A significant increase in pain during headache was shown while subjects concentrated on the dilation of their superficial temporal artery. Some evidence was presented suggesting that subjects

could also decrease pain during headache. A relationship between the amount of time dilated versus relative pain change magnitude was plotted. Findings were discussed and suggestions for further research were offered.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

Results of ergotamine tartrate studies performed by Graham and Wolff (1938) have shown that pain during migraine headache is directly related to the size of the superficial temporal artery. Therefore, the leading treatment method used for migraine headache relief has been the use of drugs such as ergotamine tartrate (a smooth muscle constrictor). Although chemical treatment for migraine headache is believed to be superior to any other current treatment, there is a risk of drug dependance, side effects, and withdrawal symptoms with the use of such drugs. Due to the recent development of the operant conditioning technique, biofeedback, new hope has evolved for treating migraine headache sufferers nonchemically. The rationale for many nonchemical biofeedback studies has come from these earlier chemical studies. Due to the multilevel effect such drugs may have on headpain, it is not specifically known what extent nonchemical vasoconstriction plays in pain reduction. The purpose of the present study is to further discriminate the relationship between reported headpain in migraine sufferers and the operant control of superficial temporal artery size.

The literature relevant to this study is presented in the first section. In the second section, the method of the experiment is presented. The results of this experiment

are presented in section three, and the discussion of the results, in section four. The last section includes a brief summary of the problem, the results, and conclusions.

REVIEW OF THE LITERATURE

Migraine is one of the most common neurologic disorders, characterized by its variability of symptoms and its periodicity. The concept of the symptom complex of migraine should not be limited to those varieties in which headache is the sole or main symptom and which are commonly known as classic, common, ophthalmoplegic, and hemiplegic migraine. The syndrome also includes cluster headache and a variety of disorders related to vasomotor instability (Friedman, 1975).

Definitions

The general classifications of the various forms of migraine headaches follow (Classification of Headache, 1962):

> Classic migraine. In this type, the headache is recurrent and periodic. Familial and personality factors appear to be important in its pathogensis. As a rule, the prodromes are sharply defined. Contralateral neurologic manifestations, usually visual but in some cases motor or sensory, are common. The visual symptoms include teichopsia, fortification spectrums, field defects, and amblyopia. Frequently, the patient can describe not only his visual sensations but also the exact date and circumstances of his attack. In some patients the visual phenomena are the only indication of migraine. The term retinal migraine is used if the visual hallucinations are limited to one eye rather than to one half of the visual field. In a number of instances, sensory disturbances may develop simultaneously in the fingers and tongue. In about 20 percent of the adults with classic migraine, the headache may occur on the same side as the visual or sensory deficit or may be bilateral. The pain is throbbing and lasts from four to six hours; in the later stages it often spreads to other parts of the head. Anorexia, nausea, vomiting, and photophobia are

concomitant features. Classic migraine occurs in about 10 percent of patients with migraine.

Common migraine. This is the most frequent type of migraine, occurring in over 80 percent of migraine sufferers. The prodromes of common migraine are vague; they may precede the attack by several hours or days. These vary widely from patient to patient and include psychic disturbances, fatigue, gastrointestinal manifestations, and changes in fluid balance. The actual headache episode lasts from many hours to several days. The pain is steady, unilateral, aching, or throbbing. Associated symptoms include anorexia, nausea, vomiting, fatigue, chills, localized or general edema, and diuresis. The occurrence of nasal signs and symptoms may lead the physician to ascribe the headache to involvement of nasal structures. Photophobia and sonophobia are prominent features. This type of migraine commonly occurs on weekends, holidays, and letdown periods.

Cluster migraine. This headache occurs in a series of closely spaced attacks; these may be followed by remissions of months or even years. Prodromes are uncommon. The pain may occur suddenly and wake the patient after an hour or two of sleep. Injection of the conjunction, tearing, nasal stuffiness, occasionally ptosis and miosis, and sometimes unilateral or bilateral sweating are associated manifestations. After 20 or 30 minutes, the pain stops as suddenly as it began. Cluster migraine is most common in men, but may occur in women. Not all agree that this type of headache should be classified as migraine.

Hemiplegic and ophthalmoplegic migraine. This rare type of migraine may occur in the young adult. In ophthalmoplegic migraine the pain is moderate, on the same side as the ophthalmoplegia, and accompanied by extraocular muscle palsies involving the third cranial nerve. Often the paralysis occurs as the headache subsides three to five days after the onset of a persisting headache. The hemiplegic migraine complex is characterized by neurologic deficits, hemiparesis, or even hemiplegia. The neurologic phenomena of both hemiplegic and ophthalmoplegic migraine may persist for some time after the headache has subsided. Recurrent attacks may occur over years or months. Repeated ophthalmoplegic attacks may cause permanent injury of the third cranial nerve (p. 6).

Certainly, the earliest records of man show his recognition and interest in the aching head. Through both rational and superstitious means, man has fought back at the headache. To primitive man the headache was the entrance into the body of an alien spirit, and he sought to remove it by the magic of chants and rituals. In ancient Babylon, a formal incantation existed to exorcise headache. The success of treatment was probably determined by the degree of the afflicted's faith (Friedman, 1972).

Incidence

Migraine headache is still quite prominent today. It affects a substantial minority of the population; occurs in all civilizations; and has been recognized since the dawn of recorded history. Conservative estimates of the incidence of migraine headache alone in North America is 5% of the population (Dalsgaard-Nielsen, 1971). Over 400 million dollars are spent each year on drugs prepared for headache relief (Brody, 1968) and drug dependence is seen as a serious problem among migraine headache sufferers (Friedman, 1969).

Most researchers report that female patients constitute a much larger proportion of the migraine headache population than do men (Dalsgaard-Nielson, 1971; Pearce, 1971). This phenomenon may reflect man's reluctance to seek help and the fact that menstruation and the prominent use of

birth control pills are possible precipitators of migraine headache (Desrosier, 1973; Pearce, 1971).

Age and migraine headache seem also to be associated. Waters, in a 1971 study, states that significantly more women than men had had headache in the preceding year and in both sexes the proportion with headache in the preceding year declined.with increasing age. He concludes that the prevalence of headache varies with both age and sex.

The patient with migraine headache commonly reports that some other member of the family also suffers migraine headache. Fitz-Hugh (1940) suggested that there is a constitutional predisposition inherited by migraine sufferers.

> Migraine sufferers react with headache to a variety of stimuli which, in the non-migrainous members of the population, produce no such comparable effect (p. 146).

There is little doubt that migraine is familial. Dalsgaard-Nielsen (1974) explains that recessive inheritance and regular dominent inheritance may be ruled out, whereas it is difficult to rule out more irregular dominant inheritance. The possibility of a multifactorial inheritance due to several genes may be responsible for migraine headache (Dalessio, 1972).

Psychological Precipitants in Migraine Headache

Though primitive man's spiritual approach to migraine was largely magical and superstitious, today's research suggests that the psychological factors may be significant in triggering migraine headache. The psychological component of pain has also been implicated as being a learned reaction and may be passed from parent to child. Dalessio (1972), during his years of work with migraine headache patients, has described the migraine syndrome in relation to children and adults.

Children were often delicate or treated as delicate, shy, withdrawn, and extremely obedient to their parents. They are generally well-mannered and conscientious. Their docile character was often sharply contrasted with stubbornness and inflexibility in certain instances. The apparently contrasting qualities of the child's character was a prominent feature in the child's personality. Children seemed to be restrained and proper in social situations but on occasion displayed a "chip on the shoulder" deportment. In adults the character was described as more accentuated and definite. Almost all subjects were unusually ambitious and achievement oriented. They were described as meticulous, exacting, persistent, perfectionistic, and whenever possible tried to bring order to their world (Dalessio, 1972).

Bihldorff, King, and Parnes, in a 1971 study of psychological factors in migraine, found that their results were largely in agreement with existing clinical literature. They described the migraine patient as one who reacts to many life situations with aggression, but who is, nevertheless, unable to express his anger directly and satisfactorily. The typical migraine patient is described as overcontrolled in his behavior and as having personality traits which lead others to regard him as a "nice" and "agreeable" person. A number of authors conclude that the key psychological element represented in migraine headache is unexpressed anger (Alvarez, 1974; Friedman et al., 1954).

Harrison (1975) reviewed studies in which the Minnesota Multiphasic Personality Inventory results were obtained. The major finding from these studies is that migraine patients think poorly of themselves. They generally describe themselves as more anxious, overwrought, tense, vulnerable, and depressed than do controls. Migraine sufferers had elevated scores on Psychasthenia, the obsessive-compulsive scale. Migraine profiles are also characterized by high scores on Hypochondriasis and Hysteria. Many studies report a significant relationship between migraine headaches and depression (Kashiwagi, 1972; Selby, 1960); however, in those studies reviewed by Harrison, depression scores on the MMPI were not elevated.

Another aspect of migraine headache in which there is a large psychological component is pain. Fisher (1970) explains that:

> in quest of a solution to the problem of pain, each clinical and psychological approach contains its preconceived concepts of cause. The eventuation of mental or psychologic pain in physical pain is one of the more tenuous physiological concepts, because its tenets still embrace what pure researchers label suppositions and speculations. At the same time, whatever approach we are prejudiced to follow, we cannot disassociate ourselves from the psychosomatic one entirely (p. 74).

Fisher goes on to say that it is the well-adjusted, verbal, socially proficient individual who is most often afflicted with pain coming episodically. These people seem to use the pain release mechanism to divest themselves of affect which would be discordant with their outward self-concept. He describes the mechanism of pain as:

> affective pressure within the nervous system will be felt through downward transmission into the visceral brain. At this point, the autonomic nervous system is able to take over the problem, which it does according to its auspices over the peripheral and vegetative systems. The resultant pain may be migrainous, lumbar-sacral, anginal, epigastric, or facial (p. 84).

Fisher, therefore, views pain as a release mechanism in which the individual's personality plays a major role. Reflecting upon what is known of the characteristics of the migraine personality, migraine headache must pose a particularly irritating problem to its sufferer.

Merskey (1970) defined pain as "an unpleasant experience which we primarily associate with tissue damage or describe in terms of such damage or both" (p. 116). Migraine headache pain, however, is largely experiential, and rarely results in physical damage. It is very difficult to definitively state whether or not anyone suffers from pain or to what extent the suffering exists. Pain experience is thought to be learned, though the extent the psychogenic element plays in the total pain experience is unknown. It is suggested that early perceptual experience determines, at least in part, the way in which responses to noxious stimuli occur later (Melzack & Scott, 1957; Morgenstein, 1964; Simmel, 1962). Information on cultural and social factors in the experience of pain can also be adduced to support the idea that there are learning effects on the pain experience (Mersky & Spear, 1967). The evidence is clear, that patterns of pain related to various parts of the body are learned. There is also evidence that the appraisal of noxious stimulation and the experience of pain depends upon an interaction between the maturing organism and its environment.

On one hand, the traditional model of medical pain is linked to the concept of objective causality. "On the other hand," states Fisher (1968), "pain can not only signal danger but also a message of requesting help from another

person, or a means of communicating the idea of being unfairly treated, abandoned, unloved, or punished" (p. 86).

Other precipitating factors in migraine headache include foods, atmospheric pressure, electrically charged air, hormonal changes due to menstruation, and the administration of oral contraceptives (Migraine Foundation, 1974).

Major Headache Theories

Two major areas of study regarding migraine headache have been clearly synthesized by Sacks (1970).

Chemical theories of migraine. Chemical theories of migraine headache have risen from the obvious autonomic components of the attack. The most obvious and common symptoms of migraine are the vascular components, dilation of extracranial vessels and the nausea and increased glandular activity.

The histamine theory of migraine is associated with the name of Horton (1956), who proposed that some migraine headaches arise from a form of histamine sensitivity. He stated that in some patients, migraine headache attacks could be induced through injecting histamine, and be averted through histamine desensitization. Horton's successes in treating patients through histamine desensitization have not been sufficiently duplicated to rule out simple placebo

effect, and attempts to demonstrate elevated levels of histamine in migraine have failed.

The acetylcholine theory of migraine is associated with the name of Kundle (1959), who examined acetylcholine levels in the spinal fluid during attacks of migrainous neuralgia. Though Kundle's research was moderately successful, his findings have not been duplicated. It is still not known what extent acetylcholine plays in a migraine attack. The acetylcholine studies and histamine studies have been displaced from the attention of researchers due to the rise of the serotonin theory.

The serotonin theory of migraine launched by Sicuteri (1950) grew from the findings that methysergide, a potent serotonin inhibitor, could be used as a migraine prophylactic. Lance et al. (1967) directly measured plasma serotonin before, during, and after migraine attacks, and observed a drop in serotonin at the onset of migraine headache. Injections of serotonin into the carotid arteries produces tonic constriction at extracranial sites. Lance postulates that a lack of serotonin may cause the painful distension observed in migraine headache.

In summary, evidence presented by the advocates of the histamine and acetylcholine theories seems weak and questionable. The evidence presented by Lance et al. (1967) and others who have studied the involvement of serotonin in migraine headache seems encouraging.

Electrical theories of migraine. Direct examination of cerebral activity during migraine headache is unfeasible. Researchers, therefore, must limit themselves to electroencephalographic (EEG) studies and interpret these findings.

Gibbs and Gibbs (1951) were among the first to study EEG activity in migrainous patients. Since that time, many abnormalities have been associated with migraine, such as: generalized slow wave dysrhythmias (Dow & Smitty, 1947; Engel et al., 1945; Selby & Lance, 1960; Strauss & Selinsky, 1941); convulsive patterns (Gibbs & Gibbs, 1951; Whitehouse et al., 1967); and focal abnormalities (Engel et al., 1945). However, there fails to appear a specific pattern to migraine as does epileptic wave and spike patterns. Most studies have not obtained tracings during the actual migraine aura or headache, for there is no reliable method of eliciting a migraine. Reasons given by researchers for their inability to find characteristic patterns include: they cannot justify exposing the brain or using deep electrodes; they cannot identify migraine in animals; and that EEG monitoring may be highly inappropriate for studying the migraine process.

It is certain that some form of electrical disturbance accompanies migraine. Lashley (1941) plotted his own scotomata and estimated that its enlargement was due to a wave of excitement moving across the cortex at about 3 mm per minute, followed by a wave of total inhibition. Milner (1957) has

remarked on the quantitative similarity of scotomata spreading and that of Leão's spreading depression.

Much is still unknown of the relationship if migraine headache and electrical activity. With the advent of more sophisticated monitoring equipment, researchers may, in the future, determine an underlying physiological cause which produces electrical abnormalities and vasomotor changes in the migraine patient.

Vasomotor Activity and Pain

Pain experienced in migraine is vascular in origin and primarily associated with extracranial vasodilation, specifically localized in the external carotid arteries (Graham & Wolff, 1938). The injection of ergotamine tartrate (a smooth muscle constrictor) reduced the amplitude of pulsation of these scalp arteries and at the same time patients reported reduction and relief from headpain. Graham and Wolff also recorded the pulsation of cerebral spinal fluid as an indirect method of measuring dilation of intracranial vessels. They found no significant reduction of these vessels after the injection of ergotamine tartrate.

Later studies by Schumacher and Wolff (1941) found that by increasing intracranial cerebral spinal fluid and thereby decreasing intracranial vessels, there was an immediate relief of histamine headaches, but when the same method was

used with migraine patients, no relief was found. These authors concluded that dilation of intracranial vessels is responsible for histamine headache, and that dilation of extracranial vessels causes migraine headpain. Further evidence of the vascular nature of migraine was shown by Ceres (1941). Patients were spun in a human centrifuge at a positive acceleration of 2.0 Gs with complete relief of headache. Results were attributed to the hydraulic reduction of blood engorgement in the cranial arteries.

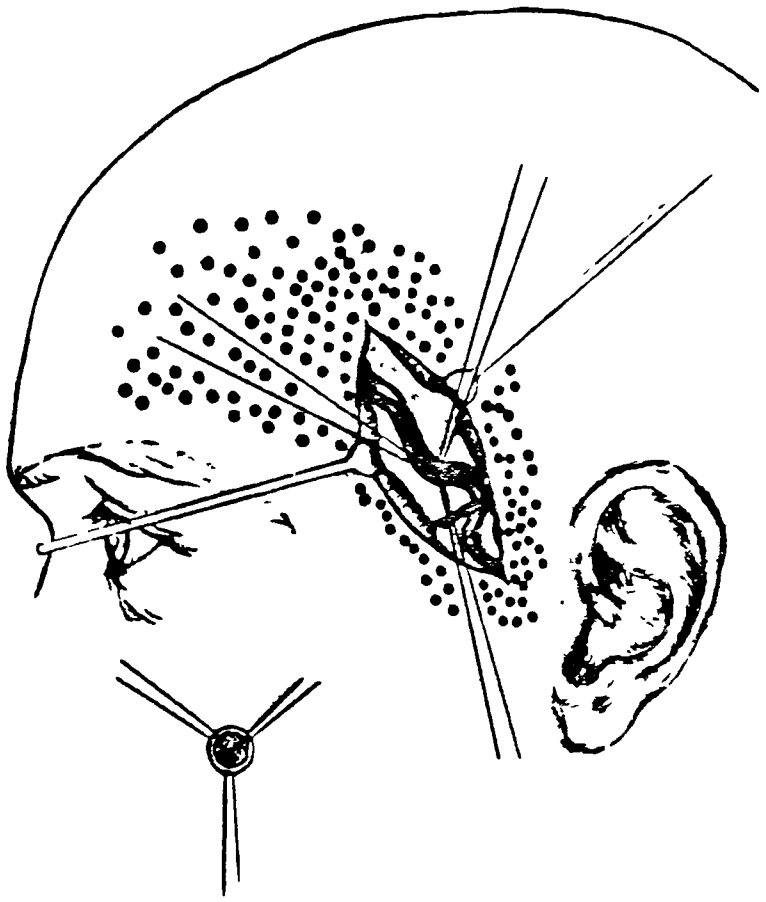
In a surgical study, Ray and Wolff (1940) showed that artificial distention of a superficial temporal artery produced pain in the temporal region (Figure 1). Pain persisted as long as distension was maintained and promptly ceased when discontinued. When two separate sites were distended simultaneously, the headache resulting from this distension was associated with a feeling of nausea or sickness, characteristic of migraine.

These studies make it clear that migrainous headpain may be caused by the dilation of scalp arteries. An interesting corollary to migraine headache is that a normal individual experiences headache after strenuous exercise. One explanation is that the dilation of the capillaries does not keep pace with the arteries during migraine, so the artery wall becomes over distended (Lance, 1973). An analagous situation is presented by Hertzman and Roth (1942). They found that when

The arteries of the scalp; ● indicates the point of stimulation causing pain. The diagrams show the area of pain following stimulation of (1) the occipital arteries; (2) the supra-orbital and frontal arteries; and (3) the superficial temporal artery.

Figure 1. Points of Artery Distention Used in the Surgical Study of Migraine (Ray & Wolff, 1940).

cold is applied to the forehead of normal persons, pain is experienced. They suggested that when the small vessels constricted, the large vessels must overdistend to accommodate the blood flow. Although the pain experienced by these normal subjects is not the same as that of migraine, the area in which cold was applied and, presumably, where the arteries were distended was the area in which pain was experienced. Similarly, if the arteries become overdistended, pain would logically be experienced in that immediate area, and this was found to be true. In the Ray and Wolff study (1940), previously cited, distension of the temporal artery was produced in six subjects by passing three curved needles with silk thread through the outer layers of the vessel. When the artery was distended, there was no interference with blood flow. It was observed that, by pulling the three threads simultaneously, a well localized pain was produced in the temporal region in an area of about 5 cm in diameter (see Figure 2). By repeatedly and rhythmically distending and collapsing the artery, a throbbing quality could be given to the headache. When this procedure was performed in two separate areas along the temporal artery, the pain was also separately located and corresponded to the site of distension. When both sites were pulled simultaneously, the ache was much more widespread than the sum of the two painful areas produced previously. All adjacent nerves were separated from the artery before the experiment began.



Traction sutures symmetrically placed in the wall of the superficial temporal artery. The mechanical distention of the artery resulting from simultaneous traction on all three sutures caused pain in the dotted area.

Figure 2. Illustration of Temporal Artery Distention (Ray & Wolff, 1940).

Vasomotor Changes During Migraine

Paulson and Skinhoj (1969) have reported their observations in regional blood flow during a migraine attack by using radioactive isotope injections. Regional cerebral blood flow measurements during the prodromal phase revealed a pronounced flow reduction in the entire carotid system. From their findings they postulate that this severe reduction in blood flow to various cortical areas explains the various symptoms of the prodromal phase (i.e., visual disturbances occur as a result of blood flow reduction in the occipital area). During the headache (pain) phase, there was observed a massive dilation of the external carotid arteries suspected to be a "reaction" to the severe vasoconstriction phase. The effect of prolonged dilation is a thickening of the arterial wall called edema (Dalessio, 1972). During this phase the arteries become rigid with a pipelike texture.

> Many patients report that after the first hour or two of a migraine headache, the quality of the headache changes and that pain becomes a steady ache; administration of ergotamine tartrate at this time often fails to give relief (p. 270).

Treatment

Vascular evidence has shown that a relationship exists between chemically induced vasoconstriction and pain reduction of headache relief. The treatment of migraine has,

therefore, been almost exclusively limited to chemical intervention. Friedman (1972) estimates a tenfold increase in chemical headache remedies over the past 25 years. He states, however, that the increase in headache remedies in no way parallels their effectiveness. Diamond (1972), in an extensive review of the drugs used in the therapy of migraine, has reported on the efficacy of various drugs in their reduction of headpain. Effectiveness for these drugs ranged from about 20% to 80% in the reduction or abortion of headache. Friedman and Merritt (1969) found that the frequency and severity of their patients' attacks were significantly decreased in over 50% of the patients. Friedman added:

> The results achieved with these agents were not appreciably different from the results obtained with placebos which registered a 45 percent improvement; with few exceptions our recent studies do not differ from our earlier reports (p. 153).

Although chemical treatment for migraine headache is believed to be superior to any other current treatment, there is risk of drug dependance, side effects, withdrawal symptoms, and increased frequency of headache with the use of such drugs. Among the researchers involved in the chemical treatment of migraine, Lance (1973), Friedman (1972), Diamond et al. (1972, and Dalessio (1972) have all found strong side effects associated with the use of drug therapy. Friedman (1972) reported the problems of drug dependance,

withdrawal complications, along with loss of a feeling of well-being, fatigue, let-down periods and depression as side effects of ergotamine tartrate, the leading drug used in migraine treatment. In the same article, he explains that the difficulty in drug therapy is due to the vast differences in patients' responses to these drugs. He also mentioned the problem of spontaneously recurring headache upon termination of drug usage. Horton (1959) explains that side effects can range from numbness to gangrene with long-term usage of ergotamine tartrate. Raynauds' phenomenon, Buerger's disease, angina and hypertension may be worsened with ergotamine usage. Rowsell et al. (1973) have reported that not only are there side effects with ergotamine and its derivatives, but that in some cases patients report an increase in frequency of headaches while on the medication. It is recommended by Rowsell that only 10 mgm of ergotamine be administered per week. This being the case, many patients would have more headaches than could be safely handled with the drugs prescribed. Due to the large variety of side effects, drug dependance, and withdrawal, many nonchemical approaches have been attempted to relieve migraine headache pain and frequency.

Among the more drastic approaches in the treatment of migraine is surgery. The most widely used forms of surgery are the removal of affected arteries or the removal

of the nerves mediating the area of headache pain. Dalessio (1972) has summarized the use of surgery in migraine headache relief:

> In summary it can be said that in migraine it is not justified to try the effects of interruption of the various extracranial and middle meningeal arteries and their accompanying nerves. The expected results will fall short of what is desired. Good initial results are often temporary. Other types of operation are usually unsuitable, either because they are ineffective or because they may be attended by complications unacceptable in exchange for migraine (p. 608).

There are a number of isolated case studies and original experiments that have not been replicated, involving other treatments for migraine headache.

Howeler et al. (1973) studied the effects of electrostimulation on 11 migraine sufferers. Only moderate results were found and they have reported that their study was probably confounded due to not controlling drug usage.

Hypnosis has been used by various authors (Edmeads, 1973; Greenleaf, 1974), but generally these are case studies and their personal successes may not be an effective guideline as to how others will succeed using hypnosis.

Benson et al. (1974) tested the use of transcendental meditation in the treatment of migraine headache. He concluded that meditation should be considered for patients with severe headache when other forms of therapy have proven unsuccessful, but that transcendental meditation has limited usefulness in therapy of migraine headache.

Edmeads (1973), in a review of the treatment forms currently used in migraine headache, reported that yoga, transcendental meditation, and accupuncture are controversial and that:

> there are yet no adequately controlled studies of any of them that will permit a meaningful evaluation of their efficacy. Until such studies have been devised and executed, these methods must be viewed with a rational blend of open-mindedness and scientific skepticism (p. 94).

Behavior modification techniques were used by Gentry (1973) in a case study. He reported curing a 29-year-old lady by using directive therapy. Mitchell and Mitchell (1971) concluded that a combined treatment program (relaxation, desensitization, and assertive therapy) was superior to single model approaches, obtaining significantly greater reduction in migraine episodes and hours duration of headache.

With the advent of operant autonomic conditioning, new hope has evolved for treating disorders such as migraine nonchemically. Korn (1949) was the first to report evidence that the temporal artery was amenable to Pavlovian conditioning. Later, Trowill (1967) demonstrated that the autonomic nervous system was responsive to conditioning. Trowill injected rats with d- tubocurarine chloride, more commonly known as curare, which anaesthesized the skeletal muscles. Under curare, the rats were totally incapable of voluntary muscle control. Stimulation was given to the rats medial forebrain bundle,

or pleasure center, when the rat's heart rate changed in the desired direction. These rats were thus successfully trained to increase or decrease their heart rates. Miller (1969) demonstrated that the autonomic nervous system was responsive to operant conditioning methods and from his work the operant conditioning method of biofeedback was begun. The first use of biofeedback in the treatment of migraine headache was reported by Sargent, Green, and Walters (1972). Of the 62 patients treated with "Autogenic Feedback Training",[1] 74% were considered improved. The rationale behind their study was: Artery distention is due to excessive sympathetic activity and that through handwarming, learned with the aid of temperature biofeedback, subjects learn to reduce sympathetic outflow. In the migraine headache literature, this study was the pioneer in biofeedback treatment. Although this study was accepted by many researchers, many others have criticized the study on the basis of questionable rationale, subjective reporting, and lack of control groups (e.g., no placebo effect control or control for possible Hawthorn effect) (Blanchard & Young, 1974). The focus of a more

---

[1] Autogenic training involves the simultaneous regulation of mental and somatic functions. The desired somatic responses are brought about by passive concentration upon phrases of preselected words (Schultz & Luthe, 1959).

current approach has been upon the conditionability of arterial size. Christie and Kotses (1973) were the first to study the conditionability of the cephalic vasomotor response (or the ability of subjects to dilate or constrict the temporal artery through operant conditioning methods). The conclusion of this study was that this operant conditioning method was successful in cueing subjects in the change of their vasomotor response. The study was of a preliminary nature and there were only four subjects in each group. However, it did apparently stimulate a new approach to treating migraine. Koppman et al. (1974), in a similar biofeedback experiment, used this operant conditioning method to give direct feedback of blood volume pulse amplitude from the superficial temporal artery. The blood volume pulse amplitude was measured with a photoelectric plethysmograph. His data indicated that 7 of the 9 migraine patients achieved both dilative and constrictive control of the superficial temporal artery. However, all data was obtained while subjects were not experiencing headpain. Feuerstein and Adams (1975), using superficial temporal artery feedback, observed a progressive decrement in the number of migraine headaches per month occurred concomitantly with continued biofeedback training of cephalic vasomotor responses (i.e., vasoconstriction of the superficial temporal artery). Feuerstein and Adams'

1975 study was later confirmed by Friar and Beatty (1976) in which a group of 19 migraine sufferers significantly reduced the monthly incidence of major migraine headaches.

In all of these biofeedback treatment studies, the assumption has been made that there is a direct relationship between superficial temporal artery size and headache pain. The rationale behind these assumptions has been borrowed from earlier chemical studies previously mentioned (Graham & Wolff, 1938). Due to the fact that drugs work on many levels simultaneously, it is not specifically known what extent nonchemical vasoconstriction plays on pain reduction. Many authors suggest that chemical agents make the headache site more sensitive to pain (sterile inflammation) or change the vessels' elasticity (edema). Factors other than arterial stretching, therefore, likely contribute to the pain experience. Nonchemical biofeedback treatment studies have failed to establish a causal relationship between arterial size and headpain and only a decrease in headaches per month and medication usage have been reported (Feuerstein & Adams, 1975; Friar & Beatty, 1976). Furthermore, control procedures such as different electrode locations (Friar & Beatty, 1976) are essential in biofeedback studies to aid in determining treatment validity.

The purpose of the present study is to further discriminate the relationship between reported headpain in migraine sufferers and the feedback control of superficial temporal artery size. In an attempt to demonstrate such a

relationship, a method similar to those of earlier superficial temporal artery biofeedback studies will be used. Studies by Christie and Kotses (1973), Koppman et al. (1974), and Friar and Beatty (1976) suggest that migraine sufferers can rapidly learn dilative and constrictive control of superficial temporal artery diameter during headache free periods. In this experiment, subjects will be monitored to determine vasomotor control during non-headache periods and again during headache to discern whether they can maintain previously learned control and whether pain changes as a function of superficial temporal artery size. In the absence of evidence that cranial vasoconstriction by nonchemical techniques (e.g., biofeedback) can reduce pain, the following questions will be examined.

1. Can classic migraine headache sufferers learn bidirectional control of their superficial temporal artery pulse amplitude?

2. Can classic migraine headache sufferers maintain bidirectional control while experiencing headpain?

3. Does pain increase during migraine as superficial temporal artery pulse amplitude increases?

4. Does pain decrease during migraine as superficial temporal artery pulse amplitude decreases?

These questions, stated in the statistically null form, are:

1a. There is no significant difference between blood volume pulse amplitude during the resting baseline period and blood volume pulse amplitude during the dilative task period.

1b. There is no significant difference between blood volume pulse amplitude during the resting baseline period and blood volume pulse amplitude during the constriction task period.

2a. There is no significant difference between blood volume pulse amplitude during the resting baseline period and blood volume pulse amplitude during the dilative feedback period while subjects are experiencing migraine headache.

2b. There is no significant difference between blood volume pulse amplitude during the resting baseline period and blood volume pulse amplitude during the constrictive feedback period while subjects are experiencing migraine headache.

3. There is no significant increase in reported headpain with an increase of dilation time during headache.

4. There is no significant decrease in reported headpain with a decrease in dilation time during headache.

METHOD

In this section, a detailed description is given of the design of the study in four main parts. The first part deals with the subjects who participated in the study. The apparatus used for training and data collection is explained in the second part. In the third part, the experimental procedures used and the design are discussed. Part four concludes the section with a presentation of the statistical procedures used in the analysis of the data.

Subjects

Subjects were drawn from a population of 300 migraine headache sufferers. All subjects were first seen by a neurologist, diagnosed, and referred for biofeedback training. Nineteen of the twenty subjects in this experiment were female. This ratio of women to men was unplanned and likely occured due to the high incidence of females in the migraine population, (Pearce, 1971). The 20 subjects were chosen on the basis of their fulfilling five experimental criteria:

1. All subjects were interviewed to determine their willingness and motivation for completing the training program.

2. All subjects chosen has a minimum of three headaches per month prior to training, and no maximum limit was set.

3. All subjects had pain in the fronto-temporal region of the head and headache that occurred reliably on one side.

4. All subjects had a minimum of one-half hour warning of the onset of headache pain.

5. All subjects were willing to experience at least one migraine headache in the laboratory for the purpose of the experiment.

An attempt was made to select patients below the age of 50 due to possible arterio-sclerotic development with age (Dalessio, 1972). The ages of the subjects ranged from 20 to 53 years. Subjects were screened out of the experiment if the referring neurologist suspected a strong psychogenic component, large tension component, or arterio-sclerotic involvement as adjuncts to their migraine activity. Those subjects who reported any prior biofeedback experience were also dropped from the experiment.

A Melzack Pain Questionnaire and a history questionnaire were completed regarding their headache, medications previously taken, and an explanation of what they thought caused their headpain physiologically. Subjects were also asked to record any headache activity prior to training for a period of three weeks.

Four of the original 19 female subjects dropped out due to scheduling conflicts. In the final section of this experiment, (Pain Control), only eight of the sixteen subjects participated. This subject loss was due to unpredictability of each subjects headache.

Apparatus

Subjects received training in a comfortable, dimly lit room (2 m X 2-1/2 m). Temperature remained between 18-24° centigrade, and an attempt was made to make the subject as comfortable as possible. Subjects were seated upright in a large, comfortable, high-backed lounging chair. A Fanon 10 cm X 10 cm speaker used for auditory feedback was 60 cm to the left of the subject, and to the subject's right was a Fanon Classic-IN 400 Intercom which provided constant auditory access. Recording and programing equipment were located in an adjacent room.

Physiological records were obtained on an 8-channel Nihon-Khoden Model RM-85 polygraph, with each channel also displayed on a visual monitor. Four photoelectric plethysmograph sensors were attached to the subject. Two sensors were located on the head and were held in place with an elastic headband and adhesive tape. The subject's index fingers were placed inside two other plethysmograph sensors. Plethysmographic sensors were interfaced to a Nihon Khoden battery-operated transducer Model MPP-3S, and the gain was maintained at a constant level throughout the experiment. The signal was led into the main Nihon Khoden amplifiers and polygraph recordings were then obtained from these signals. Amplifiers were set for bandpass of 0.1 sec to 15 hz with gain adjustable per person. The amplified signal from the plethysmograph

sensor, of the site that was to receive feedback, was then fed into a Coulbourn Modular logic biofeedback instrument. A contour following integrator integrated the signal over a 2-sec period, and a bipolar comparator determined if threshold was reached. From the bipolar comparator, an OR-gate switching module was activated during the time tone was off. This signal was sent to a Mousseau dual channel elapsed time meter Model BFTC, and back to an amplifier on the Nihon Khoden to be recorded on the polygraph readout. From the meter, the amount of time the feedback tone was off could be recorded for each trial. For headache pain recording, a linear sliding potentiometer was connected to the Nihon Khoden and recorded on the polygraph readout. See Appendix 1 for acquisition of pain score data.

Procedure and Design

Part A (Training). Prior to the first session, subjects were oriented to procedure and equipment and read experimental instruction (see Appendix 2). All questions were answered as clearly and honestly as possible without revealing the theoretical relationship between artery size and pain. The instructions were read again if there were any procedural questions.

Subjects were divided into two groups. Treatment for both groups was identical. Each subject received a minimum of

13 training sessions lasting approximately one hour. Two sessions per week were required of all subjects. Upon arriving at the laboratory, subjects were seated, and four photoelectric plethysmograph sensors were placed on the subject, one on each index finger, one on the superficial temporal artery approximately 1.5 cm above the ear canal, directly in front of the ear, and on the same side a sensor was placed on the superorbital artery located on the forehead approximately 2.5 to 5.0 cm above the pupil eye level. The accuracy of the sensor implacements were checked by inspecting the visual monitor. Subjects were then asked to sit quietly while the experimenter adjusted the equipment. Each session was divided into two identical parts. During part one, the subjects concentrated on voluntarily altering the superficial temporal artery on the side of the head pain usually occurs. A baseline peirod (no tone) of 10 minutes was recorded, followed by dilating and constricting periods of 3 minutes each. Another 5-minute baseline was then recorded, followed again by constricting and dilating periods of 3 minutes each. The second part of the session was identical, except the subject was instructed to concentrate on dilating and constricting arteries in their index finger, homolateral to the aforementioned superficial temporal artery site. Dilation and constriction, along with head versus hand

periods were alteranted to minimize order effect. The finger location was used in this experiment to determine if there was any placebo effect, Hawthorn effect, or reduction in pain simply due to distraction.

The feedback threshold was defined as the point at which resting blood volume pulse amplitude is exceeded 50% of the time, during the last 3 minutes or the preceding baseline. Artery size or blood volume pulse amplitude cannot be measured directly. A change in the amount of light transmission is what was measured. This technique has been used as a method of inferring artery size (Koppman et al., 1974). Therefore, if dilation time scores are significantly above resting baseline, then it can be assumed that the artery is dilated and, conversely, if the dilation time scores are significantly below resting baseline, it can be assumed that the artery is constricted in relation to resting baseline. The amount of time spent in the dilated state was recorded rather than attempting to measure and sample light reflectance. This measurement was used because time was a more objective measure and time scores were mechanically recorded, therefore minimizing measurement error. The dependent variable for all control sessions was time above threshold. On the Coulbourn instrumentation, a switching device enabled the experimenter at position 1 to record the amount of time above threshold, which equalled dilation time, and at position 2, time below threshold was recorded. While

the device was switched to position 2 the total time minus the time below threshold equalled dilation time. Dilation time scores should not be confused with dilation and constriction instructions. The threshold level was determined by visually inspecting tone on-off recordings displayed on the polygraph readout. Feedback tone was heard when the desired task was not being accomplished. Tone pitch decreased as constriction occurred and increased as dilation occurred. Frequency modulation was used to aid subjects in discriminating and maximizing bidirectional control.

During the latter sessions (beginning with session 6), if the subject was able to keep the tone off for a period exceeding 10 seconds, the threshold was systematically changed a predetermined amount by the experimenter, such that it was more difficult to keep the tone off. This shaping was done to maximize control. The original threshold was maintained, however, as the point at which tone-off time per minute was recorded. Subjects were informed of this procedure. Also, beginning with session 6, subjects were asked to practice at home during the days they were not receiving training, and record the number of sessions and length of time they had practiced at home per week. Training session 13 dilation scores were recorded for each task (dilation or constriction) at both sites for each subject. Only those subjects showing superficial temporal artery dilative and constrictive control

significantly different from baseline proceeded to Part B. In Part B, eight subjects were involved, 7 female and 1 male.

Part B (Pain Control). Subjects were asked to come to the laboratory when they began experiencing prodromal signs and before headpain began. This was required so that subjects would be attempting dilative and constrictive control during the first stage of headache, prior to the onset of edema (Dalessio, 1972). The complete procedure for this session was previously explained and reviewed to each subject. Subjects were instructed not to injest any food or medication that may affect their headache. Sensors were attached in the standard manner and resting blood volume pulse amplitude measures were recorded for 10 minutes. The remainder of the session was divided into five 5-minute periods: dilation and constriction (hand), baseline, and dilation and constriction (head). The subjects' tasks were alternated, as earlier explained, to control ordering effect. Recordings of performance on the last 3 minutes of each 5-minute task period was used as dilation time and subjective pain report data. Subjective headpain was conveyed by moving a lever with a finger on the hand opposite the headache site (see Appendix 1). They were instructed to move the lever away from them if pain decreased, or toward them if pain increased. With this method, subjects could signify whether pain remained constant, increased, or decreased, and to what extent. If

vomiting occurred, the session was temporarily interrupted. Transportation for each subject who participated in this experiment was prearranged.

Statistical Procedures

By simply comparing the subjects' dilative control scores to their constrictive control scores, while concentrating on altering artery size in the head or finger, the reader would be unable to determine the influence that each task had on the results. Because dilative and constrictive control are of major importance in this study, they will be studied separately, and under stringent conditions. In the analysis of dilative and constrictive control at each location, comparisons were made between dilation time scores and baseline time scores for each task. With each session, a resting baseline was determined for each subject, due to individual pulse differences. The threshold level was then adjusted as previously explained (p. 32) and there was no significant variation in baseline threshold levels between subjects. Confidence intervals were determined for the control scores on each task, and it was then determined whether the baseline score was within or outside of the confidence interval at the .05 probability level. These analyses were completed after the eighth session, thirteenth session (control), and the headache session. One-tailed t test probability levels were

used due to the predicted directionality of dilation and constriction. Since only half of the subjects in this study were able to accurately anticipate a migraine headache and come to the laboratory for testing, a *t* test was run comparing the group experiencing headache in the laboratory to the group unable to be tested during headache. These analyses were completed for each task at each location, using a two-tailed *t* test.

The analysis completed on headache session pain data is comprised of two parts. The first part is a one-way analysis of variance for each task at each location. Relative pain change scores from baseline were used. Post hoc procedures were completed if significant results occurred. The second part is a three-way analysis of variance with repeated measures and simple main effects statistics calculated upon significant interaction. The three variables include: (1) location--one level being the head, another level being the finger; (2) instructions--one level being dilation instructions, another level being constriction instructions; and (3) sequence of observation--the last third, fourth, and fifth minute of each task during headache. A repeated measures design was used, før each subject received each instruction at each location. Two dependent measures were analyzed: time--dilation time for each task, and pain--relative pain change magnitude. Due to a lack of objective

pain scoring methodology in the field of migraine headache research, a subjective method of pain reporting was devised for this experiment. A graphic presentation displaying the relationship between dilation time scores and relative pain change magnitude was completed for each task at each location.

RESULTS

This section is divided into three main parts. Part one refers to the amount of time subjects spent practicing control. Part two presents the analyses and data relevant to hypotheses one and two. The analyses of data dealing with hypotheses three and four are displayed in the last part.

Task Control Analyses

For the sake of clarity, null hypotheses 1 and 2 will be repeated in this section, and the statistical results will be presented after each null hypothesis.

Presented in Table 1 are the amounts of time each subject spent in training and practice prior to the migraine headache session. Null hypothesis one, stated in two parts, reads as follows: there is no significant difference between blood volume pulse amplitude during the resting baseline period and (a) blood volume pulse amplitude during the dilative task period, or (b) blood volume pulse amplitude during the constriction task period while concentrating on the superficial temporal artery. Confidence intervals established at the .01 probability level yielded significant results for both the dilation task and the constriction task for head location. Both sections of null hypothesis one were rejected at session 8. Confidence intervals established at the .01

Table 1

Time Involved in Training and Practice Prior to the Headache Session

Subj.	Weeks in training	No. of hrs. of training prior to headache session	No. of home practice sessions	Average time for practice session (in minutes)	No. of hrs. of practice prior to headache session	Sum of training and practice (in hours)
1	6	13	18	15	4.5	17.5
2	7	14	16	22	5.9	19.9
3	6	14	23	14.5	5.6	19.6
4	6	16	21	31	10.9	26.9
5	8	17	19	20	6.3	23.2
6	7	14	18	24	7.2	21.2
7	5	16	19	17	5.4	21.4
8	8	17	32	26	13.9	30.9
$\bar{X}$	6.625	15.13	-	-	7.5	22.6

N=8

level also yielded significant results for the finger dilation and finger constriction task. Table 2 displays data for session 8.

Session 8 data was analyzed to determine progress of task control. Session 13 data was used (1) to determine more accurately subjects' control, and (2) to determine which subjects could proceed to Part B (headache session).

Confidence intervals were established for session 13 data for the head dilation task and head constriction task. Both yielded significant results at the .01 probability level. This adds support to the previous rejection of null hypothesis one. Finger dilation task and finger constriction task confidence intervals were established at the .01 probability level for session 13 and both yielded significant results. This data is displayed in Table 3.

By subtracting dilation time scores during the constriction task from dilation time scores during the dilation task, difference scores could be obtained for sessions 8 and 13. These difference scores were compared to determine if improvement occurred in bidirectional control at the head location and finger location. A one-way analysis of variance was completed comparing session 8 with session 13 at the finger and head locations. Both yielding significant results at the .05 and .01 probability levels, respectively. Newman-Keuls post hoc analysis revealed that bidirectional

Table 2

Group Dilation Time Score Confidence Intervals for Session 8

Location	Instruction	Mean no. of secs. dilated/3 min.	SD	Baseline no. of secs. dilated/3 min.	Confidence interval	Level of significance*
Head	Dilation	132.75	7.30	90	121.51-143.99	> .01
	Constriction	49.88	17.02	90	38.79-60.95	> .01
Finger	Dilation	132.06	24.37	90	116.32-147.90	> .01
	Constriction	64.94	19.62	90	52.20-77.68	> .01

N=16

*All significance levels were determined for a one-tailed test.

Table 3

Group Dilation Time Score Confidence Intervals for Session 13

Location	Instruction	Mean no. of secs. dilated/3 min.	SD	Baseline no. of secs. dilated/3 min.	Confidence interval	Level of significance*
Head	Dilation	160.94	14.25	90	151.68-170.19	>.01
	Constriction	31.25	19.79	90	26.30-36.19	>.01
Finger	Dilation	143.41	21.13	90	129.57-157.05	>.01
	Constriction	50.56	24.78	90	34.45-66.67	>.01

N=16

*All significance levels were determined for a one-tailed test.

control improved from session 8 to session 13. Figure 3 displays these differences.

Tests of significance for each task at each location were conducted comparing those subjects who participated in the headache pain session and those who did not. Student t test scores at the .05 probability level failed to show significance on all tasks: head dilation, $t = .566$; head constriction, $t = -.542$; finger dilation, $t = -1.29$; finger constriction, $t = -1.09$.

Null hypothesis two, stated in two parts, reads: There is no significant difference between blood volume pulse amplitude of the superficial temporal artery during the resting baseline period and the blood volume pulse amplitude (a) during the head dilation task period, and (b) during the head constriction task period while subjects are experiencing migraine headache. Each task at each location was analyzed separately. Confidence intervals were established at the .01 level for the head dilation task and the head constriction task with subjects showing control for each task. Both parts of null hypothesis two were rejected. Intervals established at the .01 level for finger constriction showed that subjects maintained significant control for that task. Finger dilation task control failed to show a significant difference from baseline. This data is displayed in Table 4.

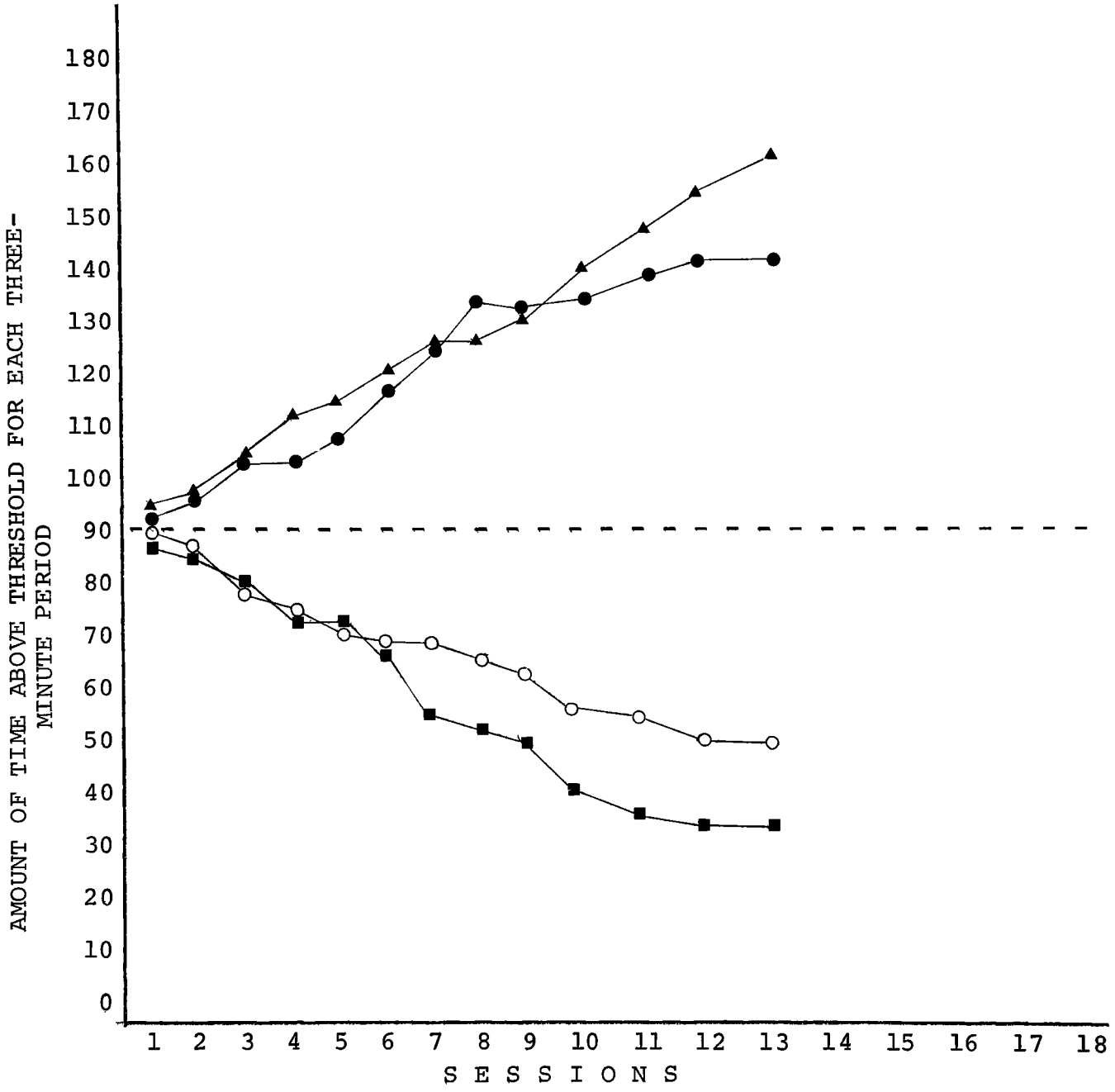


Figure 3. Total Mean Dilation Time Scores for All 16 Subjects from Session 1 (Baseline) to Session 13 (Control).

▲ Head dilation instructions
■ Head constriction instructions
● Finger dilation instructions
○ Finger constriction instructions

Table 4

Group Dilation Time Score Confidence Intervals for the Headache Session

Location	Instruction	Mean no. of secs. dilated/3 min.	SD	Baseline no. of secs. dilated/3 min.	Confidence interval	Level of significance*
Head	Dilation	172.50	12.95	90	159.28-185.72	.01
	Constriction	30.13	29.62	90	.22-60.48	.01
Finger	Dilation	123.13	58.15	90	84.28-191.98	ns
	Constriction	17.63	31.77	90	-14.94-50.19	.01

N=8

*All significance levels were determined for a one-tailed test.

Pain Differences During Headache Session

In this section, hypotheses 3 and 4 will be presented. For the sake of clarity, these hypotheses read as follows: (3) There is no significant increase in reported headpain with an increase of dilation time during headache. (4) There is no significant decrease in reported headpain with a decrease in dilation time during headache.

One subject's pain scores were highly inconsistent with the results obtained from all the other subjects. With her scores deleted, significance was found on a number of variables not previously different from baseline. This subject's scores, however, could not be overlooked, because (1) her scores varied systematically for location, and (2) with such a small sample (N = 8), it was not feasible to discard one subject's set of scores and possibly further bias the characteristics of the population. The analysis of pain data with one subject scores removed will be displayed in Appendix 6. The reader is advised to examine these analyses with extreme caution for the analyses may be greatly biased by dropping this subject.

Analysis of data with N = 8. A one-way analysis of variance was completed comparing both tasks at both locations. Relative pain change scores for the head dilation task were significantly different from baseline at the .01 probability level. Pain significantly increased during the head dilation task. All other analyses failed to show

significant differences at the .05 level. A Newman-Keuls post hoc test revealed that during the head dilation task, pain increased significantly from baseline at the .01 probability level. Table 5 displays analysis of variance data.

A three-way analysis of variance using pain scores revealed a significant interaction. This data is displayed in Table 6. Simple main effects were employed to determine significant differences between means. This analysis revealed a significant difference in pain (.05 level, $\underline{F}$ = 6.76) between head dilation and constriction tasks. There also existed a significant difference in pain (.05 level, $\underline{F}$ = 7.038) between the head dilation task and the finger dilation task.

A graphic representation of the relationship between dilation time and relative pain change magnitude is displayed in Figures 4 and 5.

Table 5

One-way Analyses of Variance for Each Task Using Scores of the Relative Change in Magnitude of Headache Pain
(N=8)

Location	Task	Baseline mean	Baseline SD	Task mean	Task SD	Mean squared error	df Nun	df Den	F	p
	Dilation			646.08	134.47	11269.48	1	46	33.722	.01
Head		468.13	66.75							
	Constriction			457.29	180.48	18515.47	1	46	.076	ns
	Dilation			691.208	414.95	95620.17	1	46	1.15	ns
Finger		595.46	138.06							
	Constriction			720.29	433.13	103330.10	1	46	1.18	ns

Table 6

Three-way Analyses of Variance with Repeated Measures Using Relative Pain Change Magnitude Scores (N=8)*

Source of Variation	SS	df	MS	F	p
Location(A)	712315.1	7	712315.1	1.0227	ns
Instruction(B)	62781.51	7	62781.51	7.705	.05
Sequence of observation(C)	977.083	14	488.54	.372	ns
A X B	307700.26	7	307700.26	6.696	.05
Error			45952.05		

*Three observations were recorded for each subject.

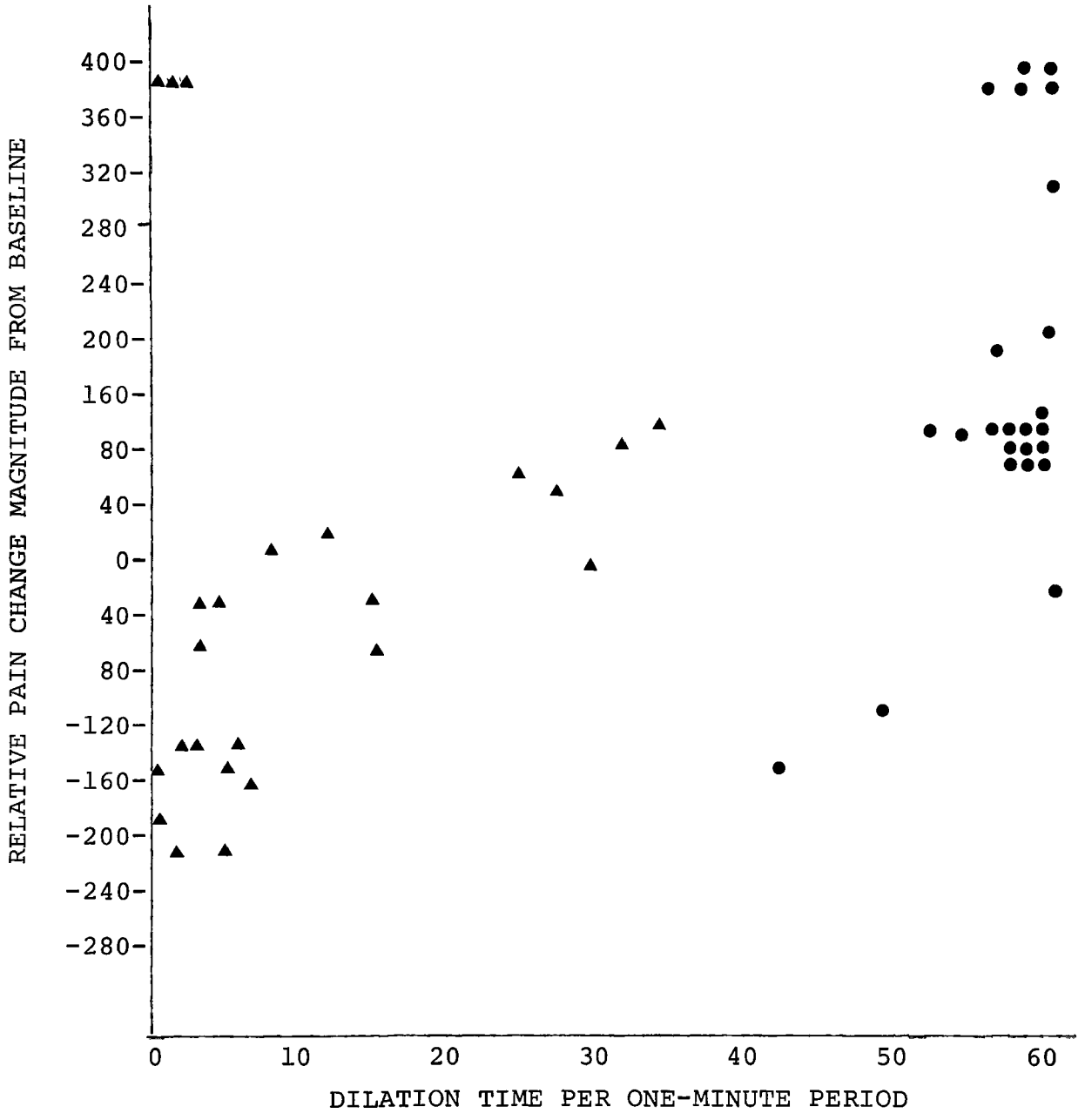


Figure 4. Relative Pain Change Magnitude Scores versus Dilation Time Scores for Dilation and Constriction Tasks at the Head Location during Headache (N=8).

Head dilation ●
Head constriction ▲

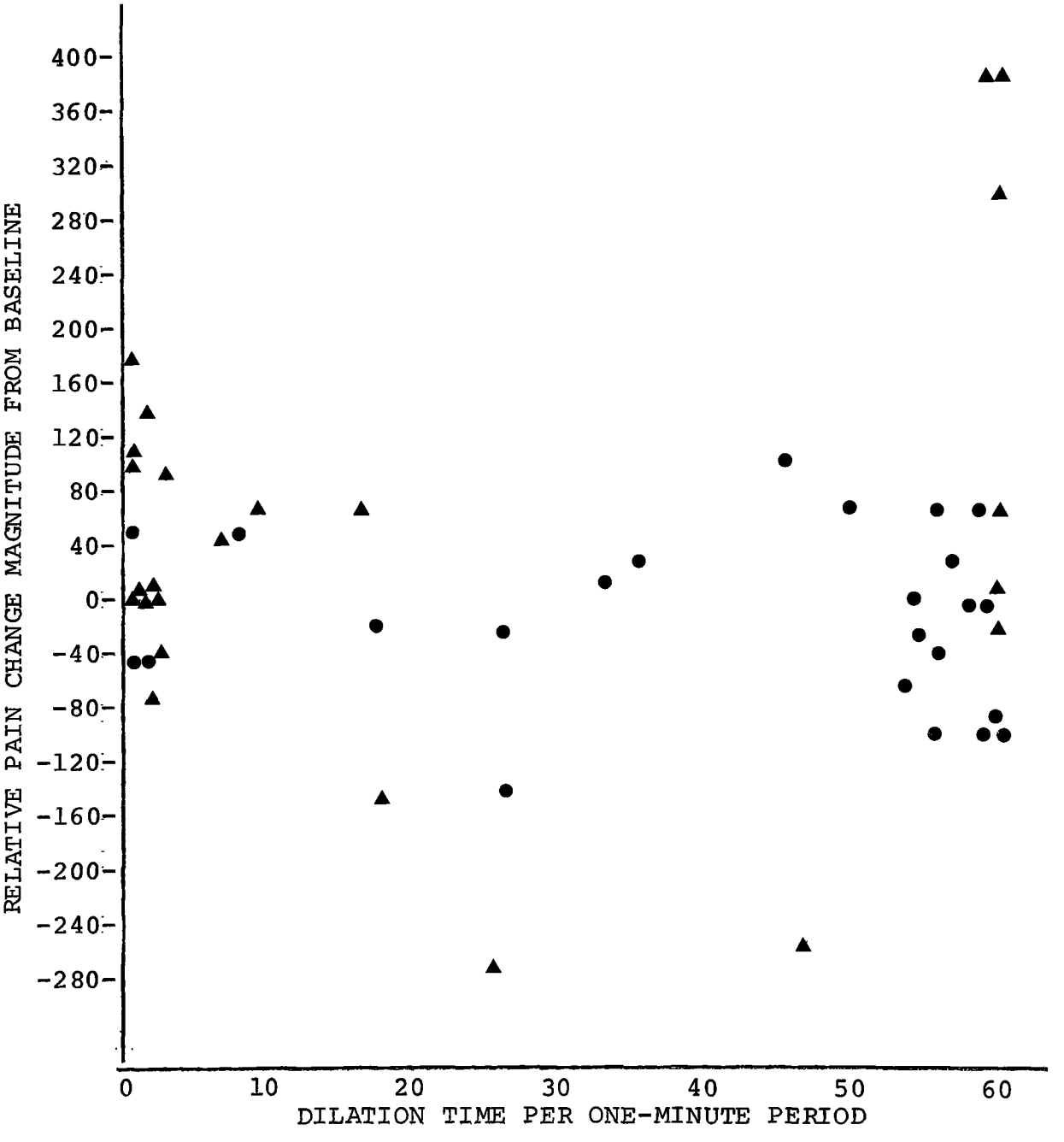


Figure 5. Relative Pain Change Magnitude Scores versus Dilation Time Scores for Dilation and Constriction Tasks at the Finger Location during Headache (N=8).

Finger dilation ●
Finger constriction ▲

Newman-Keuls post hoc procedure revealed that pain during the head dilation task was significantly greater than baseline and that pain during the constriction task was significantly less than baseline at the .01 probability level.

Using a three-way analysis of variance with repeated measures, significant interactions were observed for pain during different tasks (see Table 8).

Simple main effects were employed to determine significant differences between means. This analysis revealed a significant difference in pain (.05 level, $\underline{F}$ = 7.62) between dilation and constriction tasks when subjects concentrated on the head location. There also existed a significant difference in pain (.05 level, $\underline{F}$ = 8.02) between the head dilation task and the finger dilation task.

A graphic representation of the relationship between dilation time and relative pain change magnitude is displayed in Figures 6 and 7.

DISCUSSION

In this section, a discussion of the experimental results is presented. The limitations of the conclusions of this study are pointed out and the implications of the results in relation to theory and practice are mentioned.

A serious attempt was made to select appropriate and motivated subjects for this experiment. Motivation was reflected in subjects' willingness and reliability to make each appointment. More objectively, willingness was also judged by the amount of training subjects completed and the amount of home practice they reported.

With this highly selected group of classic migraine headache sufferers, it was shown that within eight sessions of auditory feedback, subjects could significantly affect their superficial temporal artery blood volume pulse amplitude bidirectionally. Though it is not known to what degree the actual size of the artery is affected, light transmission was greatly increased or decreased by using this operant technique. Previous research has reported that size or amplitude can be inferred from differences in light transmission (Koppman, 1974).

It was demonstrated with this study that improvement continued and bidirectional control increased from session 8 to session 13. It it hypothesized that home practice, systematic shaping, and modulated tone cues

accounted for this continued improvement. Bidirectional change from baseline was used as the criterion for control in this experiment and various analyses showed that subjects could perform both tasks (dilation and constriction) equally well. Both parts of null hypothesis one were rejected at session 8 and again at session 13. During the early stages of a migraine attack (within the first hour of headpain), subjects could maintain bidirectional control, as compared to baseline and null hypothesis two was rejected. During the headache session it appeared that subjects could dilate their superficial temporal arteries much more easily than constrict them. The standard deviation for the head dilation task is much smaller than for the head constriction task during the headache session. This indication, along with the difference in estimated confidence intervals for these tasks, suggest a qualitative difference in dilative versus constrictive ability during headache (Table 4). Theoretically, the size of the artery increases during migraine headache, and attempting to dilate likely contributed to the ongoing physiological process. Interestingly, it was observed that following the head dilation task, pulse amplitude reduced, although this was not systematically analyzed. Dilation time scores for the head constriction task were more variable than dilative task scores, and this was reflected in the standard

deviations for these tasks. Within the same framework, possibly this can be explained as: (1) opposing the physiological process of the migraine attack, or (2) difficulty in concentrating during headache.

The purpose of this experiment disabled the experimenter from inducing headache chemically or by other means; therefore, it was extremely difficult to coordinate the time subjects had headaches to the availability of the experimenter. Due to the importance of recording data within the first hour of headache, subjects, on many occasions, could not contact the experimenter in time to participate. Another complicating factor involved the continuous training of subjects: (1) if they failed to have a headache; or (2) if they were unable to come to the laboratory. For these reasons, training continued for four weeks after the control session (session 13). Most subjects qualified for the headache session, but for various reasons only eight were able to participate. With such a small number of subjects, extreme caution must be taken in the interpretation of headache results.

Within Subject Experimental Control

The within subject controls (finger tasks) were used to determine whether pain differences were due to (1) distraction or experimental task, and (2) whether subjects were trying to please the experimenter with their responses. Two subjects

were aware of the physiological dynamics of migraine headpain, while the other six had no knowledge of vascular theory. Subjects learned tasks for the finger and for the head equally well. Significant improvement was seen from session 8 to session 13 for both locations. This indicates that subjects paid equal attention to all four tasks. Subjects reported pain changes for each task whether it be at the finger location or head location, implying they did not think of treatment in terms of location. It can be assumed that tasks on the finger were thought of as legitimate treatment tasks by the subjects. Results, therefore, can be interpreted without over-concern with subject expectancy.

The within subject control method was used because migraine is characteristically different for each person and since the headaches are so unpredictable there was a concern that if more than one group was used, the sizes of the groups and the amount of training for each group would be different.

Discussion of the Analyses of Headpain

Five of the eight subjects reported a similar, systematic increase in pain during the dilation task with a decrease in pain during the constriction task (see pain data, Appendix 3). The remaining subjects showed scores that were much more variable, and subject 8 had pain scores consistently

high for the head tasks and consistently low for the finger tasks. There were systematic deviations among subjects who were opposite to that of group data but not sufficiently large to affect results. Subject 8's data, however, greatly affected the outcome of the significance tests. All subjects, with the exception of subject 7, reported an increase in pain during the dilation task. With all eight subjects' pain scores included, null hypothesis three could be rejected, but null hypothesis four could not.

It can be said that, for most subjects in this study, pain decreased during head constriction and increased during head dilation.

Discussion of Data in Relation to Theory

Pain during migraine headache has been shown to be positively related to the size of external carotid arteries (Graham & Wolff, 1938).

This study presented evidence that subjects can maintain significant dilative and constrictive control of superficial temporal artery size during headache. While subjects are concentrating on dilating the superficial temporal artery during headache, pain significantly increases. Conversely, with one subject data deleted analyses showed that while subjects concentrated on constricting their superficial

temporal artery during headache, pain significantly decreases. Considerable caution must be taken in the interpretation of the constriction task with one subjects data deleted, however it is suggested that with a larger sample bi-directional pain control can be demonstrated. This evidence, though not conclusive, is consistent with vascular theory and the findings of Graham and Wolff (1938).

The application of this operant conditioning technique to the therapeutic reduction of headache has been shown in earlier studies (Feuerstein & Adams, 1975; Friar & Beatty, 1976). The rationale for these two studies evolved from the earlier drug treatment studies previously mentioned. The current experiment has added support to the rationale and has provided evidence that subjects can affect a change in pain while experiencing migraine headache. Experimental data from this study also clearly indicated that subjects can maintain dilative and constrictive artery control during headache. Attempts at constricting the artery for those subjects trained with this technique should continue, even though headpain has begun.

Suggestions for Further Research

Due to the difficulty with interpreting the results of such a small sample, replication research is essential.

The characteristically well defined prodrome periods of the classic migraine headache sufferer aid in the prediction of an upcoming headache, and the feedback tone modulation was reported to be useful in the learning of bidirectional control. These two observations may aid researchers in attempting to replicate this study.

More accurate or direct methods of measuring artery size must be found before clear statements can be made of the effectiveness of this biofeedback technique. The point at which artery control is begun in the headache experience may be a significant factor in pain control and could be investigated. With greater awareness of this technique's effect on artery size, the efficacy of constriction drugs may be more accurately assessed. The combined effect of drugs and biofeedback could be investigated. And, finally, the subject's ability to produce changes in the artery size at various stages of headache could add information about the occurrence of edema in the artery during headache.

SUMMARY AND CONCLUSIONS

The purpose of this study was to determine (1) whether classic migraine headache sufferers could learn bidirectional control of their supervicial temporal artery; (2) whether these subjects could maintain bidirectional control during headache; and (3) what effect the dilative and constrictive tasks have on headpain.

Statistical analyses showed that subjects could learn bidirectional control and maintain control during headache. Subjects could significantly increase headache pain but could not significantly decrease pain during headache. Some evidence, however, suggests the possibility that with a larger group of subjects, a decrease in headpain during headache can also be demonstrated. A graphic representation of the relationship between the amount of time in the dilated state and relative pain change magnitude was plotted. It is concluded that a positive relationship exists between the superficial temporal artery amplitude and the magnitude of subjective pain reported.

REFERENCES

Alvarez, W. C. The migrainous personality and constitution: The essential features of the disease; a study of 500 cases. American Journal of Medical Science, 1974, 213, 2.

Benson, H., Klemchuk, H. P., & Graham, J. R. The usefulness of the relaxation response in the therapy of headache. Headache, 1974, 14(1), 49-52.

Bihldorff, J. P., King, S. H., Parnes, L. R. Psychological factors in headache. Headache, 1971, 11(3), 117.

Blanchard, E. B., & Young, L. D. Biofeedback and self control. Chicago: Aldine, 1974.

Brody, J. E. America's $400,000,000 headache. Science Digest, August 1968, 17.

Ceres, F. Aviation medicine in the United States Navy. War Medicine, 1941, 1, 43.

Christie, D. J., & Kotses, H. Bi-directional operant conditioning of the cephalic vasomotor response. Journal of Psychosomatic Research, 1973, 17, 167.

Classification of Headache. Journal of the American Medical Association, 1962, 179, 717.

Dalessio, D. J. Wolff's headache and other head pain (3rd ed.). New York: Oxford Press, 1972.

Dalsgaard-Nielsen, T. Migraine attack as a socio-medical problem. Headache, 1971, 11, 47-50.

Dalsgaard-Nielsen, T. The nature of migraine. Headache, 1974, 14(1), 13-25.

Desrosier, J. J. Headaches related to contraceptive therapy and their control. Headache, 1973, 14(3), 117-124.

Diamond, S., Baltes, B. J., & Levine, H. W. A review of the pharmacology of drugs used in the therapy of migraine. Headache, 1972, 12(2), 37-43.

Dow, D. J., & Whitty, C. W. Electroencephalographic changes in migraine. Lancet, 1947, 2, 52. In O. W. Sacks. Migraine: The evolution of a common disorder. London: Faber & Faber, 1970.

Edmeads, J. Management of the acute attack of migraine. Headache, 1973, 13(3), 91-95.

Engel, G. L., Ferris, E. B., & Romano, J. Focal electro-encephalographic changes during the scotomas of migraine. American Journal of Medical Science, 1945, 209, 650. In O. W. Sacks. Migraine: The evolution of a common disorder. London: Faber & Faber, 1970.

Feuerstein, M., & Adams, H. E. Cephalic vasomotor feedback for the treatment of migraine headache: An alternative therapeutic approach. Paper presented at the Ninth Annual Meeting of the Association for Advancement of Behavior Therapy. San Francisco, December 1975.

Fisher, A. L. A psychobiological reconsideration of pain. Headache, 1970, 10(2), 74-86.

Fisher, K. H. The problem of pain from psychiatrist's viewpoint. Psychosomatics, 1968, 9, 319-325.

Fitz-Hugh, T. Precordial migraine: An important form of angina innocens. New International Clinics, 1940, 1(Ser. 3), 143.

Friar, L. R., & Beatty, J. Migraine: Management by trained control of vasoconstriction. Journal of Consulting and Clinical Psychology, 1976, 44(1), 46-53.

Friedman, A. P., Von Storch, J. C., & Houston, M. H. Migraine and tension headaches: A clinical study of 2,000 cases. Neurology, 1954, 4, 773-788.

Friedman, A. P. The (infinite) variety of migraine: Sandoz foundation lecture. Background to Migraine, 1969, 3, 165-180.

Friedman, A. P. Reflections on the treatment of headache. Headache, 1972, 11(4), 148-155.

Friedman, A. P. Migraine headache. Clinical Neurology, 1975, 2, 6.

Friedman, A. P., & Merritt, J. B. 1969 study cited in A. P. Friedman. Reflections on the treatment of headache. Headache, 1972, 11(4), 153.

Gentry, D. L. Directive therapy techniques in the treatment of migraine headache: A case study. Psychotherapy: Theory Research and Practice, 1973, 10, 303-311.

Gibbs, E. L., & Gibbs, F. A. Electroencephalographic evidence of thalamic and hypothalamic epilepsy. Neurology, 1951, 1, 136. In O. W. Sacks. Migraine: The evolution of a common disorder. London: Faber & Faber, 1970.

Graham, J. R., & Wolff, H. G. Mechanism of migraine headache and action of ergotamine tartrate. Archives of Neurology and Psychiatry, 1938, 39, 737.

Greenleaf, E. Defining hypnosis during hypnotherapy. The International Journal of Clinical and Experimental Hypnosis, 1974, 22, 120-130.

Harrison, R. H. Psychological testing in headache: A review. Headache, 1975, 13(4), 177-185.

Hertzman, A. B., & Roth, L. W. The absence of vasoconstrictor reflexes in the forehead circulation: Effects of cold. American Journal of Physiology, 1942, 136, 692.

Horton, B. T. Histamine cephalgia: Differential diagnosis and treatment. Proceedings of the Mayo Clinic, 1956, 31, 325. In O. W. Sacks. Migraine: The evolution of a common disorder. London: Faber & Faber, 1970.

Horton, B. T. Management of vascular headache. Angiology, 1959, 10, 43-56.

Howler, M., DeMunnik, D., & Boerlien, A. H. The effects of mild electrostimulation on migraine and tension headache. International Medical Health Research Newsletter, 1973, 15, 11-13.

Kashiwagi, T. Headache and psychiatric disorders. Headache, 1972, 12, 659-663.

Koppman, J. W., McDonald, R. D., & Kunzel, M. G. Voluntary regulation of temporal artery diameter by migraine patients. Headache, 1974, 14(3), 133-137.

Korn, R. R. Vasomotor conditioning and the vascular headache: Toward a therapeutic synthesis (preliminary report). Intercollegiate Psychology Association Journal, 1949, 1, 51-67.

Kundle, E. C. Acetylcholine in the mechanism of headache of the migraine type. Archives of Neurology and Psychiatry, 1959, 81, 135. In O. W. Sacks. Migraine: The evolution of a common disorder. London: Faber & Faber, 1970.

Lance, W. J. The mechanism and management of headache (2nd ed.). New York: Butterworth, 1973.

Lance, W. J., Anthony, M., & Hinterberger, H. The control of cranial arteries by humoral mechanisms and its relation to the migraine syndrome. Headache, 1967, 7, 93-100.

Lashley, K. S. Patterns of cerebral integraiton indicated by scotomas of migraine. Archives of Neurology and Psychiatry, 1941, 46, 331. In O. W. Sacks. Migraine: The evolution of a common disorder. London: Faber & Faber, 1970.

Melzack, R., & Scott, T. H. The effects of early experience on the response to pain. Journal of Comparative and Physiological Psychology, 1957, 50, 155-161.

Melzack, R., & Torgerson, W. S. On the language of pain. Anesthesiology, 1971, 34(1), 50-59.

Merskey, H., & Spear, F. G. The concept of pain. Journal of Psychosomatic Research, 1967, 11, 59-67.

Merskey, H. On the development of pain. Headache, 1970, 10(3), 116-123.

Migraine Foundation. Migraine headache literature sent in correspondence. Migraine Foundation, 390 Brunswick Avenue, Toronto, Ontario, 1976.

Miller, N. E. Learning of visceral and glandular responses. Science, 1969, 163, 434-445.

Milner, P. M. Note on a possible correspondence between the scotomas of migraine and the spreading depression of Lẽao. Electroencephalography and Clinical Neurophysiology, 1958, 10, 705. In O. W. Sacks. Migraine: The evolution of a common disorder. London: Faber & Faber, 1970.

Mitchell, K. R., & Mitchell, D. M. Migraine: An exploratory treatment application of programmed behavior therapy techniques. Journal of Psychosomatic Research, 1971, 15, 137-157.

Morgenstern, F. S. The effects of sensory input and concentration on post-amputation phantom limb pain. Journal of Neurology, Neurosurgery and Psychiatry, 1964, 27, 58-65.

Paulson, O. B., & Skinhoj, E. Regional blood flow in internal carotid. British Medical Journal, 1969, 3, 569. In D. J. Dalessio. Headache and other head pain (3rd ed.). New York: Oxford Press, 1972.

Pearce, J. General review: Some aetiological factors in migraine. Backgrounder to Migraine, 1971, 4, 1-7.

Ray, B. S., & Wolff, H. G. Experimental studies on headache; Pain sensitive structures of the head and their significance in headache. Archives of Surgery, 1940, 41, 813.

Rowsell, A. R., Neylan, C., & Wilkinson, M. Ergotamine induced headaches in migrainous patients. Headache. 1973, 13(2), 65-67.

Sacks, O. W. Migraine: The evolution of a common disorder. London: Faber & Faber, 1970.

Sargent, J. D., Green, E. E., & Walters, E. D. The use of autogenic feedback training in a pilot study of migraine and tension headaches. Headache, 1972, 12(3), 120-124.

Schultz, J., & Luthe, W. Autogenic training; A psychophysiological approach in psychotherapy. New York: Grune & Stratton, 1959.

Schumacher, G. A., & Wolff, H. G. Experimental studies of headache: Contrast of histamine headache with headache of migraine and that associated with hypertension. Archives of Neurology and Psychiatry, 1941, 45, 199.

Selby, G., & Lance, J. W. Observations on 500 cases of migraine and allied vascular headache. Journal of Neurology Neurosurgery and Psychiatry, 1960, 23, 23-32.

Sicuteri, F. Prophylactic and therapeutic properties of methyl lysergic acid butanolamide in migraine. International Archives of Allergy, 1959, 15, 300.

Simmel, M. L. Phantom experiences following amputation in childhood. Journal of Neurology Neurosurgery and Psychiatry, 1962, 25, 69-78.

Strauss, H., & Selinsky, H. EEG findings in patients with migrainous syndrome. Trans-American Neurological Association, 1941, 67, 205. In O. W. Sacks. Migraine: The evolution of a common disorder. London: Faber & Faber, 1970.

Trowill, J. Instrumental conditioning of the heart rate in the curasized rat. Journal of Comparative and Physiological Psychology, 1967, 63, 7-11.

Waters, W. E. Epidemiological aspects of migraine. Backgrounder to Migraine, 1971, 4, 36-41.

Whitehouse, P., Pappas, J. A., Escala, P. H., & Livingston, S. Electroencephalographic changes in children with migraine. New England Journal of Medicine, 1967, 276, 23. In O. W. Sacks. Migraine: The evolution of a common disorder. London: Faber & Faber, 1970.

APPENDIX 1

METHOD OF ACQUIRING PAIN SCORE DATA

APPENDIX 1

METHOD OF ACQUIRING PAIN SCORE DATA

A box which had a sliding lever with a total range of approximately 13 mm was used by the subject to report headpain. The middle point on the box was clearly identifiable to the subject, for there were placed two large screws on either side of the lever at this point. Instructions during the headache session for recording pain were identical for each subject, and were as follows:

> You are presently experiencing headpain, and your current headpain will be represented by the midpoint on the box. With any decrease in headpain, slide the lever away from you corresponding to the degree in which pain has been alleviated. The endpoint will represent no headache. With any increase in headpain, move the lever toward you corresponding to the degree in which pain has increased. The endpoint in this direction corresponds to the most severe headpain you have ever experienced. Remember, the midpoint represents the degree of headpain you are now experiencing. When I leave the room, you may begin reporting any change in pain or simply leave the lever at midpoint if the pain remains at its current intensity.

This procedure was explained to each subject during training and again just prior to the headache session. All subjects were also asked to try the device in order to become familiar with the range that the lever could travel and discover any difficulties they might have; this was also done during training sessions. The signal from the box was lead into an amplifier and transposed onto polygraph paper.

No pain equalled zero on the polygraph readout. There were eight equal intervals, equal intervals on the paper itself, with a score of 80 being maximum pain and 45 being midpain. Each minute was divided into 33 equal intervals and a score was recorded for each interval. These scores were summed and a total score per minute was recorded. The average score for the third, fourth, and fifth minutes of each task at each location was determined. The average baseline score for the last three minutes of the immediately preceding baseline period was then subtracted from the last third, fourth, and fifth minutes of each task. By using this procedure, the relative change in pain magnitude could be determined, with the preceding baseline pain level being taken into consideration.

APPENDIX 2

EXPERIMENTAL INSTRUCTIONS

APPENDIX 2

EXPERIMENTAL INSTRUCTIONS

Biofeedback is a method of monitoring body functions and amplifying those functions so that the individual can become aware of them. Through this method, individuals can learn to control responses, previously imperceivable.

There have been a number of studies regarding the control of migraine headache using biofeedback. This particular method is very new, but very promising. In a recent study, researchers have explained the success of training upon increasing artery muscle tone. Generally, success in treatments of this type is contingent upon motivation and hard work on the part of the individual. Your role in this treatment is to dilate your temporal artery (make it larger in circumference), and to constrict your temporal artery (make it smaller in circumference). This will, in effect, exercise the artery and therefore increase muscle tone. In other prominent studies, blood flow was shown to be an important factor in migraine headache, therefore, you will also learn to increase and decrease artery size or blood flow to your index finger.

Learning to control your artery size or blood flow is entirely individual. You must use your own method to learn control. A helpful hint in dilating your artery is to visualize the artery expanding or visualize a small tunnel getting bigger. A helpful hint in constricting your artery is to visualize the artery getting smaller. Your dilating and constricting will be aided by a tone. When you no longer hear the tone, you will be achieving the correct response, whether you are dilating or constricting. When the tone is on, it will change in pitch corresponding to the size of your artery. If your artery grows in size, the pitch of the tone will become higher; if your artery shrinks in size, the pitch of the tone will become lower. Remember, you want to keep the tone off!

Some individuals can achieve control within a few sessions, others will take much longer. It is important that you progress at your own rate. It is also important that after sensors have been attached and you are seated comfortably, to refrain from moving your head or fingers extensively. During rest periods, you can stretch or move around. It is important to remember that your task is to exercise your arteries at both sites and, therefore, each part of your task is equally essential.

APPENDIX 3

HEADACHE SESSION, DILATION TIME AND RELATIVE PAIN CHANGE MAGNITUDE SCORES FOR THE LAST THIRD, FOURTH AND FIFTH MINUTE OF EACH TASK

HEAD

Last Three Minutes of Each Task

	Task: Dilation		Task: Constriction	
	Dilation Time	Relative	Dilation Time	Relative
Subj.	Time above threshold/min.	pain change magnitude	Time above threshold/min.	pain change magnitude
	60	394	25	74
1	60	394	32	84
	60	314	36	124
	60	77	13	27
2	60	77	29	47
	60	77	8	17
	60	217	0	-153
3	60	117	4	-153
	60	107	7	-163
	60	110	0	-210
4	60	110	5	-220
	60	110	1	-220
	60	-22	2	-142
5	60	108	6	-142
	57	188	3	-142
	58	105	16	-35
6	54	105	4	-35
	57	105	3	-35
	41	-150	3	-60
7	49	-110	30	0
	52	100	17	-75
	59	385	0	385
8	58	385	0	385
	56	385	2	385

FINGER

Last Three Minutes of Each Task

	Task: Dilation		Task: Constriction	
	Dilation Time	Relative	Dilation Time	Relative
Subj.	Time above threshold/min.	pain change magnitude	Time above threshold/min.	pain change magnitude
	46	114	60	304
1	8	54	60	384
	0	54	60	384
	53	-60	7	50
2	56	-40	2	-40
	56	-100	2	-70
	54	-33	60	17
3	18	-33	60	77
	28	-33	60	-33
	33	15	10	70
4	57	30	2	140
	37	30	0	180
	60	-110	1	110
5	60	-110	0	100
	60	-95	2	0
	59	70	18	70
6	50	70	3	90
	57	70	0	0
	27	-140	19	-150
7	0	-45	26	-270
	0	-45	49	-260
	58	0	0	0
8	59	0	0	0
	54	0	0	0

APPENDIX 4

DILATION TIME SCORES FOR EACH TASK AT EACH LOCATION FOR ALL SESSIONS

HEAD DILATION TIME SCORES

Average Number of Seconds above Threshold/3 Mins.

Subj.	Session 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
Pain Session Participants																		
1	86	90	97	99	102	107	106	121	137	154	159	140	156	180				
2	97	95	104	104	114	129	135	134	109	137	141	159	157	164	179			
3	93	114	120	143	148	160	157	171	174	179	175	173	170	176	180			
4	96	97	98	98	106	124	129	133	140	154	166	169	174	175	172	177	180	
5	87	96	109	113	120	123	127	130	125	131	137	138	139	152	160	162	170	177
6	90	101	112	118	109	122	125	140	134	145	148	161	175	134	169			
7	87	89	91	97	100	99	104	119	97	140	141	165	177	179	169	170	142	
8	95	97	98	98	97	104	114	125	120	92	138	158	156	162	114	153	160	173
Non-Pain Session Participants																		
9	100	105	124	125	128	139	140	138	136	134	154	173	171	177	160			
10	98	104	112	127	134	136	147	153	154	153	151	158	169	172	170	165	169	
11	98	99	97	106	116	117	145	149	146	144	143	156	157	155	159	150		
12	95	97	104	113	114	114	121	146	155	159	159	164	172	167	165			
13	80	90	108	125	130	126	127	126	120	160	172	164	172	150	163			
14	100	104	110	106	104	108	120	128	124	121	126	132	132	137	135	130		
15	86	94	103	109	118	119	117	114	102	128	157	155	160	158	170	171	174	
16	90	94	90	93	96	92	89	97	107	114	123	126	138	129	137			

HEAD CONSTRICTION, DILATION TIME SCORES

Average Number of Seconds above Threshold/3 Mins.

Subj.	Session 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
Pain Session Participants																		
1	93	82	70	69	63	63	46	45	41	40	30	42	37	87				
2	88	85	84	81	79	66	53	46	47	32	49	41	45	50	50			
3	93	84	75	72	54	34	12	19	25	18	16	11	6	14	11			
4	91	85	73	69	70	68	46	37	41	41	38	49	60	44	23	19	6	
5	96	93	90	80	73	65	57	44	39	40	43	46	48	30	26	21	25	12
6	85	72	63	59	46	44	42	52	50	14	20	22	11	2	23			
7	89	86	81	82	79	68	57	44	46	32	14	11	10	6	30	11	50	
8	70	76	95	100	95	93	90	82	75	69	60	42	61	56	50	60	42	2
Non-Pain Session Participants																		
9	87	85	80	76	76	72	66	57	53	33	20	16	12	18	20			
10	84	77	68	59	54	50	42	40	39	40	42	35	37	38	40	41	36	
11	76	73	70	70	69	61	59	37	40	41	13	7	9	15	24	21		
12	90	86	84	81	86	64	46	41	39	43	44	40	48	47	50			
13	98	96	97	96	97	83	64	70	78	73	50	53	22	16	10			
14	89	86	86	84	82	79	75	67	60	51	42	34	28	27	31	29		
15	74	62	55	43	31	28	33	38	29	18	11	11	12	13	21	18	12	
16	100	95	87	81	78	73	75	79	74	61	55	51	54	50	60			

FINGER DILATION TIME SCORES

Average Number of Seconds above Threshold/3 Mins.

Subj.	Session 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
Pain Session Participants																		
1	96	104	111	112	120	127	128	133	131	133	142	139	135	54				
2	89	87	86	86	95	109	119	123	120	119	116	115	145	124	165			
3	98	104	115	116	133	136	151	176	164	155	155	161	150	141	100			
4	94	101	102	105	112	117	116	118	120	129	135	139	141	138	145	143	122	
5	95	96	98	97	101	114	124	126	129	131	129	124	91	119	124	128	140	180
6	81	61	123	130	144	152	155	159	140	145	159	168	161	169	166			
7	85	90	94	97	107	121	142	156	167	164	157	152	145	134	129	132	27	
8	90	95	97	102	86	110	113	117	74	114	125	133	125	137	124	138	143	171
Non-Pain Session Participants																		
9	91	92	97	105	122	128	129	125	130	135	139	135	133	130	142			
10	75	94	99	98	100	128	146	147	143	137	134	120	177	173	154			
11	101	105	106	103	104	111	139	144	150	162	167	163	166	170	160	165		
12	84	93	107	118	140	165	174	172	163	150	151	163	160	158	157			
13	95	96	97	100	98	94	81	104	94	115	130	145	150	161	114			
14	90	92	94	95	97	94	98	100	106	113	124	134	132	140	145	131		
15	85	104	91	89	85	91	101	106	110	119	140	149	163	172	175	154	150	
16	111	110	104	102	97	98	102	107	114	119	124	123	119	120	122			

FINGER CONSTRICTION, DILATION TIME SCORES

Average Number of Seconds above Threshold/3 Mins.

Subj.	Session 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
Pain Session Participants																		
1	71	74	75	72	69	12	79	73	66	63	64	55	60	0				
2	83	79	77	60	46	51	53	55	30	22	24	22	65	20	11			
3	96	93	92	76	65	66	43	46	47	56	62	53	41	41	0			
4	87	84	80	81	84	90	94	89	83	74	70	71	80	20	72	70	12	
5	93	93	90	88	86	86	84	80	70	65	58	42	50	36	31	26	8	3
6	102	103	47	46	12	35	40	42	29	23	18	41	53	3	21			
7	83	81	77	79	80	77	52	32	25	21	28	20	20	27	30	22	94	
8	97	94	89	83	78	70	59	45	88	47	46	37	8	2	0	14	18	0
Non-Pain Session Participants																		
9	85	83	79	78	77	76	68	65	66	64	60	49	28	33	60			
10	95	80	65	66	57	60	71	86	96	93	90	85	87	70	84			
11	83	88	80	71	63	56	56	56	57	45	42	46	49	45	39	43		
12	100	94	92	95	99	96	100	95	90	78	82	88	90	89	85			
13	100	86	70	77	86	90	83	66	57	75	91	69	60	52	50			
14	83	78	73	65	51	49	46	45	43	41	36	31	28	24	31	30		
15	102	99	96	94	94	88	83	78	62	51	46	34	21	14	40	40	21	
16	96	91	93	87	86	90	89	86	83	77	70	68	69	54	66			

APPENDIX 5

FORMS FOR ILLICITING INFORMATION FROM SUBJECTS

APPENDIX 5

UNIVERSITÉ D'OTTAWA UNIVERSITY OF OTTAWA

OTTAWA, ONTARIO
K1N 6N5 Canada

FACULTÉ DE PSYCHOLOGIE
DÉPARTEMENT DE PSYCHOLOGIE GÉNÉRALE ET EXPÉRIMENTALE

FACULTY OF PSYCHOLOGY
DEPARTMENT OF GENERAL-EXPERIMENTAL PSYCHOLOGY

CONSENT FORM

I consent to participate in an experiment on non-chemical treatments for migraine headache. I further understand that all procedures will be explained to me in advance. I also understand that no guarantees are made or implied that the treatment given me will be successful in treating my headaches. I understand that no medical responsibility is assumed or implied by the University of Ottawa or the experimenter by my participation in this experiment.

Signature of Participant: _____ Date: _____

Witness: _____ Date: _____

APPENDIX 5

HISTORY QUESTIONNAIRE
INFORMATION AND CHECK LIST

Length of prodrome_____ Age_____ Family history_____

No. of headaches per month_____

? Awaken with headache_____How often_____

Where pain begins_____How reliably_____

Knowledge of vascular theory (What causes the pain)

Explain the experimental nature, and that no fee will be charged other than their willingness to pay for transportation.

Willing?_____

Willingness to have headache(1 or 2) without medication.

Willing?_____

Available treatment times.

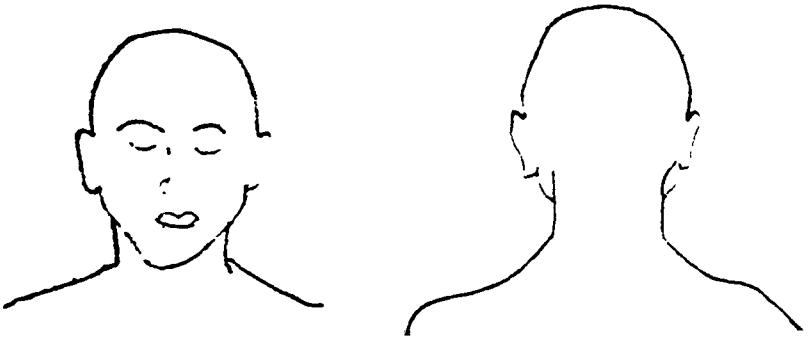
_____

Signed release form?_____

Pain questionare?_____

Patients questions._____

Comments:

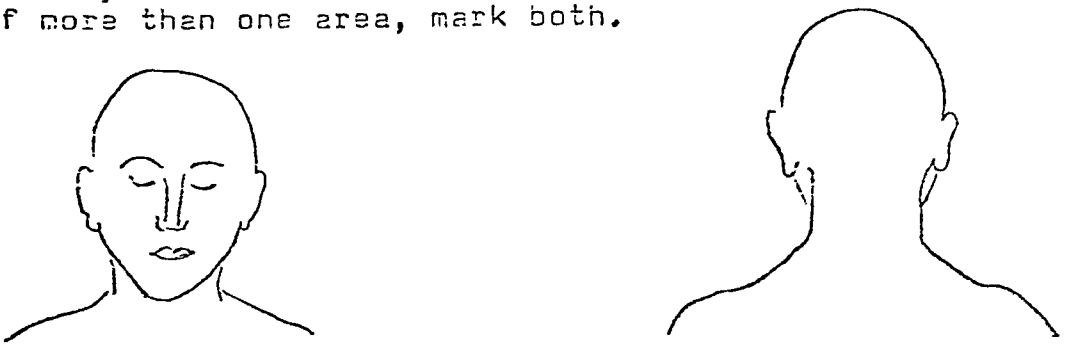


Name:_____
Phone:

MELZACK HEADACHE PAIN QUESTIONNAIRE

Name:_____ Date: _____

Part 1: Please mark, on the drawing below, the areas where you feel pain. Put E if external, or I if internal, near the areas which you mark. Put EI if both external and internal. If more than one area, mark both.



Part 2: What does your pain feel like? Some of the words below describe your headache. Circle only those words that best describe it. Leave out any category that is not suitable. Use only a single word in each appropriate category - the one that best applies.

Flickering	Jumping	Pricking	Sharp	Pinching
Quivering	Flashing	Boring	Cutting	Pressing
Pulsing	Shooting	Drilling	Lacerat-	Gnawing
Throbbing		Stabbing	ing	Cramping
Beating		Lancinating		Crushing
Pounding				
Tugging	Hot	Tingling	Dull	Tender
Pulling	Burning	Itchy	Sore	Taut
Wrenching	Scalding	Smarting	Hurting	Rasping
	Searing	Stinging	Aching	Splitting
			Heavy	
Tiring	Sickening	Fearful	Punishing	Wretched
Exhausting	Suffocating	Frightful	Gruelling	Blinding
		Terrifying	Cruel	
			Vicious	
			Killing	
Spreading	Annoying	Tight	Cool	Nagging
Radiating	Troublesome	Numb	Cold	Nauseating
Penetrating	Miserable	Drawing	Freezing	Agonizing
Piercing	Intense	Squeezing		Dreadful
	Unbearable	Tearing		Torturing

Part III: How Does Your Pain Change with Time?

1. Which word or words would you use to describe the pattern of your pain?

1	2	3
continuous	rhythmic	brief
steady	periodic	momentary
constant	intermittent	transient

2. What kind of things relieve your pain?

3. What kind of things increase your pain?

Part IV: How Strong is Your Pain?

People agree that the following 5 words represent pain of increasing intensity. They are:

1	2	3	4	5
Mild	Discomforting	Distressing	Horrible	Excruciating

To answer each question below, write the number of the most appropriate word in the space beside the question.

1. Which word describes your headache generally? _____
2. Which word describes it at its worst? _____
3. Which word describes it at its least? _____
4. Which word describes the worst toothache you ever had? _____
5. Which word describes the worst stomach-ache you ever had? _____

APPENDIX 6

ANALYSES WITH ONE SUBJECTS DATA REMOVED

APPENDIX 6

With the removal of one subject data, it was observed that there was a significant difference in pain during the constriction task while subjects were experiencing headache. This difference was not found when all eight subjects' data was analyzed. The power of the test for N = 8 was calculated showing a calculated power of .25. It is estimated that a sample size of 33 for .3 SD or 12 for .5 SD is necessary to bring the power of the test up to the desired level and show pain reduction during headache without bias.

The readers of this thesis are advised to examine the following analyses with extreme caution for the results gained with one subject dropped may be highly biased.

Using a one way analysis of variance, the head dilation task pain scores and head constriction task pain scores were found to be significantly different from baseline pain scores at the .01 probability level. Finger dilation task and constriction task pain scores failed to show a significant difference from baseline at the .05 level. This data is displayed in the following tables.

Newman-Keuls post hoc procedure revealed that pain during the head dilation task was significantly greater than

baseline and that pain during the constriction task was significantly less than baseline at the .01 probability level.

Using a three-way analysis of variance with repeated measures, significant interactions were observed for pain during different tasks.

Simple main effects were employed to determine significant differences between means. This analysis revealed a significant difference in pain (.05 level, $\underline{F}$ = 7.62) between dilation and constriction tasks when subjects concentrated on the head location. There also existed a significant difference in pain (.05 level, $\underline{F}$ = 8.02) between the head dilation task and the finger dilation task.

A graphic representation of the relationship between dilation time and relative pain change magnitude is displayed in the following figures.

APPENDIX 6

Three-way Analyses of Variance with Repeated Measures Using Relative Pain Change Magnitude Scores (N=7)*

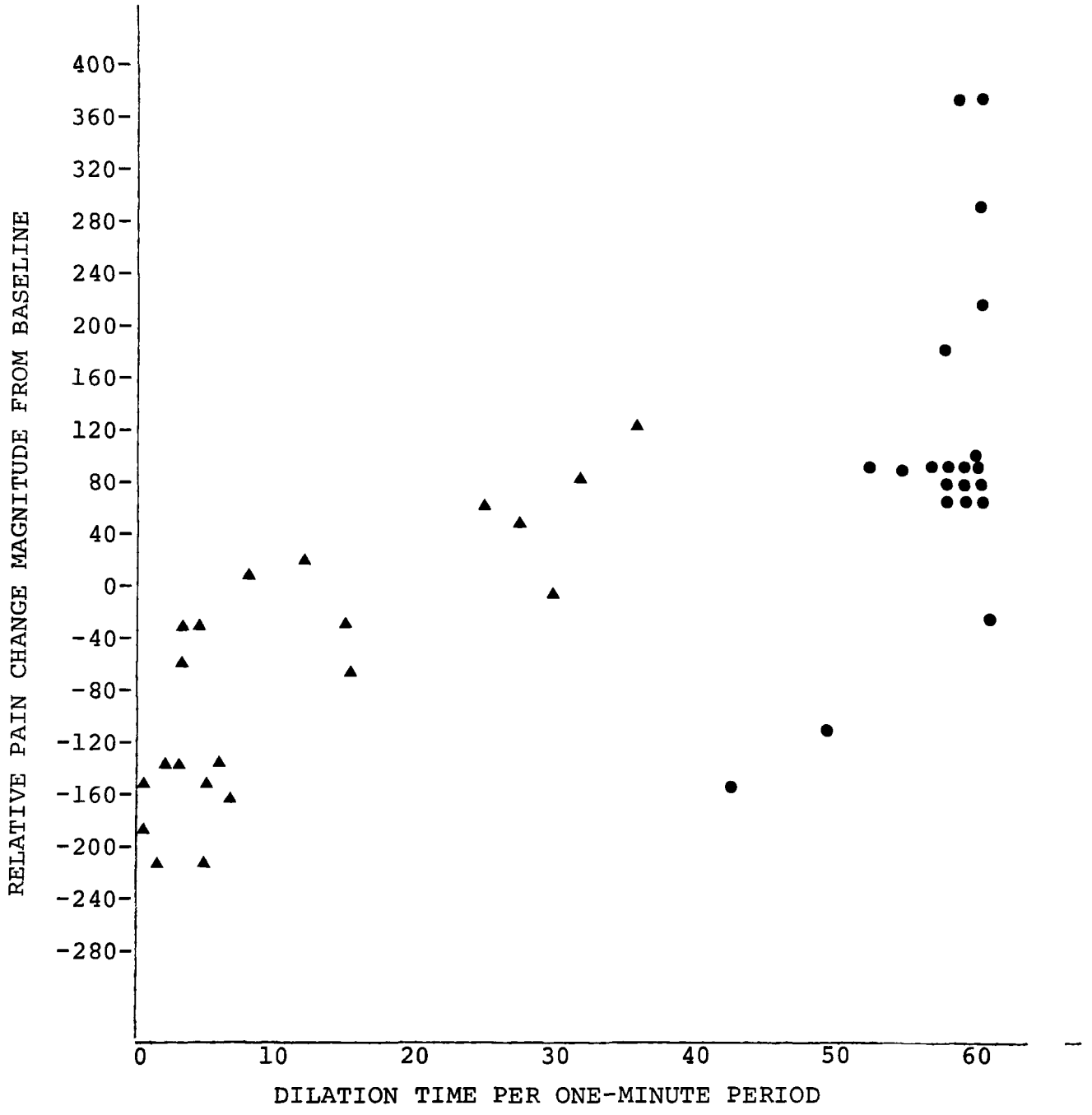
Source of Variation	SS	df Num	df Den	MS	F	p
Location(A)	1107.44	1	6	1107.54	.588	ns
Instruction(B)	71750.29	1	6	71750.29	8.956	.05
Sequence of observation(C)	1116.66	2	12	558.33	.367	ns
A X B	351657.44	1	6	351657.44	7.597	.05
Error				46284.52		

*Twelve observations were recorded for each subject

One-way Analyses of Variance for Each Task Using Scores of the Relative Change in Magnitude of Headache Pain
(N=7)

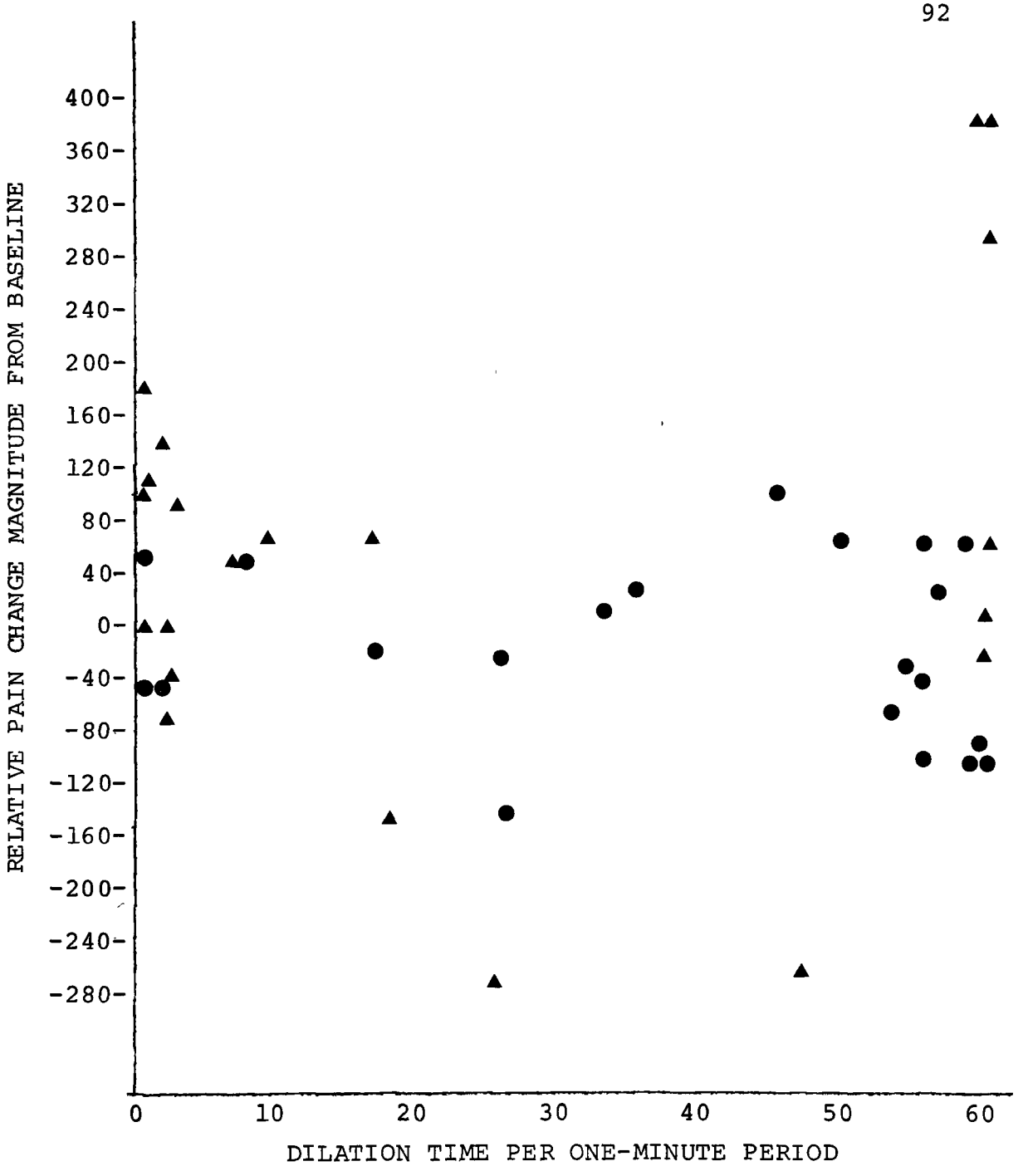
Location	Task	Baseline mean	Baseline SD	Task mean	Task SD	Mean squared error	df Nun	df Den	F	p
	Dilation			612.66	106.84	8207.97	1	40	28.16	.01
Head		464.28	70.72							
	Constriction			396.90	82.64	5915.25	1	40	8.06	.01
	Dilation			538.52	46.70	5108.60	1	40	.542	ns
Finger		554.76	89.64							
	Constriction			609.48	148.26	15009.13	1	40	2.094	ns

Three observations were recorded for each subject for each task at each location.



Relative Pain Change Magnitude Scores versus Dilation Time Scores for Dilation and Constriction Tasks at the Head Location during Headache (N=7)

Head dilation ●
Head constriction ▲



Relative Pain Change Magnitude Scores versus Dilation Time Scores for Dilation and Constriction Tasks at the Finger Location during Headache (N=7).

Finger dilation ●
Finger constriction ▲