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Autogenic Relaxation And Hand Temperature Biofeedback For Migraine

Barton Allen Jessup

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AUTOGENIC RELAXATION AND HAND TEMPERATURE BIOFEEDBACK FOR MIGRAINE

by

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Submitted in partial fulfillment
of the requirements for the degree of
Doctor of Philosophy

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Abstract

This study assessed the placebo effect and mediational processes in the treatment of migraine by autogenic relaxation with or without hand temperature biofeedback.

Subjects were 13 male and 47 female volunteers suffering at least 2 migraines per month. Mean age was 41. Throughout the 12 week program all subjects recorded headache frequency, intensity, duration, density (intensity X duration), and drug use. At weekly lab visits forehead and finger temperature and frontalis muscle tension (EMG) were monitored for 15 minutes while subjects engaged in their experimental activity for that week. Over the first four weeks physiological baselines ("sit and relax"), headache symptoms, a pain self-report on Melzack's McGill Pain Questionnaire (MPQ) and personality data from Cattell's 16PF, Jackson's Personality Research Form (PRF) and Jackson's Differential Personality Inventory(DPI) were collected. During the remaining eight weeks, two random groups of 12 subjects attempted to learn hand warming at home by listening daily to an autogenic cassette. The tape was augmented by a finger temperature biofeedback meter for four of the eight weeks. The two groups differed only in the order in which they used the meters. Similarly, two groups attempted to learn hand cooling, while the fifth group continued to keep the headache record and visit the lab only (i.e. continued baseline).

The physiological and headache record variables were analyzed in 5 X 3 X 4 (treatments X blocks X weeks within blocks) analysis of variance (ANOVAs). The 14 subscales of the MPQ were analyzed in a 5 X 2 (treatments X pre/post) ANOVAs.

End of session forehead temperature and reported migraine frequency

and intensity declined significantly over blocks (Univariate F ratios $p < .003$ each, $p < .05$ analysis-wise) but these decreases were not related to physiological control, which the subjects did not exhibit. Mean finger temperature increased 1.6°C across the 720 sessions monitored. Although both headache frequency and the end of session forehead temperature decreased they did not covary (Pearson's r=.02). Factor analysis clearly separated the physiological and headache record variables, further suggesting that improvement report was unrelated to physiological changes. Nearly significant pre/post trends appeared for the "temporal" and "fear" subscale of the MPQ ($p < .007$ and $p < .005$ respectively, $p < .05$ analysis-wise). The five treatments did not differ significantly on any of the dependent variables.

When participants were divided, regardless of original treatment, into 15 best "improvers", with more than a 50% decrease in reported headache frequency from Block 1 to Block 2, 20 moderate improvers, with less than a 50% decrease and 25 non-improvers, several distinguishing features emerged. Multivariate discriminant function analysis showed that the best improvers had headaches which were helped by sleep, not present on awakening, occured in clusters, and were accompanied by miosis and sensitivity to odours, light and sound. Both improvement groups appeared depressed, scoring high on the DPI scales of Cynicism, Depression, Self Depreciation, Ideas of Persecution, Broodiness and Desocialization. The best improvers and, to a lesser extent the moderate improvers, were also emotionally reactive, scoring low on DPI scale Shallow Affect and high on Mood Fluctuation, Panic Reaction, and Irritability. Depression and emotional reactivity were low in the non-improvers.

Autogenic training and hand temperature biofeedback apparently are not specific, effective treatments for chronic migraine. They reduced reported frequency and intensity of headaches no more than record keeping and experimental attention. Migraine sufferers who are apt to report improvement under the placebo effect of experimental participation can be distinguished from non-improvers in terms of psychological distress and headache symptoms.

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> He knew her headaches and they were dreadful. They twisted her face and reduced her to a panting, sweating, grinning, quivering blob of pain. They filled a room and a house. They got into everyone around her. Mr. Pritchard could feel one of her headaches through walls. He could feel it all over his body, and the doctor said there was nothing to do about it. They injected calcium and they gave her sedatives. The headaches usually came when she was nervous and when things, through no fault of her own, were not going well.
>
> (Steinbeck, 1947, p.210)

Statement of the Problem

Numerous treatments for migraine have been attempted, including a wide variety of drugs, psychotherapy, and more recently, biofeedback (Sovak, Fronek, Helland & Doyle, 1976). Handwarming training (biofeedback) for migraine has become increasingly popular (e.g. Medina, Diamond and Franklin, 1976) but has not been rigorously evaluated. The present study assessed the specific effectiveness of hand temperature biofeedback, and related physiological, experiential, symptomatic and personality variables to headache change.

The role of all of these variables in migraine remains controversial. Accordingly the main issues are briefly outlined in the next three pages, and then background material is reviewed under the headings "Description and Epidemiology", "Physiological, Pharmaceutical and Chronic Pain Aspects" and "Personality Variables". "Behavioral Programs for Migraine", including biofeedback, are reviewed separately before going on to theoretical issues and specific hypotheses.

The American Medical Association has defined migraine as:

> recurrent attacks of headache, widely varied in intensity, frequency and duration. The attacks are commonly unilateral in onset; are usually associated with nausea and vomiting; in some are preceded by, or associated with conspicuous sensory, motor and mood disturbances; and are often familial. Evidence

> supports the view that cranial arterial distention and dilatation are importantly implicated in the painful phase but cause no permanent changes in the involved vessels.
>
> (Friedman, A., Finley, K., Graham, J., Kunkle, C., Ostfield, A., Wolff, H., 1962, Pp. 717-718)

Biofeedback has recently been applied to the problem of migraine. In human biofeedback, information about one or more of an individual's ongoing physiological processes is returned to him as an overt sensory representation (Birk, 1973). Biofeedback therapy for migraine began when Sargent, Walters and Green (1972, 1973) discovered that migraine sufferers benefitted substantially from hand warming training. Sargent's et al (1973) procedure combined autogenic relaxation training (Schultz & Luthe, 1969) with hand temperature biofeedback. Hand temperature was used as a single measure index of sympathetic arousal; increased arousal resulted in vasoconstriction at the extremities and thereby cooler hands. Sargent et al (1973) speculated that migraine was due to excessive sympathetic arousal. Hand warming training was thought to reduce migraine by reducing the activity of the sympathetic nervous system. Over a dozen hand warming migraine therapy studies quickly followed Sargent's et al (1973) lead (e.g. Andreychuk & Skriver, 1975; Medina, Diamond & Franklin, 1976; Turin, Note 1; Wickramaskera, 1973). However, except for a few cases (e.g. Turin & Johnson, 1976; Wickramaskera, 1973) the reports did not include hand temperature data to support the sympathetic over-arousal rationale, nor did they examine the relative contribution of autogenic training and biofeedback to the overall treatment outcome.

More recent research has used procedures which seem to contradict the earlier rationale. Using a direct feedback technique, Friar & Beatty (1976) trained nine migraineurs to constrict their temporal artery, while

a matched control group learned to reduce their hand blood flow (thereby making their hands cooler). The number of major headaches suffered by the temporal-artery-trained group fell to .54 of baseline, compared to .86 for the control group ($p<.05$). Friar and Beatty's (1976) results suggested that increased sympathetic activity (at least artery-specific activity) is beneficial, contrary to Sargent's et al (1973) rationale.

Three other factors further complicate these apparantly contradictory research findings. First, migraine is susceptible to a pronounced "placebo" effect. For example, Waters (1970) found that, of 79 women given a widely prescribed migraine medication and placebo, 51% responded to the medication, while 58% responded to the placebo. Mitchell & Mitchell (1971) emphasized that behavioural variables are relevant to migraine. They demonstrated a significant reduction in headaches following a combined program of relaxation training, systematic desensitization and assertiveness therapy. Stroebel and Glueck (1973) also noted the large placebo effect in many biofeedback programs. Adequate migraine biofeedback research must control for non-specific, placebo effects.

Secondly, the physiological mechanisms controlling migraine are not clearly understood. A great deal of migraine research examines possible biochemical mediators (c.f. Friedman, 1972), but the emphasis on pharmacological approaches is partially due to the difficulty of studying migraine by any other technique (Sicuteri, 1972a). Some researchers hold that the biochemical evidence is so far inconclusive (Dalessio, 1974; Sjaastad, 1975), and suggest instead a neurological basis for migraine Graham, 1968; Edmeads, Note 2). Sicuteri (1972b, 1976) has developed an "impaired central inhibition" theory of migraine that unites both neurological and biochemical factors, acting in the hypothalamus. As well,

clinical (Bruyn, Bootsma & Klawans, 1976) and experimental (VonZwieten, 1973; DeJong, Zandberg & Bohus, 1975) evidence suggests that inhibitory interneurons in the brain stem may effectively reverse some peripheral vascular effects of hypothalamic activity. No unequivocal predictions about the effects of biofeedback therapy for migraine can be made on the basis of physiological findings.

A third factor complicating the assessment of non-pharmaceutical migraine treatment is the probable effect of symptom and personality variables on treatment outcome. Diamond & Franklin (Note 3, Note 4) found that patients suffering from clearly diagnosed migraine, without muscle contraction or psychogenic components, responded best to hand warming biofeedback training. Depressed clients, and those with mixed or psychogenic symptoms benefitted only moderately. Clinicians have long viewed migraineurs as unusually ambitious, perfectionistic, hardworking, tireless and exacting (Wolff, 1937; Kolb, 1963). Psychometric assessments of migraine sufferers are sparse, although the few that have been reported tend to support the clinical impression (e.g. Kudrow, 1974). The relationship of symptom and personality variables to migraine, and to treatment outcome, requires systematic evaluation.

Seemingly contradictory biofeedback therapies have ameliorated migraine (Sargent et al, 1973; Friar & Beatty, 1976). Placebo, personality and symptom effects also influence treatment outcome (Diamond & Franklin, 1974; Waters, 1970). The physiological processes underlying migraine are still unresolved: biofeedback, as physiological learning, offers a possible technique for clarifying some of the biological issues. To examine these process and outcome variables, the present study compared opposing biofeedback treatments (hand warming versus hand cooling)

and the placebo effect of experimental attention. Symptom and personality variables were also assessed.

Description and Epidemiology of Migraine

> The more fundamental definition of what is migraine has eluded our best endeavours.
>
> (Pearce, 1975, p. 1)

> La classification des céphalées est un des problèmes nosologique les plus malaisés.
>
> (Nick, 1968, p. 359)

> Attempts to find a single defining criterion of migraine have been productive of disagreement.
>
> (Ziegler, Hassanein & Hassanein, 1972, p. 353)

Migraine is a functional disorder that has no known anatomical pathology (Walshe, 1969). Consequently, "migraine" is a clinical rather than a physiological diagnosis. Most definitions simply describe typical attacks (Waters & O'Connor, 1971). The descriptions have not specified how many of the characteristic symptoms must be present to establish a diagnosis of migraine (Waters, 1973). Tissot, in 1790, emphasized that migraine was "a disease entity distinctly separate from common headache" (p. 613), a view that continues to be held today. In 1888, Gowers described the principle features of migraine:

> Migraine is an affection characterized by paroxysmal nervous disturbance, of which headache is the most constant element. The pain is seldom absent and may exist alone, but is commonly accompanied by nausea and vomiting, and it is often preceded by some sensory disturbance, especially by some disorder of the sense of sight. The symptoms are frequently one-sided and from this character of the headache the name is derived...
>
> (p. 613)

More recent definitions of migraine by the American Medical Association (Friedman et al, 1962) and the World Federation of Neurology (1969) have been similar to Gower's (1888). The World Federation of Neurology

(1969) emphasized, however, that "all the characteristics are not necessarily present in each attack or in each patient" (p.3). Since migraine is not objectively measurable (Walshe, 1969), nor do consistent symptoms differentiate migraine from other syndromes, a diagnostic (and epidemiological) problem follows: a population cannot be divided clearly into migrainous and non-migrainous groups, except by arbitrary criteria.

Waters and O'Connor (1971) found that 87.5% of a random sample of women who reported having headaches with unilateral onset, warning signs and nausea were diagnosed clinically as having migraine. On the basis of that data Waters (1975) suggested:

> One approach to the problem of defining migraine is to look for evidence that these 3 migrainous features form a syndrome. A syndrome is a running together or concurrence of certain symptoms or signs. It is usually implied, although often not stated, that the concurrence is more than could be accounted for by chance.
>
> (p. 87)

However, when Waters (1973) compared the observed concurrence of any two or all three of the diagnostic migraine symptoms to their chance concurrence, he found no significant difference. Based on the prevalence of the three symptoms in the 12 months preceeding the survey, the observed concurrence exceeded the chance concurrence by only 35 people in a general population sample of 1,718.

Waters (1973) offered four possible interpretations of his findings. (1) The prevalence of "real" migraine in the population was extremely low, consisting of the 35 cases in 1,718 (1.3%) not accounted for by chance. (2) The prevalence of migraine was extremely high. Since most of the diagnoses of migraine in the earlier studies (Waters & O'Connor, 1970, 1971) could be accounted for by the chance concurrence of the three

diagnostic symptoms, perhaps individuals with only one or two of the symptoms should also be considered migrainous. Following this line of reasoning, the prevalence of migraine became 39.9% for men and 57.5% for women. Both figures were much higher than the 10% prevalence estimate commonly held by clinical opinion (British Medical Journal, 1963). (3) A "neurotic" tendency to reply positively to any medical questionnaire may have slightly inflated the number of people reporting all three symptoms of migraine. Waters (1973) found that "neuroticism", as measured by the Cornell Medical Index Health Questionnaire, covaried with the number of migraine features reported. The neuroticism score was higher for individuals with all three migraine symptoms than for those with one or two symptoms, non-migrainous headaches or no headaches. (4) Finally, Waters (1973) suggested that,

> migraine might be an extreme in a continuum rather than a completely distinct clinical entity which the patient either has or has not got.
>
> (p. 193)

This idea was supported by the finding that symptoms were more prevalent in people who rated their head pain as more severe. No matter which interpretation is accepted, the possibility of confounding chance and "real" migraine syndromes remains.

Other researchers have also attempted to clarify the definition of migraine, using different techniques. Ziegler et al (1972) factor analyzed 289 headache patients' response to 27 items on a self-administered questionnaire. Seven migraine factors emerged (varimax solution). None of the factors contained all three of Waters' (1973) defining symptoms, indicating that the three symptoms were not highly interrelated, contrary to clinical belief.

Waters' (1973) and Ziegler's et al (1972) findings were surprisingly convergent. Despite different populations (Welsh general public versus a Kansas medical center, respectively), different statistical techniques (frequency distribution versus factor analysis), and different questionnaires, both researchers found that the "syndrome" of migraine can be considered a clinical diagnosis only. Waters (1973) noted that the the concurrence of the symptoms defining migraine could virtually be accounted for by chance. Ziegler et al (1972) found that the defining symptoms did not highly intercorrelate, which could be due to randomness or to other characteristics of the symptom distribution (c.f. Weckowicz, 1973).

The problem of clearly defining the subject population is extremely important in migraine research. Numerous studies reported abnormal physiological and biochemical processes in migraine sufferers, yet attempts at replication often provided inconclusive or contradictory results. For instance, blood serotonin levels were observed to stay constant during migraine (Kimball & Friedman, 1961), to drop sharply (Lance, Anthony & Gonski, 1967) and to vary unreliably (Sjaastad, 1975). Similarly inconsistent findings have appeared for electroencephalographic (EEG) abnormalities (Weil, 1952; Giel, DeVlieger & Van Vliet, 1966). Although researchers may simply be examining irrelevant variables, the inconsistent findings could be due to differences in the subject populations under study. A clear definition of the research population is obviously desirable, both to minimize within group error variance and to facilitate comparisons between experiments.

Some researchers have defined their study population by using subject selection criteria. The criteria have varied between studies.

Ostfield (1962) defined as migrainous only those subjects whose headaches responded to ergotamine but not to placebo. Lance and Anthony (1966) selected subjects who had "recurrent severe headache with gastro-intestinal disturbance or focal neurological symptoms or both" (p.112); response to ergotamine was ignored. Ekbom (1970) used the same criteria as Lance and Anthony (1966) but further restricted his sample to people who had more than a five year history of chronic headaches, with each headache lasting at least three hours. Whitty (1968) studied patients who had:

> recurring throbbing headache and in addition two of the following five features: unilateral headache, associated nausea with or without vomiting, visual or other sensory aura, cyclical vomiting in childhood, and a family history of migraine. (p. 735)

Vahlquist (1955) and Bille (1962) both defined migraine as paroxysmal headaches with at least two of four additional characteristics; unilateral pain, nausea, visual aura and family history. The explicit selection criteria used in the studies just discussed are more informative than global clinical diagnoses. However, a detailed description of the subjects' symptoms, and the relationship of those symptoms to treatment outcome, would further enhance the value and replicability of migraine research.

Several investigators have developed diagnostic migraine questionnaires (e.g. MacNeal, 1964). Some of the questionnaires permitted computerized diagnoses (Freemon, 1968; Toole, Brady, Cochrane & Almos, 1974). Diagnostic differentiation of migraine was based mainly upon the patients' head pain history and symptom pattern (Diamond, 1974). Using questionnaires, the symptoms were summed to generate a differential diagnosis.

This procedure proved effective in one study (Freemon, 1968) but failed to replicate in another (Toole et al, 1974). Both studies used a neurologist's opinion as the accurate diagnostic criterion.

Headache is normally classified into three main sub-groups; (1) vascular, (2) psychogenic and (3) traction and inflamatory (Diamond & Dalessio, 1973; Friedman et al, 1962). Vascular headaches include classic and common migraine, cluster, opthalmoplegic, hemiplegic and hypertensive and toxic headaches. The common pain mechanism is vasodilation at the head (Friedman, 1964). Vasoconstriction may precede the vasodilation and account for the painless sensory and motor disturbances preceeding some forms of vascular headaches, such as classical migraine.

Psychogenic headache is charcteristically seen in anxiety, depression and sometimes after head trauma. The pain mechanism is thought to be sustained contraction of the muscles of the head, neck and face producing a dull bandlike pain which may last for days, months or years (Rodbard, 1970). This type of headache is termed psychogenic because, while not organic, it is persistant and typically appears during disturbances of mood.

Traction and inflammatory headaches can be caused by brain tumors, subarachnoid hemorrhage and meningitis. Breutman (1974) emphasized that headache is infrequently caused by organic disease. He found only one tumor in a series of 100 headache cases that he assessed. Heyck (1964) noted that 54.2% of 778 patients with growing tumors had headaches as an early symptom. That is, although very few headache cases prove to be organic, half of the tumor cases had headache as an early symptom, and half did not. Breutman (1974) noted that,

> ...statistical data are unavailable from a large population on the number of patients with headache as an only symptom who already have a brain tumor or in whom a tumor later develops." (p. 120)

The main overall diagnostic problem, then, is to differentiate migraine from psychogenic headache. If possible, the separation of organic cases from the two types of functional headaches would also be highly desirable, but the low frequency of organic pathology in headache populations makes differentiation impossible without enormous samples.

A comprehensive diagnostic questionnaire should incorporate clinical lore, research findings and possibly etiological speculations. Diamond & Dalessio (1973) emphasized that,

> a detailed and relevant history, geared to the headache patients is the most important factor in making a correct diagnosis. (p. 10)

Clinicians are essentially in agreement about the requirements for an adequate headache history. Young (1966) suggested that headache be assessed under the headings "location, severity, onset and duration, character and modalities" (p. 67). Diamond (1974) offered a headache history outline that included:

> types of headache present, onset, location, frequency, duration, prodromata, associated symptoms, precipitating factors, sleep pattern, emotional factors, family history, medical history, allergies, surgical history and medication history. (p. 70)

MacNeal (1964) formulated a thirty-seven item questionnaire which assessed the clinical variables also discussed by Young (1966) and Diamond (1974) and added the results of several biomedical tests. Fremon's (1968) computerized diagnostic form used thirty clinical items "based

mainly on the patient's history" (p. 49). Toole et al (1974) developed a questionnaire from:

> (1) the questions routinely asked by the neurologists at our hospital for evaluating patients with headache, (2) a review of standard tests and (3) other questionnaires which contain information pertaining to headache. (p. 73)

Epidemiological survey questionnaires, such as those used by Ogden (1952) and Taylor, Pocock, Hall & Waters (1970) included items similar to those in the clinical forms. A comprehensive headache questionnaire then, should tap the pertinent variables while permitting statistical analysis and test refinement.

A major weakness in the migraine questionnaire studies has been their failure to relate symptoms to treatment outcome. With the exception of Ziegler et al (1972) researchers have simply summed symptoms to arrive at a diagnosis. The symptom summation procedure fails to assess the effectiveness of each item (or factor of items) for discriminating patients who respond to a treatment from those who do not.

The evaluation of questionnaire data could be enhanced by relating symptoms to treatment outcome. One possible technique is Multivariate Discriminant Function Analysis (MDFA: Klecka, 1971; Van de Geer, 1971). Veldman (1967) noted the main application of MDFA:

> The problem is to determine the extent and manner in which two or more previously defined groups of subjects may be differentiated by a set of dependent variables acting together. (p. 268)

The number of dependent variables may be reduced to the most salient features of the data by pre-processing with factor analysis (Tatsuoka, 1971). Migraine patients could be divided into groups on the basis of their symptomatic improvement, - e.g. best improvers, moderate improvers, non-

improvers - and then their factor scores from a headache questionnaire could be analysed by MDFA to determine which (if any) features of their symptoms were related to clinical improvement. This technique has two advantages. It begins by separating patients into the groups that are ultimately of most clinical importance, improvers and non-improvers. Secondly, the improvement groups are described on the basis of their original symptoms, thereby providing a basis for future triage.

Sacks (1970) noted the variability of migraine symptoms and emphasized the importance of distinguishing homogenous patient groups in research.

> Actual clinical experience soon persuades anyone who works with migrainous patients that they are an exceedingly heterogeneous group. Some have classical migraines, some common: some have striking family histories, many have no family histories; some have allergies, some do not; some react to particular drugs, some do not; some are sensitive to alcohol or passive motion, some are not; some outgrow their attacks at a youthful age, others start them at a later age; some have red migraines and some white; some have predominent visceral components, and others chiefly cephalgic components; some are hyperactive, some are lethargic; some are obsessional, others are sloppy; some are brilliant, and some are simpletons. ... In short, migraine patients are as remarkable for their diversity as any other section of the population. Such heterogeneity of the population and the symptoms under survey may invalidate and render meaningless any statistical survey, and demand, for investigative purposes, that the clinical material be broken down into smaller and more homogeneous groups. If the data are disparate, they must not be put together for purposes of comparison. (p. 147-148)

In clinical migraine research the most appropriate procedure for defining the subject population might involve two stages, an initial global selection followed by a more exact symptom analysis. The initial selection, using traditional diagnostic criteria (e.g. Bille, 1962; Whitty, 1968; Vahlquist, 1955), would provide a group of subjects fitting the overall migraine pattern. The symptom analysis - e.g. by MDFA of

questionnaire responses - would serve to identify the symptoms characteristic of patients responsive to treatment, within the broad framework established by the initial selection. By using this two stage procedure, a manageable segment of headache problems could be studied without loosing descriptive precision.

Epidemiologists have assessed the prevalence of migraine in various populations. Ogden (1952) and Waters (1974) examined extensive general population samples. Other researchers evaluated migraine incidence in medical practices (Fitz-Hugh, 1940; Fry, 1966; Logan & Cushion, 1958; Newell, Note 5). The prevalence figures reported differed widely, probably due to two main research problems. First, the definition of migraine varied from study to study, and some studies used no definition at all, but relied simply on clinical diagnosis. Secondly, some studies surveyed only patients who attended physicians. Patients seen in hospitals or private practice are obviously not representative of the general population in that area. Cochrane emphasized this problem in 1965:

> One would imagine that it would be self-evident that those coming into hospital with a particular disease are not usually typical of all people suffering from that disease, and that those, without a particular disease, in hospital, are not typical of all those, in and outside hospital, lacking the disease. But a great deal of clinical research has been ruined by neglect of these simple facts and research into syndromes has suffered more than most. (p. 440)

In reviewing the epidemiological data on migraine the population base must be kept in mind.

Ogden (1952) performed the first major epidemiological study of headache. He surveyed 4,634 employed adults, 30.2% male and 69.8% female, in the New Orleans area of the U.S.A. Sixty-five percent of all subjects answered "yes" to the question "do you have headaches?", 50.7% of the

males and 70% of the females. The sex difference was statistically significant (chi squared, $p < .001$). Only 18% of the sample had sought medical attention for their headaches. For males and females combined, the frequency of headaches was highest in the 21-30 age group (37.8%) and declined with age to only 2.2% of those over 60. The decline with age was also statistically significant (chi squared, $p < .001$). One percent of all subjects had daily headaches, and 31% had more than 2 headaches per month. Most headaches lasted 1-6 hours (44%), but 9.1% lasted for more than 24 hours. Headaches in Ogden's (1952) survey included all types; vascular, psychogenic and organic.

The overall proportion of vascular headaches in Ogden's (1952) survey can be roughly estimated by examining his findings for specific symptoms. Unfortunately, Ogden's (1952) data presentation precludes calculating the concurrence of two or more symptoms. One defining migraine symptom is the location of the headache pain; migrainous pain is characteristically unilateral or behind the eyes (Friedman et al, 1962). Thirteen percent of the headache sufferers in Ogden's (1952) survey reported that their pain occured "most often" on one side of the forehead. Another 5.6% had headaches on one side of the head, and 12.6% noted pain around the eyes. If all three of these head pain locations are taken to indicate migraine, then 20% of Ogden's (1952) total sample might have suffered migraine (64.8% X (13%+5.6%+12.6%)). Migraine pain is often described as "throbbing"(Friedman et al, 1962). Nearly 30% of Ogden's (1952) headache fraction reported throbbing pain, suggesting a migraine prevalence of 19% (64.8% X 29.8%). Similarly, since 28.1% of the headache fraction became nauseated or vomited during their headaches, the

migraine prevalence using this index would be 18%. If a visual disturbance before or during the headache is used as the migraine index, the prevalence estimate becomes 19% (64.8 X 28.9%). Thus, in Ogden's (1952) survey four migraine symptoms (unilateral location, throbbing pain, nausea and visual disturbances) were present in about 30% of headache sufferers. In an employed, adult population the prevalence of headaches with at least one migraine symptom was approximately 20%.

Waters (1974) examined headache variables in a random sample of 2,000 adults in the Welsh Parliamentary Constituency of Pontypridd; which has a social class composition similar to that of England and Wales as a whole. A total of 1,718 completed symptom questionnaires were obtained. The proportion of respondents reporting at least one headache in the year preceding the survey was 63.5% for men and 78.4% for women. The sex difference was significant (chi squared, $p < .001$). Across seven categories of headache severity, from "very mild" to "almost unbearable" women reported more severe headaches than men. Women also tended to have headaches of longer duration; 14% reported that their headaches lasted over 12 hours, versus 7% for men. Women also had significantly more frequent headaches; 67% of women with headaches reported more than one per month, versus 48% of men. The prevalence of three migraine symptoms (unilateral onset, nausea, and warning) increased linearly with self-rated headache severity. In the total sample of 1,718 only 38 men (8% of male headache sufferers, 4.9% of all males) and 86 women (12% of female headache sufferers, 9.1% of all females) had all three migraine features in the preceding year. The prevalence of the three migraine symptoms declined with age, as did the overall prevalence of headaches.

The distribution of migraine in the general population varies then with both sex and age. Water's (1974) findings suggest that a reasonable estimate of migraine prevalence would be 5% for males and 9% for females, although earlier studies by the same author (Waters, 1971; Waters & O'Connor, 1971) offered prevalence estimates of migraine as high as 19% for women.

Waters and O'Connor (1971) described the validation of the symptom questionnaire used in the Pontypridd headache survey. A random sample of 117 women from various symptom subgroups identified by the questionnaire were examined clinically. The concordance between the presence of unilateral onset, nausea and prodome as reported on the questionnaire, and a blind clinical diagnoses of migraine was 87.5%. The presence of only one or two of the three symptoms predicted the clinical diagnoses much less reliably. Of the headache sufferers examined, the clinician diagnosed 58% with unilateral pain and prodrome as migrainous, 32% with unilateral pain and nausea, and 60% with prodrome and nausea. Extrapolating the proportions of each subgroup diagnosed as migrainous to the entire sample population, Waters and O'Connor (1971) suggested that 558 out of 2,933 women aged 20 to 64 years, or 19% suffered migraine. (Dalsgaard-Neilsen (1970) also reported a 19% prevalence of migraine in women by age 40, versus 11% for men, based on a survey of 2,027 Danish school children and adults.) Only 23% of those diagnosed as having migraine by Waters & O'Connor (1971) had consulted a doctor about their headaches.

The finding that nearly one half of female migraineurs had never seen a physician about their headaches (Waters & O'Connor, 1971) empha-

sizes the problem of subject selection in migraine research. Headache patients attending a medical practice may not be representative of all sufferers. Further research by Waters (1971) clarified two variables tending to discriminate migraineurs who seek medical assistance from those who do not. Clinical opinion has long held that migraine sufferers tend to be above average in intelligence and social class (Friedman, 1964; Lennox, 1941). Waters (1971) randomly sampled 400 individuals who had been identified by questionnaire as falling into four groups; (1) non-migrainous headaches, (2) unilateral headaches, (3) migraine (unilateral pain, nausea and prodrome) and (4) no headache in the previous year. Waters (1971) found no correlation between social class or intelligence and migraine. However, migraineurs who were more intelligent or in higher social classes were more apt to see physicians. In a four category social class system, 81% of migraineurs in the upper two social classes had consulted a doctor, compared to 62% in the lower two classes. The clinical opinion that migraine sufferers are of higher than average intelligence and social class probably arises from physicians seeing only a selected sample.

Despite the sampling bias, several authors have assessed the prevalence of migrainous patients in medical practices. Fitz-Hugh (1940) examined 4,000 case records from general practice and found that 26% of females and 16% of males had migraine at sometime during their lives. Walker (1959) found that 4.85% of 5,785 patients seen in general practice reported a past or present history of "recurrent" headache. Reviewing his own practice, Fry (1966) noted that 7.7% of his female patients had had migraine in a 10 year period. Nick (1968) found that migraine was

diagnosed in 15% of 2,350 cranio-facial pain cases seen at a French neurological clinic. By comparison, muscle contraction headache was diagnosed in 52% of the cases. Selby and Lance (1960) found that 12% of all patients seen at an Australian neurological clinic suffered from "vascular" headaches. The prevalence of migrainous patients seen in medical practice varies from 5 to 26 percent, depending upon the type of practice and the time span of the case records under study.

Selby and Lance (1960) described selected characteristics of 500 migraine sufferers who visited a neurological clinic. Sixty percent were female. The female preponderance was similar to the 62% figure reported by Fitz-Hugh (1940), but slightly lower than the 71.6% female preponderance noted by Wilson (1955) in a review of 3,278 published cases. Selby and Lance (1960) found that the modal age of onset was from 20 to 30. Migraine attacks began before the age of 40 for 92% of their sample. More than half of the patients suffered one to four headaches per month. One third of the patients reported headaches lasting longer than 24 hours. The head pain was unilateral in 190 of the 500 cases, and bilateral in 191 cases. "Mixed" muscle contraction and migraine symptoms appeared in 113 patients.

Selby and Lance (1960) included cases with bilateral pain in their migraine series on the basis of associated clinical symptoms. The observation that 38% of migraines were bilateral was in accord with earlier estimates by other authors (Critchley & Ferguson, 1933). Symptoms accompanying migraine included nausea (87% of cases), vomiting (56%), photophobia (82%), visual disturbances (41%), parasthesia (33%), and dizziness (72%). Of 263 migraineurs who used ergotamine tartrate, 47% obtained "excellent" relief, 34% "good" relief and 19% no relief. The response

to ergotamine was similar to that of 1,070 cases reported by Vallery-Radot (1955), and differed less than 2% between both unilateral and bilateral headaches and the presence or absence of classical migraine symptoms. Selby and Lance (1960) noted that the frequency of symptoms accompanying migraine was "remarkably similar", regardless of the laterality or neurological characteristics of the headache. They concluded:

> The similarlity of the clinical picture in the various (headache) groups indicates that there is no justification for regarding "classical migraine" as an entity distinct from other forms of extracranial vascular headache. (p.30)

Another epidemiological feature of migraine that has been assessed is the proportion of medical consultations accounted for by the syndrome. Logan and Cushion (1958) studied 106 general practices in England and Wales, and found that the annual proportion of consultations for migraine was 0.8% in women and 0.3% in men. Newell (Note 5) in a Canadian study noted that headache was a presenting symptom in 8.5% of all patients attending a family practice in one year, and that headache accounted for 2.5% of all visits. Migraine formed, however, only a small proportion of these totals. Migraine was the diagnosis in 0.14% of female consultations and in 0.8% of male consultations. The differences between the British and the Canadian findings could be due to differences in patient behaviour or diagnostic practices. Since the prevalence of migraine falls in a similar range across countries, the difference in consultation rates probably does not reflect national differences in the incidence of migraine.

As well as causing considerable personal suffering, migraine also costs the economy a great deal each year. The British Office of Health Economies estimated that migraine cost the National Health Service

£2,800,000 in 1970. This cost was made up of £1,600,000 in pharmaceutical payments, £700,000 in general practice payments, and £500,000 in hospital services (Pearce, 1975). The private purchase of analgesics doubtlessly increases the total cost considerably. In the year 1968-69 470,000 working days (.008% of all working days) were lost to migraine in Britain, although even this figure is a gross understatement, since only absences of three days or longer were included (Pearce, 1975). The cost of migraine in suffering, medical care and lost production is substantial.

For many years researchers have debated the possibility of a genetic factor in migraine. Studies have claimed to show a strong hereditary component (e.g. Goodell, Lewontin & Wolff, 1954) or a weak hereditary component (Lucas, Note 6; Waters, 1971a) multifactorial inheritance (Refsum, 1968) or inheritance of a specific end-organ defect (Lance, 1974). Variations between studies in sampling, methodology, interpretation and the definition of migraine have made comparison difficult. Nonetheless, migraine appears to have a slight genetic component, and possibly a somewhat stronger familial (environmental) influence.

The bulk of genetic research on migraine consists of familial incidence studies. Allan (1928) interviewed 382 migrainous patients and found that 349 or 91.4% reported migraine in one or both of their parents. In Lennox's (1941) series of 425 migraineurs, 61% recalled that "some relative" also suffered "sick headache" compared to 11% in a control group of student nurses and general hospital patients. Selby and Lance (1960) noted a "family history" in 50% of 290 neurological clinic patients. Familial incidence figures in the 50% to 90% range have also been repor-

ted by numerous other authors (e.g. Burns, 1965; Childs & Sweetnam, 1961; Dalsgaard-Neilsen, 1965; Lance & Anthony, 1966). In an extensive review Refsum (1968) found that familial incidence figures ranged from 57% to 91.4%. Similarly, Friedman (1968) affirmed:

> The incidence of migraine in the offspring of an affected parent usually is reported at 50 per cent or higher. (p. 43)

One of the most ambitious studies of migraine heredity was by Goodell, Lewontin & Wolff (1954), who carefully interviewed 119 migraine patients with "severe headache recurring usually over many years" (p.326). The patients described the incidence of migraine in their families, sometimes presenting information on up to 5 generations. The final tabulation contained data on 832 offspring in the 119 migrainous families. Goodell et al (1954) found that 28.6% of those with neither parent affected had migraine, 44.2% of those with one parent affected had migraine and 69.2% of those with both parents affected had migraine. Since "the probability of migraine headache increased with the number of parents affected" (p. 332), a heredity factor seemed likely.

Unfortunately, when Goodell et al (1954) attempted to interpret their findings in terms of the Medelian laws of inheritance, they failed to note that their data refuted their conclusions. Goodell et al (1954) argued that migraine was due to a recessive gene with 70% penetrance. This conclusion followed from the assumption of a recessive gene for migraine, and the observation that approximately 70% of children with two migrainous parents were also migrainous. Since both parents, suffering migraine themselves, must each carry the recessive gene pair for migraine, all their children would also carry the migraine gene pair. Since

only 70% of the children suffer migraine when both parents do, the penetrance must be limited (presumably by another related gene) to 70%. Goodell's et al (1954) interpretation fits the results for children of two migrainous parents. However, the interpretation does not fit the results for children who have one or neither parent with migraine. When one parent is migrainous he or she must carry the recessive migraine gene pair. The other parent could homozygously carry the dominant non-migraine gene pair, or be heterozygous, carrying one dominant non-migraine gene and one recessive migraine gene. When the migraine free parent is homozygous, none of the children would have migraine, since they would all be heterozygous. If the migraine free parent was heterozygous, half of the children would carry the recessive migraine gene pair. Given 70% penetrance only 35% (70% of 50%) of the children who have one migrainous parent could suffer migraine themselves, even under the optimal (and improbable) assumption of heterozygosity in all non-migrainous parents. The 35% maximum possible incidence of migraine in children with one migrainous parent implied by Goodell's et al (1954) theory is substantially less than the 44% actually observed in their study. Similarly, the maximum theoretical incidence of migraine in the children of two headache free parents is 17.5%, again much less than Goodell's et al (1954) observed incidence of 29% for such children. Goodell's et al (1954) data do not appear to support their genetic interpretation. Sampling biases, noted by the authors, also weaken the study. The Goodell et al (1954) study, frequently quoted as support for a genetic basis of migraine, does not appear to offer as much proof as it claims.

Another major study of migraine heredity used an entirely

different approach, with findings notably divergent from those usually reported. Using a validated diagnostic questionnaire, Waters (1971a) identified individuals with no headaches, headaches, unilateral headaches or migraine (unilateral headaches with prodrome and nausea) in a random sample of an electoral district in Wales. A random subsample of the first degree relatives (parents, siblings, children) of people in the no headache, headache and migraine groups were visited and administered a short headache questionnaire. The family tree was confirmed during the visit. The data collectors were unaware of the headache histories in the original sample. In all, 519 first degree relatives of 155 original probands completed the questionnaires. Each relation was categorized as migraine (three features), possible migraine (one or two features), headache, or no headache. The prevalence of migraine was 10% in the families (first degree relatives) of migraine sufferers, 5% in the families of headache sufferers, and 6% in the families of the no headache group. Thus, migraine was approximately twice as prevalent in the families of migraine sufferers as it was in the families of headache free individuals, although the difference did not attain statistical significance.

Waters (1971a) reported the incidence of migraine in the parents and siblings of migrainous and headache free probands. Ten percent of the siblings of migraineurs also suffered migraine, as did 7% of the siblings of the no headache group. Conversely, 28% of the siblings of migraineurs did not suffer migraine, while 30% of the siblings of the headache free group did not suffer migraine. Waters (1971a) presented the incidence of migraine in the parents of his probands separately for

mothers and fathers. The relationship of migraine in children to its presence in one or both parents could, therefore, not be assessed. In the families of migraine sufferers 16% of the mothers and 3% of the fathers reported also being afflicted, while 19% of the mothers and 28% of the fathers were headache free. Waters (1971a) combined the parents' data for non-migrainous headache and no headache probands; a lower proportion of their parents suffered migraine (8% of mothers, no fathers), while a higher proportion was headache free (34% of mothers, 43% of fathers). Waters' (1971a) account suggests that the presence of migraine is only slightly related to its presence in siblings, and that parents of migraine sufferers are about twice as likely to have migraine themselves as the parents of people without migraine. Waters (1971a) concluded that "heredity is much less important in migraine than is usually supposed" (p. 77).

Reports of familial incidence of migraine have varied from 10% to 91.4%. Several methodological problems contributed to this wide variability in results. The lack of a clear definition of migraine prevents comparisons between studies. For example, Waters (1971a) defined migraine as a unilateral headache with warning and nausea, while Goodell et al (1954) used a broad definition incorporating a wide range of symptoms. The much smaller familial incidence observed by Waters (1971a) was consistent with his more restrictive definition. Bound up with the lack of a consistent definition for migraine is the problem of assuming genetic homogenity for all migraine populations (Sacks, 1970). The hereditary interpretation of familial incidence assumes that all migraines, no matter how divergent their symptoms, all stem from a common genetic base.

Such an assumption can only be tested by identical twin studies; there are no a priori reasons for accepting it. Furthermore, the design of familial incidence studies confounds the environmental influence of family life with genetics.

> The mere fact of having one or both parents with migraine may be an environmental influence conductive to migraine in the offspring. (Goodell et al, 1954, p. 329)

The procedures used to collect familial data also exert a strong influence on the results. Only Waters (1971a) assessed familial incidence in the general population and contacted relatives directly. Other researchers relied on information from the probands themselves, who were already a selected clinical sample. The influence of patient recall on experimental results was apparant in Goodell's et al (1954) work. Their patients reported a migraine incidence of 15% in immediate siblings versus 37% in the siblings of other family members. This bias may be due to the longer life spans of the more distant relatives (the probands being comparatively young) or the demand characteristics (Orne, 1962) inherent in clinical research. Even tension headache sufferers report a familial incidence of 40%, but no one has argued that tension headaches are inherited (Sacks, 1970). Procedural effects and disputable assumptions make familial incidence studies equivocal as support for the inheritance of migraine.

A more rigorous assessment of the role of heredity in migraine can be made by studying twins (Bakal, 1975; Sacks, 1970). Lennox and Lennox (1960) recorded five cases of monozygous (MZ) twins in which both twins suffered migraine. Refsum (1968) reported migraine concordance rates of from 60% to 100% for MZ twins and from 10% to 40% for dizygotic (DZ)

twins in a review of European literature. The twins were presumably raised together, and selection factors could well have inflated the apparant concordance. Recently Lucas (Note 6) sent a headache questionnaire to all twins over the age of 16 in the volunteer twin register of the London Institute of Psychiatry. He defined common migraine as "unilateral throbbing headache accompanied by nausea or vomiting" (p. c5). Classical migraine required in addition the presence of neurological prodomata. Lucas (Note 6) found some form of migraine in 14% of the MZ twins, 18% of same sex DZ twins, and 10% of opposite sex DZ twins. The concordance rate between MZ twins with migraine was 29% for common and 17% for classical. The concordance between the same sex DZ twins was 24% for common and zero for classical; although only 17 cases were in the latter group. Lucas (Note 6) concluded that "there is a weak genetic factor to migraine" (p. c9). However, the wide variability in the reported concordance between twins for migraine, and the possibility of selection effects (e.g. the Twin Register is disproportionately female and middle class) renders a genetic factor in migraine less likely. Families may simply treat twins differently from singly born siblings.

Family incidence studies and twin research both offer only equivocal support for migraine inheritance. Sacks (1970) expressed a final, more social, criticism of the genetic position in migraine:

> If a patient regards himself as 'doomed' to a lifetime of migraine in view of a sinister family background of the disorder, and his physician takes an equally fatalistic view of the matter, the chances of any therapeutic intervention are much reduced. Lennox and Lennox (1960), usually most reasonable, write 'persons with migraine should think twice before marrying one whose own or whose family history is positive for this disorder.' This statement, in view of the degree of doubt concerning genetic factors, and the overwhelming importance of environmental factors, is little short of monstrous.
>
> (p. 140-141)

Several epidemiologists have searched for possible environmental 'triggers' of migraine attacks. Ogden's (1952) questionnaire included a list of 16 suspected causes. Respondents reported that fatigue triggered 33% of their headaches, eye strain 35% and menstruation 31%. 'Emotions' and 'worry' were suspected by 11% and 14% of headache sufferers respectively (Respondents could select more than one cause . Not all of the headaches were migraine.) Depending upon their symptom patterns, subjects in Selby's and Lance's (1960) migraine series thought that 'emotion' was the most frequent precipitating factor in their headaches (56%-72%), followed by glare (34%-57%), food (12%-43%) and menses (45%-76%). Arthur (1974) examined 35 male and 67 female migraine sufferers and found that 88% were able to identify at least one suspected cause of their migraines. Many patients reported multiple triggers. The triggers most suspected were: emotional stress and upsets, 37%; hormonal factors, 34% (52% of women); alcohol, 34%; sunlight, 30%; foodstuffs, 25%; travelling, 23%; relaxation after stress and on weekends, 17%; glare and shimmering light, 15%; and bending, lifting or stretching, 14%. Dalton (1975) surveyed 1,883 female, volunteer migraine sufferers in England. In response to the question "what brings on an attack?", 28% specified food while 5% specified fasting. "Stress, worry, frustration and tension received frequent mention" (p. 189), but tabulated data was not presented for these variables. Twenty-nine percent of all attacks occured during the first four days of menstruation, a proportion significantly different from chance. In epidemiological surveys then, migraine sufferers have identified a wide variety of suspected precipitants.

Other researchers, however, have suggested that the potency of suspected migraine triggers is over-rated. Waters (1970) found no dif-

ference in visual defects between headache sufferers and headache free individuals. Citing observations by Drews (1957), who found that giving plain glass lenses to patients with normal vision often reduced their headaches, Waters (1970) argued that a placebo effect could account for glasses seeming to help migraine. Blood pressure also did not differ between migraine, headache, and no headache groups (Pfeiffer, Dreisbach, Roby and Glass, 1943; Waters 1971b). In two double blind studies Moffett, Swash and Scott (1972; 1974) showed that two freqently suspected causes of migraine, dietary chocolate and cheeze (tyramine), did not trigger more headaches than placebos. As well Blau (1971) pointed out that the long and variable delay described by migraineurs between ingestion of suspected foodstuff and migraine attack was not at all characteristic of pharmacological effects. Sacks (1970) stated:

> I am not convinced that a migraine can ever be ascribed to a specific food-sensitivity, and I would suspect any association of the two to the establishment of a conditioned-reflex. (p.168)

Research on the acquisition and maintainance of conditioned taste aversions in man and animals supports Sacks' (1970) notion of a conditioned reflex basis for suspected food triggers in migraine. Garcia, Hankins and Rusiniak (1974) and Garb and Stunkard (1974) have described in detail the characteristics of learned taste aversion in animals and humans. Learned taste aversion occurs when a taste is paired with nausea in an extended classical conditioning paradigm. The results in this paradigm violate the usual laws of classical conditioning but are congruent with the observations of migraineurs concerning suspected food triggers. The conditioned stimulus (taste) to unconditioned stimulus (nausea) interval can be up to 12 hours, compared to .5-3 seconds

optimal latency characteristic of classical conditioning. Learning occurs in one trial and is extremely resistant to extinction. In an assessment of 696 people of various ages some food aversions were found to have lasted up to 50 years, although taste aversions generally decline with age (Garb and Stunkard, 1974). In both humans and animals the learned aversion is specific to the taste that has been followed by nausea, and does not generalize to visual or auditory cues. Novel and disliked foods contribute disproportionately to taste aversions in humans (Garb and Stunkard, 1974). Since these characteristics of learned taste aversion fit the descriptions of dietary triggers for migraine, while pharmacological response patterns do not (Blau, 1971), the suspicion by migraine sufferers that their headaches are due to something they ate is probably an instance of adventitious conditioning. The powerful effect of learned taste aversion would also tend to mask environmental events potentially related to the headaches. Migraine sufferers would then be all the less likely to search their environment - physical, social, emotional - for possible headache triggers.

In summary, epidemiological studies offer a variety of information pertinent to the description and occurence of migraine. The clinical features used to diagnose migraine may not form a valid syndrome. Subsets of symptoms may have to be examined in order to find homogenous groups of research subjects. Combining diagnostic criteria with a detailed headache questionnaire and applying MDFA to the questionnaire factors appears to be a potentially fruitful research approach. Studies purporting to show a genetic basis for migraine are inconsistent and inconclusive. The effects of suspected dietary triggers are similarly unreliable. The individual's family and learning history may prove to be

a powerful influence in the development of migraine.

Physiological, Pharmaceutical and Chronic Pain Aspects of Migraine

Biofeedback and relaxation treatments of migraine attempt to change certain physiological events thought to be related to the migraine process. The postulated physiological change may be only tenuously linked to known migraine processes, such as the hand warming training of Sargent et al (1973). Other biofeedback programs may use seemingly physiologically opposite procedures with equally encouraging results (Friar and Beatty, 1976). Before proceeding to the literature on biofeedback and relaxation treatments for migraine, a rudimentary understanding of the physiological events that the treatments are attempting to change should be undertaken.

> It would appear that the symptoms in patients with migraine are related to a disturbance of the central vasomotor centers, the extracranial and intracranial blood vessels, and the microcirculation. This disturbance involves mechanisms in both central and peripheral vasomotor systems and is accompanied by a sterile inflammatory reaction evoked by activity of the nervous system with the eventual production of the clinical symptoms.
>
> (Friedman, 1976, p.4)

Classical migraine, which involves transient visual disturbances (and occassionally other focal neurological abnormalities) has been postulated to involve a period of cerebral vasoconstriction preceding the painful phase (Dukes and Vieth, 1966). Common migraine, the most frequent type, may not be preceded by vasoconstriction. A localized inflammatory edema, containing vasoactive and pain threshold reducing compounds characteristically occurs at the dilated arteries, but " the evidence for the role of vasoactive substances in migraine is fragmentary" (Dalessio, 1974, p. 58). Although a good deal of current migraine research is biochemical (c.f. Friedman, 1972), the technique's popularity is partially due to the nature of the headache,

> ...a nature which does not dispose of too many organic substrates, less still of anatomical evidences. In view of its impalpability the headache problem has been approached through clinical pharmacology and biochemistry. (Sicuteri, 1972, p.159)

(The availablity of research funds from large pharmaceutical companies may also be a factor.) Furthermore, biochemical literature on migraine is still inconclusive and often contradictory. For instance, one popular biochemical theory holds that a drop in blood serotonin levels is a cause of migraine (Sicuteri, Testi and Anselmi, 1961). However, Sjaasted (1975) after an extensive review of serotonin research, concluded;

> Available evidence indicates that serotonin located within (blood) platelets - or lack of it - does not precipitate migraine headache. (Sjaastad, 1975, p.200)

Other biochemical theories (e.g. Bradykinin; Dalessio, 1974) remain similarly inconclusive. Knowledge of the relevant neuro-anatomy and neuro-function is clearly a pre-requisite for understanding the migraine process.

Anatomy.

Blood is supplied to the brain and head by the cartoid system. The common carotid arteries bifurcate approximately half way up each side of the neck into the internal and external carotid systems (Goss, 1959). On each side the external carotids branch to supply blood to the face and scalp. The largest forehead supply is via the superficial temporal artery, which sometimes becomes visibly enlarged at the temple of migraine sufferers (Dalessio, 1972). The internal carotids branch repeatedly to supply blood to the meninges and tendenous covers (pia and dura mater) of the brain, the nasal and sinus cavities, the eye and its cavity. One of the retro-orbital branches passes over the eye and through a small hole (supra-orbital foramen) in the bone above the eye to become the supra-orbital artery, which lies outside the skull behind the eyebrows. Thus, arterial

pain in the forehead region can arise outside the skull, behind the eye, and in the nasal and sinus cavities.

The blood vessels receive innervation from the autonomic nervous system (ANS). The sympathetic component of the ANS exerts a vasoconstrictive control over cutaneous blood flow. (However, the sympathetic innervation to muscular blood vessels is vasodilatory; consequently, during sympathetic arousal blood is generally shifted from the skin to the muscles. Pick, 1970). The parasympathetic component of the ANS exerts a vasodilatory control over cutaneous blood flow. Throughout the body the sympathetic vasoconstrictive control is dominant, and vasodilation results passively from reduced vasoconstrictive tone (Burton, 1965). However, active vasodilation due to increased firing of the parasympathetic innervation modifies, and sometimes exceeds the effect of the vasoconstrictive sympathetic tone. The relative contribution of the two ANS components to vascular control varies in different parts of the body. For example, the vasoconstrictive mechanism predominates in the hand, while active vasodilation occurs in the forearm (Gaskell, 1956). Both vasoconstrictive and vasodilatory control is present in the head (Fox, Goldsmith and Kidd, 1962). The relative effects of the two systems are still under research (e.g. Gonzalez, Onofrio and Kerr, 1975) and are discussed in more detail later under the heading 'Experimental Findings'.

Identification of the central origin of the ANS is somewhat arbitary. Davies (1967) notes;

> It is difficult to find any specific centers in the brain concerned with the efferent side of the autonomic system, but the hypothalamus is the area most strongly implicated. (p.1119)

The hypothalmus receives connections from predominantly "cognitive" areas such as the pre-frontal cerebral cortex, "emotional" areas such as the

amygdaloid body and hippocampus, and from relay centers for sensory afferents, including pain, in the thalamus. Efferent fibers from the hypothalamus innervate the posterior lobe of the pituitary gland (hypophysis) which has been suggested as a mediator in resistance to stress (Selye, 1974). Some hypothalamic fibers return to the anterior and medial thalamic nuclei. Hypothalamic efferents also pass through the periventricular system and the dorsal longitudinal bundle of Schutz to synapse in the brain stem reticular formation. Although the brain stem vasomotor pathways have not yet been clearly mapped, Davies (1967) notes that vasoconstrictor efferents from the brain stem travel in the lateral columns of the spinal cord to synapse there with thoracic preganglionic sympathetic neurons. Other vasomotor fibers terminate in the brain stem by synapsing with parasympathetic pre-ganglionic neurons in the nuclei of the oculomotor, trigeminal, facial and glossopharyngeal cranial nerves. The peripheral compontents of both the sympathetic and parasympathetic efferents consist of two neurons and the intervening synapse. The first neurons arise in the visceral sections of the cranial nerve nuclei in the brain stem, and in the lateral grey columns of the spinal cord. Their axons follow the cranial and spinal nerves that correspond to their centers of origin, and they synapse with the second neurons in the peripheral ganglia. The axons of the second neurons are distributed to the effector organs, including the smooth muscle walls of the arteries.

The pre- and post-synaptic neurons do not join on a one-to-one basis. Post synaptic neurons are more numerous, and up to 17 may be innervated by one pre-synaptic neuron (Davies, 1967). However, one pre-ganglionic fiber usually synapses only with the postganglionic neurons that innervate one type of effector. Autonomic functions in the same ganglion, such

as vasomotor and sudomotor control, can thereby remain dissociated. The synaptic branching widens the area of effect of ANS activity. The disproportion between pre- and post-synaptic neurons though, is less in the parasympathetic system than in the sympathetic. Consequently parasympathetic reactions tend to be more localized, while sympathetic effects are often diffuse.

The peripheral ganglia are more central in the sympathetic system than in the parasympathetic. The cell bodies of the sympathetic post-synaptic neurons are generally in ganglia adjacent to either side of the spinal cord. These ganglia are fused, forming the paravertebral sympathetic columns. The preganglionic sympathetic efferent fibers emerge from the thoracic portion of the spine, and travel up and down the paravertebral columns to their synapse. The parasympathetic ganglia are usually close to the organ or blood vessel which they innervate and sometimes are diffused throughout the wall of the viscera itself. The preganglionic parasympathetic efferents emerge only with the cranial nerves, and the sacral spinal nerves.

The sympathetic preganglionic vasoconstrictive fibers for the head ascend from the first five (sometimes four) thoracic segments to the superior cervical ganglia at the top of the paravertebral columns. There the fibers synapse, and the post-ganglionic fibers form a neural network around the carotid artery called the carotid plexus. This plexus follows the ramifications of the common, internal and external carotid systems and supplies sympathetic innervation to the head. Fibers from the carotid plexus terminate on blood vessels and the sweat, lacrimal and salivary glands (Clark, 1975). In the internal carotid system the main vasoconstrictive effect occurs at the proximal portions of the main branches,

and along the meningeal arteries (Knight (1968). Outside of the skull the sympathetic fibers influence the external carotid and the small scalp arteries.

The parasympathetic vasodilatory innervation to the head originates chiefly in the facial nerve nuclei and to a lesser extent in the trigeminal nuclei in the brain stem. The facial nerve has two nuclei, a large motor root and a smaller sensory-autonomic root (Goss, 1959). The smaller nerve is called the nervus intermedius or the nerve of Wrisberg. It contains secretomotor fibers arising in the Nucleus Salivatoius Superior to supply the lacrimal, nasal and oral glands, and vasomotor fibers arising with the facial nucleus to supply vasodilatory fibers to the face. The preganglionic fibers leave the brain stem with the facial nerve and, branching from the geniculate ganglion, form the greater superficial petrosal (GSP) and the external superficial petrosal nerves. The GSP is joined by the large deep petrosal to become the Vidian nerve (nerve of the pterygoid canal) which enters the sphenopalatine (Pterygopalantine) ganglion (Goss, 1959). As well, the GSP supplies fibers to the trigeminal ganglion (Gonzalez et al, 1975). The parasympathetic efferents synapse in the sphenopalatine ganglion or near their end-organ (Wegner, 1968) and the post-ganglionic fibers supply secretomotor and vasodilatory fibers to the nasal and sinus mucosa. The vasomotor post-synaptic fibers from the trigeminal ganglion are distributed to the face with each of the three divisions of the trigeminal nerve. As well, "a moderate number of vasodilator fibers also appear to leave the brain stem directly with the trigeminal nerve" (Gonzalez et al, 1975).

One difficulty in elucidating the activity of the vasodilatory supply to the face and head is a physiological difference between man and

experimental animals; 24% of the facial nerve fibers are autonomic in man, while only 7% are in the dog, and 2% in the cat (Van Buskirk, 1945). These marked species differences suggest that neurovascular effects demonstrated in the dog and cat may well be more marked in humans.

While the position of their ganglia and their antagonistic effects are used to differentiate the sympathetic and parasympathetic systems, another important difference is the neurotransmitter liberated at the terminals of the post-ganglionic neurons. The transmission of sympathetic impulses to the effector cell is due to the liberation of noradrenaline (norepinephrine) at the terminal. The post-ganglionic parasympathetic fibers liberate acetylcholine. Thus sympathetic nerves are termed adrenergic, while parasympathetic nerves are called cholinergic. (The one exception is the cholinergic sympathetic supply to sweat glands). The synapses between pre- and post-ganglionic neurons are cholinergic.

The difference in neurotransmitter between sympathetic (adrenergic) and parasympathetic (cholinergic) innervation to the blood vessels has permitted elegant histochemical examination of the neural network surrounding the cerebral arteries and arterioles. When examined by these techniques, the number of adrenergic and cholinergic fibers were found to be roughly equal in the larger pial arterioles, with a greater density on the anterior pial circulation than on the posterior (Edvinson, 1975). Vascular reactivity was proportional to the density of innervation. Adrenergic and cholinergic fibers ran intermingled along the pial vessels. The vascular neuroreceptor activity proved to be fairly complex. Only alpha-adrenergic receptors were vasoconstrictive, while beta-adrenergic receptors were dilatory. The cholinergic receptors were dilatory at low concentrations of cholinergic agonist, but constrictive at high concen-

trations. Dilatory histamine H_2 - receptors were also present. Electrical stimulation of the perivascular sympathetic nerves released enough noradrenaline to cause vasoconstriction. The cholinergic and adrenergic nerve endings were in close apposition, enabling cholinergic axons to inhibit the vasoconstrictive activity of the alpa-adrenergic axons, thereby, promoting vasodilation (Edvinson, 1975). Axon endings, identified by electron microscopy, reach even the capillary beds deep in the brain (Rennels & Nelson, 1975). While these sophisticated studies of cerebrovascular innervation illustrate the complexity of neural activity, they also emphasize the ability of the nervous system to influence the blood flow.

The blood vessels receiving autonomic innervation consist of 3 coats; inner - tunica intima, middle - tunica media, and outer - tunica adventitia (Davies, 1967). The tunica media consists mainly of smooth muscle cells, arranged helically, or, when the artery is small, transversely. Membrane contacts (demosomes) between adjacent muscle cells are frequent, providing mechanical strength and possibly conducting excitation. The effector neurons are unmyelineated. They come into near apposition with the smooth muscle cells of the tunica media and follow grooves of various depths at the muscle cell surfaces. The autonomic fibers do not exhibit any structural specializations at their endings, nor do they show a polarization of vesicles towards the apposed plasma membrane, as is found in striped muscle innervation. Rather, the ANS neurons exhibit a series of bead-like enlargements containing clusters of vesicles and mitochondria. The synaptic contacts are often elongated. One neuron is apt to synapse with several smooth muscle cells, and each cell may receive terminals from more than one neuron. However, the total number of enlar-

gements is always less than the total number of smooth muscle cells in any particular section of artery, suggesting that some propogation of excitation occurs through the muscle cells themselves. Davies (1967) notes that each enlargement may function as an individual nerve ending.

The vesicles in the terminal enlargements differ in sympathetic and parasympathetic neurons. Adrenergic sympathetic neurons contain large granular vesicles from 800 to 1,000 Å in diameter, with a dense granule at the center. Cholinergic parasympathetic neurons contain small clear 500 Å diameter vesicles. The average distance between the terminal enlargements and the apposed muscle cells in small arteries ranges from 600 to 4,000 Å, with a mean of about 1,000 Å. This distance is substantially larger than the gap at a neuromuscular junction in striped muscle, and also somewhat larger than the corresponding gap at the other ANS effectors such as glands. Transmitter substance is released into the tissue spaces, and it moves to the receptor sites on the smooth muscle by diffusion. The distance between the terminals and the effectors may be related to the spontaneous activity and speed of reaction of the smooth muscle cells.

The afferent component of the neurological process in migraine is somewhat simpler than the efferent system. The pain carrying afferent fibers accompany the efferent fibers, and terminate in the inner and outer coats of the arteries. The terminals have been described as "knovs, loops rings and tendril-like endings" (Davies, 1967). All the terminals are unmyelinated but the afferent axons are both unmyelinated and myelinated to various thicknesses. The terminals are probably excited by chemicals released by the adjacent tissue during noxious stimulation (Clark, 1975).

Excessive contraction of smooth muscle can produce visceral pain (Davies, 1967). The neurons are pseudo-unipolar cells; their peripheral processes (dendrites) travel with the ANS fiber network on the arteries, while their central processes (axons) follow the corresponding autonomic efferents back to the central nervous system (CNS). The afferents pass through the autonomic ganglia without synapsing. Most pain fibers from the face, sinuses and mucosa follow the trigeminal nerve to the brain stem.

> "Upon entering the brain stem in the pontine region, these fibers form a descending tract, the spinal tract of V. Terminals of the spinal tract of the trigeminal nerve make synapses in an adjacent nucleus, the nucleus of the spinal tract of V. Axons originating in the nucleus of the spinal tract of V cross to the opposite side and ascend as the anterior (ventral) trigeminothalamic tract to the ventral posteromedial nucleus (VPM) of the thalamus. This pathway also sends fibers to synapse in the reticular formation and probably to the other thalamic nuclei (aside from VPM) that receive projections from the anterolateral system."
>
> (Clark,1975:p.26)

The pain signals carried by unmyelinated fibers are experienced and processed in the CNS somewhat differently than those carried by myelinated fibers. "Slow" pain, felt as a burning or dull, diffuse aching sensation, travels on unmyelinated C fibers. "Fast" pain, transmitted via lightly myelinated A-delta fibers, is sharp, pricking, and localized (Clark, 1975). The slow pain fibers synapse in the brain stem reticular formation, which relays the signals to hypothalamic and thalamic nuclei involved in emotional behavior. The slow pain pathways are not arranged to provide discrete spatial or temporal information. The fast pain fibers project without intervening synapses to the thalamus, where they are somatotopically arranged, thereby enabling such pain to be localized. The conscious experience of pain occurs at the thalamus.

> "In man, the posterior and intralaminar nuclei must be destroyed in order to relieve intractable pain. Destruction of the cerebral cortical projection areas does not eliminate pain."
>
> (Clark, 1975; p.27)

The experience of pain can also be influenced by cortical areas involved in cognition, sensation and emotion. Descending pathways from the cerebral cortex and other supraspinal centers synapsing in the reticular formation and posterior horn directly modify the rate of firing of the pain carrying neurons. Some somatic sensory fibers also serve a similar pain-modification function. These "gate-control" mechanisms (Melzack and Wall, 1965) provide the basis for emotional, sensory and cognitive influence in the pain experience.

To summarize, the neurogenic substrate of migraine forms a full circle. Beginning possibly as high as the cortex, vasomotor pathways leave the hypothalamus and pass through vascular control centers in the brain stem to supply vasodilatory fibers to the face via parasympathetic components of cranial nerves, and vasoconstrictive fibers via the paravertebral sympathetic trunks and carotid plexuses. Pain sensitive fibers arise at the arteries and course back to the CNS along the efferent pathways, to synapse in the brain stem and thalamus, centers which are also under cortical influence. Dysfunction in either the hypothalamus or the brain stem could cause a disruption of the sympathetic-parasympathetic balance, potentially leading to the migraine attack. Research examining functional autonomic control of the cranial circulation offers further support for the important role of the nervous system in migraine.

Experimental Findings

Chorobsky and Penfield (1932) were the first to establish experimentally the functioning of vasodilatory fibers to the cerebral blood vessels. They inserted a window in the skull's of monkeys, and observed the effect on arterioles in the pia mater of electrical stimulation of the facial and GSP nerves. They found that:

> ... stimulation of the facial nerve produces dilation of certain pial arteries. Section of the greater superficial petrosal nerve puts an end to this reaction. Stimulation of the peripheral portion of the divided GSP produces energetic dilation of the same artery..... Response of the facial muscles to a stimulation of the seventh nerve is prompt and ceases instantly following cessation of stimulation, while the associated dilation lags behind the onset of stimulation and continues for a short time after its cessation.....
>
> (p. 1275)

Chorobsky and Penfield (1932) concluded that the facial nerve, via the GSP provides cerebral vasodilatory fibers. They considered these fibers parasympathetic, on the grounds that their action was antagonistic to known sympathetic vasoconstrictive effects.

Cobb and Finesinger (1932) independently corroborated and extended the analysis of facial vasodilator fibers. In a total of 147 stimulations of the facial nerve at its exit from the medulla they observed dilatation of pial arteries during 122 stimulations and no change during the remainder. The average dilation was 16%, with a range of 4% to 60%. The larger dilations occured later in the experiment after improvements in the anesthetic procedure.

Cobb and Finesinger (1932) also demonstrated cerebral vasodilation mediated by the facial nerve during afferent stimulation of the vagus nerve. Stimulation of the vagus when both the facial and vagus nerves

were intact resulted in 33 pial dilations in 41 trials. Identical stimulation with the facial nerve severed caused no change in pial artery size. If the vagus was cut near the medulla, and the facial nerve left intact, peripheral stimulation of the vagus at the cut did not change pial artery size, while central stimulation of the vagal stump continued to cause pial artery dilation. Apparantly interconnections exist between the facial and vagal nuclei, possibly in the medulla.

These interconnections may permit bilateral pial dilation from the brain stem. During stimulation of the central vagal stump, 21 dilations occured in the contralateral pial artery in 29 trials. The amount of contralateral dilation varied from 4% to 36% with a mean of 14%. By comparison, cervical sympathetic stimulation caused ipsilateral pial vasoconstriction but no change in the contralateral pial arteries.

Chorobsky and Penfield (1932) also offered an interesting phylogenetic comment on the relationship of the facial innervation to visceral responsivity in emotional reactions.

> In the lower vertebrates the facial nerve innervates the branchial musculature and thus has to do with respiratory movements. The gradual adaptation of these muscles to movements of facial expression in mammals has not altered their innervation. From an anatomic point of view, the central nuclei of the facial nerve proper and of the nervous intermedius of Wrisberg are both situated in the visceral area of the medulla oblongata along with the nucleus of the vagus. Consequently, anatomists generally (5 references) have considered the facial nerve to be visceral.
>
> (p.1285)

The vasomotor control of cutaneous circulation in the head of the cat was examined by Gonzalez et al (1975). They stimulated each division of the trigeminal nerve and observed a consistent increase in skin temperature, indicating vasodilation at the cutaneous area innervated by that division. Stimulation of the ipsilateral sympathetic chain resulted

in vasoconstriction at the face. The vasodilatory response was usually larger than the vasoconstrictive response. Gonzalez et al (1975) also found vasodilation during stimulation of highly specific centers in the facial nerve nucleus, and in the spinal nucleus of the trigeminal (subnucleus interpolaris). The results of their brain stem stimulations suggested that the vasodilator nerves supplied to the face may be topically organized.

> With regard to the central origin of the vasodilator fibers to the face, we still have only very limited data. The centers or fiber systems concerned with this phenomenon, however, are found in the two nuclei, facial and trigeminal, most directly concerned with the innervation of the face.
>
> It is interesting that already at this central level evidence suggests that the vasodilator supply to the face is topically organized, since responses disappear in the distribution of one division and appear in another with slight advances or withdrawals of the stimulating electrode. Thus, while the divisional organization of the vasodilator fibers to the face may be due entirely to the pattern of distribution of the GSP to the trigeminal division peripherally, a central topical organization should not be excluded. (p. 701)

Should the vasodilatory innervation to the face prove to be topically well organized, such a finding may account for the localization of migrainous pain.

The relative importance of the vasodilatory and vasoconstrictive system to the face in humans was evaluated by Fox, Goldsmith and Kidd (1962). Their technique consisted of warming the body in a controlled temperature bath, blocking various nerves on one side of the face with a 2% Xyolocaine injection, and comparing the relative heat flow from the face on the blocked and unblocked sides. During indirect heating the blood supply to the skin dilates to increase heat loss for the maintainence of homeostasis. An increase in heat flow on the blocked side would indicate a dominant vasoconstrictive innervation, with the dilation

occuring passively due to the absence of normal constrictor tonus. A dilation on the unblocked side, however, while the blocked side remained relatively unchanged, would indicate that active vasodilation is dominant. Blood flow on the scalp and forehead was assessed by blocking the (sympathetic) supraorbital nerve, and measuring heat flow 15 cm and 4 cm above the supra-orbital ridge, At the scalp and forehead sites Fox et al (1962) found,

> blocking the cutaneous nerve supply resulted in only a small increase in heat flow due to the release of vasoconstrictor tone, but during indirect heating the response from the nerve blocked side fell far short of that from the control side, indicating the presence of a large component of active vasodilation.
>
> (p.304)

The demonstrations of vasodilatory fibers in the GSP nerve (Chorobsky and Penfield, 1932; Cobb and Finesinger, 1932), of active vasodilatory dominance in the forehead and scalp (Fox et al, 1962) and of vasodilatory centers in the brain stem (Gonzalez et al, 1975) all provide direct experimental evidence of a possible neural basis for parasympathetic mediation of the painful phase of migraine. Numerous other more tangential studies, varying in rigour and pertinence, also bear upon the role of the nervous system in migraine.

Meyer, Yoshida and Sakamoto (1967) partially replicated the earlier GSP research in a study mainly concerned with assessing sympathetic control of the carotid blood vessels. Stimulation of the intact GSP in 7 monkeys was followed by an increase in internal carotid flow and an increase in blood pressure. Meyer et al (1967) argued that the internal carotid flow was a passive effect due to the increased blood pressure. They did not suggest a mechanism by which stimulation of a branch of the facial nerve could cause an increase in blood pressure. Conceivably a

spread of stimulation of the brain stem might be involved.

Meyer's et al 91967) observations on the effect of sympathetic flow was much less equivocal. Stimulation of the cervical sympathetic ganglia resulted in a 30% decrease in the internal carotid blood flow and a 68% decrease in the external carotid flow. Blood pressure increased during these stimulations. Chemical blockade or surgical section of the superior cervical ganglia did not increase internal carotid flow. Meyer's et al (1967) findings were similar to those of Lance (1973) who noted that in man "stellate ganglion blockade, unilateral or bilateral, does not augment cerebral blood flow" (p. 118). Meyer's et al (1967) work corroborates the known vasoconstrictive effect of the sympathetic nerves on the internal and external carotid system by a novel technique (electromagnetic flowmetry). The removal of sympathetic tone did not result in a passive dilation of the innervated vessels. Dilation of the internal carotid was observed during stimulation of the GSP, in conjunction with a concomitant rise in blood pressure.

Additional support for vasodilatory dominance at the forehead was presented by Hertzman and Roth (1942), who thought that vasoconstrictor responses were essentially absent from the forehead. Hertzman and Roth (1942) based their conclusion on 4 observations: 1) vasoconstrictive reflexes of the hand, elicited by startle, awakening and deep breath were absent in the forehead, 2) spontaneous rhythmic vasomotor constrictions in the fingers were absent in the forehead, 3) the vascular reactions of the forehead to cold were like those of a sympathetomized finger, and particularly 4) occassionally a vasodilative response at the head accompanied a particularly powerful vasoconstrictive response at the hand.

While extracranial arterial dilation during the painful phase of migraine is well established (e.g. Tunis & Wolff, 1953) the concurrent action of the forehead skin capillaries is less clear. Lance & Anthony (1971) report that forehead skin temperature was cooler by one degree centigrade ($1^{o}C$) on the painful side during an attack in 8 out of 12 patients. "The skin became cooler as the headache intensified" (p. 241). Between attacks forehead temperature was symmetrical in 6 out of 10 patients, with the habitually painful side being warmer in 3 cases and cooler in one. By comparison, forehead temperature was symmetrical in 14 of 15 headache free control subjects, and differed by less than $1^{o}C$ in the remaining control. However, an attempt at independent replication of Lance's & Anthony's (1971) findings failed to find any specific forehead skin temperature patterns in migraine, although a cool pattern at the site of the pain persisted in cluster headache patients (Friedman, Wood, Rowan & Frazier, 1973). Possibly skin capillaries constrict, reducing surface blood flow, in at least some cases of migraine. Such a finding would be consistent with the frequent observations of facial pallor during migraine (Best & Taylor, 1966, p. 841; Lance, 1974; Lance & Anthony, 1971; Rowbotham, 1942).

Contrast the data suggesting reduced skin surface blood flow during migraine, however, with the observation that overall fronto-temporal cutanous flow increases. Elkind, Friedman & Grossman (1964), using an isotopic sodium technique, found increased forehead blood flow during migraine, particularly on the more painful side. Forearm blood flow did not change. Vasoconstrictive chemicals usually relieved the headaches. Possibly the superficial skin capillaries constrict during migraine, while capillaries in deeper tissue dilate (Lance, 1973, p.115).

Stimulation of the sympathetic trunks can cause constriction of skin capillaries (Crawford, 1930; Crosby, Humphrey & Lauer, 1962). If the sympathetic innervation is intact, differential control of capillaries and arterioles in muscle is possible (Renkin & Rosell, 1962). These differential vascular effects are not observed with stimulation of the severed sympathetic trunks, suggesting that more centrally mediated vasoconstrictor reflexes are capable of exerting separate control of arteries and capillaries (Renkin & Rosell, 1962). Lance (1973) mentions sympathetic capillary dilator fibers to the scalp and forehead vessels, but other authorities make no mention of such a neural system except for the cholinergic sympathetic fibers to the facial muscles (Uvnas, 1954). Milor (1974) noted that parasympathetic vasodilator fibers have been suspected in various vascular beds, but "the evidence adduced thus far is not convincing" (p. 951). Neural control of fine differences in capillary and artery behaviour seems possible.

Intracerebral vasoconstriction during the prodome and cerebral vasodilation during the painful phase of migraine is increasingly well documented. Mathew, Hrastnik & Meyer (1976) used a radioactive regional cerebral blood flow (rCBF) technique to assess migrainous alterations in cerebral blood flow. They found:

> The rCBF in migraine varied with the clinical phase. During the prodrome phase the blood flow was reduced; whereas during the headache phase, a striking cortical hyperperfusion was noted. Patients who continued to have neurological symptoms of the prodrome in the headache phase showed a mixed pattern of reduced and increased blood flow
>
> The rCBF was found normal in the headache phase in patients with muscle contraction and psychogenic headache. (p252)

Cerebral blood flow increase 19% while flow through grey matter increased 43% during the headache phase, suggesting the dilation of both arteries and arterioles.

Other researchers have made similar observations and conclusions. Simard and Paulson (1973) found a 48% reduction in cerebral blood flow during prolonged and extensive prodromata that included aphasia and blurred vision in one patient. Skinhoj and Paulson (1969) observed a 51% decrease in cerebral blood flow in a patient with a severe prodrome very similar to that of Simard's and Paulson's (1973) case. Skinhoj and Paulson (1969) were able to perform an angiogram immediately, and found that no arterial spasm was present.

> A normal angiogram together with a reduced cerebral blood flow indicates that the cerebral vascular resistance is increased at the level of the arterioles... (p.570)

During the painful phase in a second migraine patient Skinhoj and Paulson (1969) noted a 47% increase above normal values of intracerebral blood flow. O'Brien (1967, 1971) reported a 20% reduction in "cerebral cortex perfusion" rate during the prodromal phase in 7 patients. (O'Brien's, 1967 technique confounded extracranial and intracranial blood flow.) The cerebral blood flow studies clearly establish that blood flow through the brain is reduced during prodromal symptoms and is increased during the painful phase of migraine (Skinhoj, 1973).

The mechanism causing these changes, however, remains unclear. Mathew et al (1976) believed that the cerebral dilation during pain still lacked a definite explanation. Similarly, Skinhoj and Paulson (1969) thought that the prodromal vasoconstriction was not due to metabolic changes in the brain but was probably "secondary to some other unknown mechanism" (p.570). Skinhoj (1973) argued that the painful dilation was a reactive hyperemia to the ischemic phase, mediated by a metabolic acidosis. Skinhoj's (1973) demonstration of cerebrospinal fluid lactacidosis following cerebral ischemia in migrainous patients supported his inter-

pretation. On the other hand, the absence of clinical signs of cerebral ischemia in common migraine sufferers renders Skinhoj's (1973) hypothesis less tenable (Mathew et al, 1976). None of the researchers using the cerebral blood flow techniques considered a possible neurogenesis of the cerebral vasoconstriction and dilatation. The role of the nervous system in migraine is neither confirmed nor disconfirmed by their studies.

Two processes sometimes related to migraine, anxiety and sleep, have been touched upon by cerebral blood flow research. Sokoloff, Mangold, Wescher, Kennedy & Kety (1955) assessed the effect of mental arithmetic levels. (Blood pressure and heart rate increased by small but statistically significant amounts.) However, the cerebral blood flow of the 13 healthy male undergraduates in the study proved to be higher even during rest, than that of a comparable sample which was not facing the math quiz. Sokoloff et al (1955) speculated that solving mathematics problems in front of their professors generated anxiety in the students in the mental arithmetic study, which led to higher cerebral blood flow. Several comments by subjects supported this notion. On the other hand Scheinberg & Stead (1949) found no difference in cerebral blood flow between "apprehensive" and non-apprehensive subjects during a study designed simply to establish technical norms. The apprehensive group was identified by "clinical appearance" and elevated heart rate and systolic blood pressure. Perhaps mathematics is more frightening to students than scientific paraphenalia.

Mangold, Sokoloff, Conner, Kleinerman, Therman & Kety (1955) investigated the effect of sleep and fatigue (sleep deprivation) on cerebral blood flow in healthy young men. Fatigue caused a moderate increase in cerebral blood flow that approached statistical significance. Sleep ele-

vated cerebral blood flow significantly, with a corresponding decrease in cerebral vascular resistance and mean arterial blood pressure. Sleep, fatigue and anxiety have all been mentioned by migraine sufferers as suspected migraine triggers (Arthur, 1974). Cerebral blood flow increases during sleep and fatigue and may increase with anxiety. Cerebral blood flow dilation clearly accompanies migraine attacks, although the etiology of the migrainous blood flow changes remains obscure.

Outside the skull the possibility of extracranial dilation (and hand vasoconstriction) during emotional arousal has been suggested by two relatively simple early studies. Helson & Quantius (1934) measured infra-orbital skin temperature reactions to a variety of noxious stimuli. Temperature rose during both the experimental and control periods, but the increase during the experimental periods was significantly greater. Finger tip temperature tended to decrease in a series of preliminary observations.

Mittleman & Wolff (1939) observed a drop in finger skin temperature during 203 stressful interviews with 47 subjects. Finger temperature decreases of 5°C were typical, with some drops as large as 13°C.

> Subjects who responded to emotional stress with major drops in skin temperature often had minor fluctuations during the control experiments. These variations which took place during supposed "relaxation and contentment" could sometimes be correlated with swings in the affective state of the subject occuring spontaneously during the control period... (p. 273)

> Here as in other experiments, a slight increase in forehead and cheek temperature was observed to accompany major drops in the skin temperature of the fingers. (p. 291)

Mittleman & Wolff (1939) did not analyze their data statistically but presented illustrative cases. The finger temperature of their fifth

subject dropped from 33°C to 21°C while forehead temperature rose from 34.2°C to 35.5°C during the stressful interview. By the end of a 30 minute relaxation period following the interview forehead temperature had decreased to 34.9°C, and hand temperature had returned to mean baseline. These observations suggest that during stress extracranial vasodilation may occur concommitantly with sympathetically mediated hand vasoconstriction.

The most direct evidence of reciprocal change of the head and hand temperature was a biofeedback study by Sovak, Fronek, Helland & Doyle (1976). During passive dilatation of the hand induced by a current of hot air, the diameter of the finger, temporal and supra-orbital arteries all increased. When subjects were trained to voluntarily increase their finger temperature, however, the finger arteries dilated while the temporal and supra-orbital arteries constricted. Even though these findings are based on only 3 normal subjects, they offer the most direct available evidence of reciprocal vasomotor action at the head and hand. Furthermore, these data suggest a common vasomotor action of both the internal and external carotid systems, represented by the supra-orbital and temporal arteries respectively.

During volitional finger dilatation (hand warming) mean blood pressure decreased slightly (-3 millimeters of mercury) and heart rate decreased moderately (-7 beats per minute; Sovak et al, 1976). Neither blood pressure nor heart rate changed during passive hand heating. Since blood pressure decreased when the supra-orbital artery dilated under volitional hand warming, the dilation of the internal carotid system could not have been a passive reaction to blood pressure increase, as argued by Meyer et al (167).

Even more striking is Sovak's et al (1976) observation that the reciprocal relationship of forehead vasoconstriction to hand vasodilation occured only with "volitional" hand warming. The finger and forehead arteries dilated together during passive heating of the hands. The reciprocal relationship between head and hand arterial reactions may appear only when higher CNS functions are involved. Mittleman & Wolff (1939) noted that the emotions which evoked forehead heating and hand cooling (i.e. the opposite but still equivalently reciprocal reaction observed by Sovak et al, 1976, with relaxation) were "always complex", and involved in different subjects to differing degrees,

> ...tension, anxiety, guilt, feelings of being pushed and of being thwarted, embarrassment, irritation, anger, rebellion, feelings of inadequacy, and of being criticized or disapproved of, insecurity, humiliation, grief, feelings of helplessness, depression, despair and pleasureable excitement. Hence, there was no specificity in the quality of the predominant emotion that was accompanied by a fall in skin temperature of the extremities. (p. 291)

Taken together Mittleman's & Wolff's (1939) and Sovak's et al (1976) findings suggest that arousal could increase blood flow to the head while decreasing it to the hand, and that relaxation augmented by hand warming training might reverse these arterial effects.

Work by Price & Turskey (1976) also bears upon the relationship between head and hand blood flow changes. Forty pairs of migraine and non-migrainous subjects received finger blood volume feedback, false feedback, a relaxation tape or a neurtral tape in a thirty-two minute session. Total blood volume at the finger and head were analyzed. The main effects of groups (migrainous versus non-migrainous) and treatments did not attain statistical significance. When each session was examined as 16 two-minute periods, the treatments by periods interaction was significant for finger volume. The neutral tape (on growing avacado plants)

decreased finger volume significantly, while feedback, false feedback, and relaxation tended to increase finger volume slightly. The group by period interaction was also significant. Finger volume increased during the session in migraine-free subjects but stayed the same or tended to decrease in migraineurs. Cephalic (head) blood volume increased for normal subjects but decreased for the migraine sufferers. Price and Tursky (1976) also calculated the mean finger and forehead blood volume for the first and second half of each session, and subtracted them to get a within session change score. The correlation between within session finger and forehead blood volume change was .666 for migraineurs and .700 for normals.

Price's and Tursky's (1976) findings have several possible implications. As they themselves noted, the high correlations between head and hand blood volume changes argues against the notion of reciprocal vascular activity. On the other hand Sovak et al (1976) found reciprocal blood flow changes only during volitional control of hand dilation. Passive hand warming was accompanied by dilation of the temporal artery. Since Price's and Tursky's (1976) subjects did not master volitional hand blood volume control, the positive correlation between head and hand blood flow change may reflect a diffuse passive dilatation due to relaxation in the normal volunteers. Conversely, the observation that the migrainous subjects' head and hand blood flows concomitantly decreased during the experimental session (yielding a positive correlation) may be "an example of how migraine sufferers respond to a demanding situation" (Price and Tursky, 1976, p.215). Rather than contradicting Sovak's et al (1976) findings then, Price's and Tursky's (1976) study may be confirmatory.

Comparison of Sovak's et al (1976) and Price's and Tursky's (1976) studies is rendered even more difficult by small but critical methodological

differences. Sovak et al (1976) recorded pulse volumes (and blood flow velocities), while Price and Tursky (1976) used total blood volume. Both researchers used the photoplythesmographic technique. With this technique pulse volume and blood volume reflect two vascular characteristics, the phasic systolic expansion of the artery, and the tonic blood level in the tissue, respectively (Brown, 1972). Since their feedback meter display followed the pulse peaks, Sovak's et al (1976) technique indicated the sum of pulse volume and blood volume. Price and Tursky (1976) used blood volume feedback, which indicated the net change in tonic finger volume with the pulse wave removed. While pulse volume and blood volume typically covary, at least in the finger, they are not measuring the same phenomenon. This difference may affect the comparability of photoplethysmographic biofeedback studies in ways that have not yet been clarified.

If migraine is neurally mediated, as circumstantial evidence suggests, then surgical section of the offending nerve fibers should prevent migraine. Surgical treatment of migraine has been attempted, but unfortunately both the therapeutic findings and their theoretical implications have been inconclusive. Dalessio (1972) explained the basic therapeutic and research problem involved in the ligation of nerves and blood vessels for migraine relief (ligation of blood vessels also severs their enveloping neural network):

> The rich anastomotic supply of small arteries, the overlapping, and the regeneration of afferent nerve supply amply account for the limitations of the use of this method of treatment. (p.606)

As well Lance (1973) noted:

> Many early reports on the effect of surgical procedures in migraine are invalidated by doubtful diagnosis and inadequate follow-up.... The only operation which is of predictable benefit is that of section of trigeminal

> pathways, which gives relief from pain at the expense of permanent facial analgesia. (p.119-120)

Surgical resection of cervical sympathetic pathways failed to benefit migraine sufferers reliably (Rowbotham, 1946). Since cervical sympathetic blockade has little effect on temporal artery size (Lance, 1973), the finding that sympathetic resection is ineffective against migraine is not surprising. The possibility of a two-stage migraine process involving sympathetically mediated vasoconstriction followed by reactive vasodilation (caused by parasympathetic activity or local vasochemical reactions) lends some attraction to the sympathetic resection treatment. Dalessio (1972) remarked:

> While reasoning would suggest that the vascular changes in headache may somehow implicate the sympathetic system, it does not follow that cervical sympathectomy will alter the tone or interrupt afferent pain fibers of the vessels concerned in vascular headache, at least in sufficient degree to have value. (p.606-607)

The data shows that severing the sympathetic innervation to the head is ineffective in reducing migraine.

Since parasympathetic vasodilator fibers travel to the intracerebral and scalp vessels via the nervus intermedius and GSP (Chorobski and Penfield, 1932; Gonzalez et al, 1975) surgical resection of those nerves would test their role in migraine. Gardner, Stowell and Dutlinger (1947) severed the GSP in 9 patients with migraine. Relief was complete or nearly complete in all but one case. Clear benefit was also obtained for a specific variant of migraine called cluster migraine, Sluder's syndrome or atypical facial neuralgia by resection of the GSP (Gardner et al, 1947) or by resection of the nervus intermedius (Sachs, 1968,1969). Gardner's et al (1947) surgery for migraine, however, confounded GSP resection and "the incidental ligation of the superficial temporal arteries (which) may have been a

factor in the relief in these cases" (p.111). None-the-less, Gardner et al (1947) thought that the best interpretation of their work was:

> ...the operation interrupts abnormal parasympathetic discharges which are causing dilatation of the cerebral, meningeal, and nasal mucosal blood vessels, the resulting pain of this dilatation being transmitted over the filaments of the 5th cranial nerve. (p.107)

Although referring only to cluster migraine, Sachs (1968) also offered a parasympathetic interpretation of the pain and its relief by neurosurgery:

> The predominantly parasympathetic nature of the attacks probably accounts for the variation in symptoms from patient to patient, the diffuseness of the pain as compared to true trigeminal neuralgia, the difficulty of diagnosis, and the tendency to classify these patients as neurotic. (p.54)

Gardner's et al (1947) and Sach's (1968,1969) studies are apparantly the only reports of parasympathetic surgery for migraine. Their results support the possibility of parasympathetic paroxisms in at least some types of migraine, but are far from conclusive. The surgical migraine literature so far does not supply definitive data on the role of the nervous system in migraine.

Brief mention might also be made of electroencephalographic (EEG) studies of migraine. While no single eeg pattern is diagnostic of migraine (Sacks, 1970; Townsend, 1967), more migraine sufferers exhibit EEG abnormalities than comparable groups. Hockaday and Whitty (1969) found EEG abnormalities in 61% of 560 migraine sufferers, compared to the usual 20% in the general population. Smyth and Winter (1964) thought that 43% of their 202 migraine records were abnormal. They further analyzed the type of abnormal pattern and could predict patients who would not respond to ergotamine on the basis of delta waves (<4 Hertz). The H response to flicker was found in 95% of the migraine sufferers, but only in 14% of

normals. Other studies, varying in both their definitions of migraine and eeg "abnormality", have formed the common view that the resting EEG tends to be abnormal in migraine sufferers (Hockaday & Whitty, 1969; Selby & Lance, 1960), although some researchers disagree, claiming that the EEG is normal in migraine (e.g. Boudin, Pepin, Barbizet & Masson, 1962). Whitehouse, Pappas, Escala & Livingston (1967) argue that the eeg findings, taken with the clinical symptoms, suggest:

> a primary autonomic disturbance in migraine with vascular and humoral factors as secondary manifestations (p. 27)

Sacks (1970), after reviewing the eeg literature on migraine, concluded:

> that some form of electrical disturbance accompanies the generation of a migraine can hardly be doubted, but the nature of this disturbance is still quite speculative. (p 206)

Another procedure that has attracted migraine researchers' attention is the assessment of vascular reflex abnormalities. Kerslake and Cooper (1950) showed that vasodilatation at the hand in response to heating of the body elsewhere is a neural reflex from heat-sensitive skin receptors. A normal reflex indicates, therefore, that certain autonomic pathways are intact. Since lesions in the brain stem and cervical cord disrupt the thermal reflex when the peripheral pathways are intact (Appenzeller & Schnieden, 1963), a central pathway extending to at least the lower brain-stem is involved. The hand is usually the site selected for examining the thermal reflex because most of the blood flow is through sympathetically controlled vessels of the skin (Burton, 1965). The thermal reflex can be used, then, to investigate abnormalities of neural vasomotor control.

Appenzeller (1969; Appenzeller, Davison & Marshall, 1963) examined

finger and scalp blood flow in 10 migraine sufferers between attacks. During 40 seconds' heating of the chest, finger dilatation occured in only 2 of the 10 migraine sufferers but in all of the control subjects. After 10 minutes of heating dilatation occured at the forehead in 2 migraine subjects. Application of iced water to the feet or doing mental arithmetic caused hand blood flow to decrease sharply in 9 of the 10 migraineurs and in all controls. Appenzeller (1969) thought his findings could indicate that the blood vessels in migrianeurs are chronically constricted due to excessive sympathetic neural discharge.

In an independent study Elliott, Frewin and Downey (1974) essentially replicated Appenzeller's (1969) results. Heating of the contralateral arm resulted in strong finger dilatation in 17 normal volunteers but weak or moderate dilatations in 12 migraine sufferers. The weak thermal responses in the migraine group tended to occur in the older patients. Elliott's et al (1974) results added support to the notion of abnormal neurovascular control in migraine.

Other researchers, however, have been unable to demonstrate the impairment of the thermal reflex with migraine reported by Appenzeller (1969) and Elliott et al (1974). Hockaday, Macmillan and Whitty (1967) failed to find any thermal reflex abnormalities during heating in 5 women whose migraines were precipitated by oral contraceptives. French, Lassers and Desai (1967) also failed to find a significant difference in hand blood flow between 15 migraine patients and a control group. Downey and Frewin (1972) found that the decrease in hand blood flow was higher in the patients than in the normals, leading Downey and Frewin (1972) to speculate that the resting sympathetic tone in the hand blood vessels of migraineurs is lower than normal - a conclusion exactly opposite to Appen-

zeller's (1969). The evidence from thermal reflex studies of "a general abnormality of vascular behaviour in many migrainous subjects" (Vascular response in migraine, 1964) remains equivocal.

The extensive and diverse literature regarding neuro-vascular processes in migraine can be summarized as follows. Parasympathetic fibers from the brain stem exert vasodilatory control of pial and scalp arteries. Sympathetic fibers from the paravertebral sympathetic chains exert vasoconstrictive control at the head. Forehead skin capillaries may act differently than underlying arterioles. Cerebral blood flow is reduce during the prodrome in patients with classical migraine, and increased during the headache phase. Weak data suggests that cerebral and extracranial blood flow increases during emotional arousal, while hand blood flow decreases.

Decreased forehead blood flow occured during learned hand vasodilatation in one study of normal subjects, but passive heat dilatation of the hand resulted in a concomitant dilatation of the forehead (Sovak et al, 1976). Head and hand blood flow covaried during passive relaxation tasks; the direction of change was vasoconstrictive in migraineurs but dilatory in normals (Price & Tursky, 1976). The relationship between head and hand blood flow, particularly in migraine sufferers, require corroboration. Surgical resection of the GSP nerve relieved migraine in the majority of patients undergoing the procedure, but the findings are confounded with simultaneous sectioning of arteries and are, therefore, not definitive. Electroencephalographic studies generally agree that the incidence of diffuse eeg abnormalities is higher in migraine sufferers than in comparable controls; but no eeg pattern reliably diagnostic of migraine has been found. Migraine sufferers may exhibit thermal reflex abnormalities, but some researchers were unable to replicate this finding.

Hypothalamic mediation has long been suspected in migraine, usually on the basis of diverse "hypothalamic" symptoms associated with migraine (Friedman, 1970; Herberg, 1967, 1975). Stimulation of the "stress" area of the posterior hypothalamus results in an immediate slowing of cerebral blood flow, followed 5 to 10 seconds later by cerebral vasodilation (Geiger & Sigg, 1955). The accumulated neurovascular data on migraine, although weak and inferential when examined study by study, tend nonetheless, as a whole to suggest that migraine is a type of nervous system dysfunction. The particular neural events leading to the migraine attack are still being identified and related to each other. A remark by Friedman (1970) to the Third Migraine Symposium remains one of the best summary statements on the etiology of migraine:

> The key to understanding the start of a migraine attack lies in the central nervous and central autonomic systems. The inseparability of psychic phenomena and physiological changes is well illustrated by this disorder, and the patient's behavour and personality are closely related to the oneset of the migraine attack. (p. 172)

Adverse Effects of Migraine Medication:

One impetus toward a non-pharmaceutical treatment of migraine is the problem of side effects and drug abuse associated with migraine medications. The side effects of the most frequently prescribed migraine medication, ergotamine tartrate, have been well documented:

> Nausea, vomiting, weakness in the legs, muscle pains in the extremities, numbness and tingling of fingers and toes, precordial distress and pain and trasient tachycardia or bradycardia. (Rotenberg, Otterbein & Hughes, 1976, p. 320)

These side effects may be prominent in some sufferers but not occur in others. Nausea and vomiting are the most common, and Sacks (1970) has remarked that patients who suffer side effects:

> may face a pretty choice between enduring a violent headache with relatively little malaise, or eliminating the headache at the expense of repeated nausea and vomiting. (p. 262)

Other migraine medications also carry the risk of dangerous side effects. Methysergide maleate, for instance, has been implicated in the development of retroperitoneal and pleuro-pulmonary fibrosis in some migraine patients (Rotenberg et al, 1976). The side effects of pharmaceutical treatment of migraine make the development of a drug free migraine treatment desirable.

While the side effects of specific migraine medications are distressing to the migraine patient, the excessive use of analgesics is dangerous and sometimes fatal. Dalessio (1970) has noted that "headache complications associated with analgesic abuse are most always more dangerous than the headaches themselves" (p. 637). Dalessio (1970) and Diamond & Dalessio (1973) have warned physicians to check for analgesic abuse in their migraine patients. "The syndrome of analgesic abuse occurs relatively frequently in patients with chronic headaches" (Diamond & Dalessio, 1973, p. 101). Headache patients are often secretive about their analgesic use (Diamond & Dalesio, 1973; Fellner & Tuttle, 1969) making drug abuse data difficult to obtain and potentially unreliable.

Medina & Diamond (1977) discovered 21 cases of drug addiction, abuse or physical or psychological dependency in a sample of 2,369 new headache patients seen at a neurological clinic. The criteria for addiction, abuse and dependency were stringent. A one percent incidence of headache-related drug problems is probably a minimum since, as Medina & Diamond (1977) noted:

> It is our belief that some patients are denying their dependency on drugs. We have limited our study to patients whom most physicians will consider mismanaged because of

excessive and prolonged use of drugs. (p. 13)

Fellner & Tuttle (1969) observed 35 patients known to have taken large amounts of phenacetin - containing compounds, and found that 30 reported long-standing migraine headaches. All 35 patients exhibited the characteristic kidney damage (pyelonephritis) that follows long term analgesic abuse. The mean age of the 24 survivors was 49.3 years. On the basis of interviews, the physicians identified psychopathology in 17 patients, most commonly "psychoneurotic depressions and anxiety reactions" (p. 380). Migraine, psychological distress and excessive drug use apparantly combine to form a drug abuse syndrome.

Even sustained high consumption of aspirin can produce kidney disorder, peptic ulcer, gastritis and reduced red blood cell production. The side effects often appear in individuals whose consumption has reached 1 kilogram (kg.). "In actual practice, one finds that (headache) patients may have ingested considerably more than 1 kg. of aspirin or aspirin compounds" (Diamond & Dalessio, 1973, p. 95). Any drug-free treatment for migraine, such as biofeedback or relaxation training, clearly deserves investigation.

Migraine as Chronic Pain:

Individuals with frequent migraines can be thought of as suffering nearly chronic pain. The experience of pain is more than a simple sensation: a gamut of cultural (Zbrowski, 1969), environmental (Fordyce, Fowler & DeLateur, 1968) and psychological (Sternbach, 1968) factors influence the "reaction" of pain (Beecher, 1957). The purpose of this section is to note the factors affecting pain experience and to examine the usefulness of standardized pain questionnaires, particularly Melzack's (1975a) for assesing migraine treatments.

Beecher (1957) and Melzack (1975b) have affirmed that the reaction to noxious stimulation is the most important aspect of the pain experience. Beecher (1957) grouped reactions to pain into three classes; musculoskeletal, autonomic and "processing by the central nervous system" (p. 164). The central processing was thought to be the most important because "it can determine the presence or absence of suffering" (p. 164). Melzack (1975b) offered similar remarks:

> Pain signals can be blocked by means of fibers that decend from the areas of the brain (predominantly the cortex) which are involved in the memory of cultural experiences, expectation, suggestion and anxiety - those psychological processes known as higher nervous system activities. (p. 257)

Psychological factors then, are thought to strongly influence pain experience (Merskey, 1975; Murray, 1971; Weisenberg, Note 7).

The psychological factors differ, however, between clinical and laboratory pain situations (Beecher, 1957; Bishop, 1946). The clinical findings appear to be more generalizable to "real life". Beecher (1975), summarized a series of 8 studies and found:

> A large dose of morphine is not capable of consistently and significantly altering the brief jabs of experimental pain, even in properly set up and controlled experiments in man. Compare this with the fact that much smaller dose of morphine consistently reduce, often check completely, the severe pain of an operative incision or great wound. It seems that the sensible conclusion is that, significantly, the two situations are not comparable, and that something more than stimulation of nerve ends is involved, believed here to be reaction. (p. 169)

Bishop (1946) also emphasized the importance of subjective reactions in determining pain experience.

> A comparison of the attitude of the subject undergoing pain stimulation as an experimental procedure with that of the sick and anxious patient whose pain is mysterious, unpredictable and of unknown causation, not to mention the factor of persistance of pain, indicates that in casual experience the reaction to pain may be of more significance to the animal than it s mere perception. (p. 97)

Clinical pain is apparently a different experience than laboratory pain.

Sternbach (1968) discussed the relative reactivity to different clinical pains in terms of the sufferer's ability to place "psychological distance" (p. 82) between himself and his pain. Thus a pain in the extremities due to external injury is less distressing than severe headache, because the former is "out there" whereas headaches (and other chronic visceral pains) "invade and occupy" the body.

> It is not any longer a matter of <u>having</u> a body that has a hurt member, but we <u>are</u> a body that is almost entirely pain. The pain is insistent, and crowds out all other feelings, ideas, and perceptions. It persists, and exhausts us as though we had fought, and lost, a strenuous battle.
>
> (Sternbach, 1968, p.82-3)

Sternbach (1968) concluded that <u>typically</u> pain involved reflexive protective behaviour, and "regressed expression of anxiety" and "interpersonal manipulation manoevers" (p. 93).

Pain reaction as a form of communication has been discussed by several authors (e.g. Merskey & Spear, 1967; Szasz, 1957; Weisenberg, Note 7; Zborowski, 1969). Pain reactions can have a number of meanings, such as "Don't hurt me", or "Help me" (Weisenberg, Note 7). Merskey & Spear (1967) used a Biblical source to illustrate pain as communication.

> It is nothing to you, all ye that pass by? Behold and see if there be any pain like unto may pain which is done unto me, wherewith the Lord hath afflicted me in the day of His fierce anger, From above He hath sent fire into my bones...
>
> (Lamentations I, 12-13)

Mersky & Spear (1967) drew attention to the "appeal for sympathy or attention" and "the criticism or resentment that others ignore misery" in this Biblical passage. Their comments were very similar to a scenario portrayed by Sternbach (1968):

> The family's response to mother's pain is usually one of frustrated anger. The anger is a reaction to the tyrannical aspect of the pain, because here the pain response is itself

> an angry one (it frustrates and provokes anger) and a plea for help (it elicits concerned attention). (p. 89)

As Sternbach (1968) illustrated, pain can express a variety of needs, such as dependency, expiation, or revenge (Szasz, 1957).

Systematic clinical research has examined several factors influencing the pain experience. Social modelling is one potent influence. Zborowski (1969) found that various American ethnic groups differed in their response to pain, suggesting learned cultural norms for pain expression. Even expression of the intense pain suffered by patients on a burn control ward appears to be regulated somewhat by social modelling processes (Fagerhaugh, 1974). Learning self-control techniques helps some chronic pain patients to cope with their pain (Gotleib, Hockersmith, Koller & Strite, Note 8), but the procedures are not always effective (Weisenberg, Note 7). Averill(1973) noted that the pain control programs incorporated behavioural, cognitive and decisional elements, and the relative importance of each was still not established. The use of reinforcers available on a hospital ward can alter overt pain behaviours such as remaining bedridden, Fordyce, Fowler & DeLateur (1968) were able to train chronic pain patients to spend more time out of bed by reinforcing walking around the ward with medication, rest and social attention. A similar token program has also been effective (Ritchie, 1976). The expression of pain apparantly exhibits a clear behavioural component.

Attitudes and anxiety have also been found to influence pain experience. Weisenberg, Kreindler, Schachat, & Werboff (1975) successfully differentiated the response to dental treatment of negroes, whites and Puerto Ricans using two anxiety and two attitudinal scales. Johnson & Baldwin (1968) found that mothers' anxiety about dental care predicted

How distressed their children were during dental treatment. Pollack (1966) showed that a placebo and topical anesthesia were equally effective in reducing pain so long as the suggestion of relief was accepted. After extensive experimentation Beecher (1972) stated "a new principle of drug action: some agents are effective only in the presence of a required mental state" (p. 178). Placebos, whose effectiveness is due entirely to suggestion, attention, and other psychological variables, continue to surprize investigators with their potency. The placebo effect in migraine research is discussed in detail later under "Research Issues", but one observation here should serve to underscore the importance of this variable. Beecher (1975) reviewed studies of 1,082 patients with a wide variety of pain problems and found that 35% had their pain satisfactorally relieved by placebos. Fearful attitudes and anxiety exacerbate the pain experience, whereas,

> Relaxation, strong suggestion, faith in the physician and his techniques and distraction have all been demonstrated to diminish both anxiety and pain. (Melzack, 1975b, p. 257)

Pain, then, is a complex experience involving as much a reaction component as the original noxious stimulus itself.

The measurement of pain poses problems that reflect the complex, subjective nature of the experience. Wolff & Langley, (1975) noted a lack of general pain terminology which has confused comparisons between studies. Two problems render the measurement of pain difficult. First of all it is virtually impossible to provide an adequate definition of pain. Operational definitions, such as withdrawal, verbal reports or analgesic use all probably indicate pain, but fail to define it, since pain is a subjective experience (Edwards, 1950). Beecher (1957) remarked:

> It seems paradoxical to speak...of measuring something which cannot be satisfactorily defined, and if this were true it would be paradox or nonsense or both. The fact is, pain is defined introspectively by every man. The difficulty comes in verbalizing this well known experience, not very difficult in terms of statements of its presence or absence in various degrees or kinds but in saying what it is. (p. 63)

The second problem in measuring pain is the different conceptual levels that have been used. Pain has been described "in neurological, physiological, behavioural and affective 'languages'" (Sternbach, 1968; p. 12). Each language carries its own terminology and assumptions about pain, again clouding comparisons between studies. The 'language' difficulties are further compounded by research design problems, as Beecher (1975) noted:

> It long ago became apparent from study of the worlds literature on pain that this subjective experience is one surrounded by many pitfalls to trap the unwary. It early became apparant that sound design of study was to be of paramount importance. (p. 39)

Clinical pain has been described in a number of ways. Keele (1975), in assessing the pain of noxious chemical stimulation, had subjects describe their experiences spontaneously. Keele (1975) categorized their responses as indicating non-painful "uncomfortabe tingling" ("metaesthesia") or truly painful sensations ("alaesthesia"). Keele's (1975) procedure suffers a number of obvious flaws, but one of the most important is lack of standardized descriptors. One man's "tingling" might be another's "prickling" but the comparability of the terms is unclear. As well, subjects often have difficulty thinking of apt words to describe pain (Melzack, 1975a), an observation which makes the use of spontaneous pain description even less desirable. Also, the relative

contribution of sensation and reaction cannot be established by Keele's (1975) spontaneous description procedure.

Petrovich (1957, 1958) developed the "Pain Apperception Test" (PAT) as a projective test of "attitudes and reactions regarding pain" (Petrovich, 1957 p.340). The test consists of 25 pictures in which someone has received or is about to receive physical pain. Subjects are asked: "How does the man/woman feel?" and "How long will it hurt him/her?" The first question is answered on a 1 to 7 scale indicating "no pain" to "can't stand the pain", and the second answer is scored in months. The pictures are intended to elicit responses involving "felt-sensation", anticipation of felt-sensation, accidental self-injury and accidental injury by others. Petrovich (1958) found split-half reliabilities for the PAT of from .56 to .85. Nine of the pictures correlated with the neuroticism dimension of the Maudsley Personality Inventory. On the basis of his PAT studies Petrovich (1958) infered that the anticipation of pain involved 5 main factors; 1) physical painfulness of the impending injury, 2) imminence of the pain, 3) degree of control by the recipient of the pain, 4) intention of the pain (harmful or beneficial), and 5) the neuroticism or manifest anxiety of the recipient. Petrovich (1958) concluded:

> It is felt that these factors all contribute to the particular pattern of past associations, present personality, and situational variables responsible for a given individual's reaction to a specific situation wherein pain is anticipated.
>
> (p. 372)

Although Petrovich's (1957, 1958) work was innovative, particularly in its attention to the reaction component of pain, the PAT is not an optimal instrument for the measurement of pain. The use of a projective test to measure a subjective response tends to further obscure the actual

nature of the experience being measured. As well, there has been some difficulty replicating Petrovich's (1958) findings (Merskey and Spear, 1967, p.165). Finally, the PAT does little to tap the sensory feature of pain, and is imprecise in assessing possible dimensions of affect and evaluation in the reaction to pain. For these reasons the PAT would not be a suitable measurement device for migraine, even though Petrovich's (1958) conceptualization of the anticipation of pain is extremely cogent to the migraine problem.

Neurological and psychological considerations were incorporated into a five-level taxonomy of pain proposed by Loeser and Black (1975). The levels progressed from the simplest neural description to the most global use of the word "pain" to describe psychological distress. Thus level 1 described pain in terms of nociception by A-delta and C neural fibers. Level 2 consisted of specific pain perception systems in the spinal cord and primitive brain. Level 3 refered to the little-understood short axon, non-specific pain system, which is most developed in the reticular formation. Pain level 4 was characterized as perceived clinical pain, or "suffering". Loeser and Black (1975) believed that level 4 pain was the type of pain most often treated by physicians, and that it was influenced by a wide range of non-neurological factors such as race, sex and situation. Level 5 pain refered to the semantic extension of a pain vocabulary into the description of psychological distress when no basis for pain was present at levels 1 to 4. Loeser's and Black's (1975) taxonomy of pain then, offered a convenient and comprehensive structure for organizing the discussion of pain. Their analysis has been cited by other researchers (Bailey and Davidson, 1976). Unfortunately, Loeser and Black (1975) did not present techniques for accurately measuring the different levels of

pain. Their conceptualization of pain has also been criticised on other grounds:

> To talk of pain levels 1-3 which are in fact physiological parameters, in the same sequence as pain levels 4-5, which are responses to a perceived input (why not themselves perceptions?), is to set up a hierarchy which is not homogenous. (Merskey, 1975 p. 301)

Loeser's & Black's (1975) taxonomy of pain does not appear to be adequate for the systematic analysis of migraine pain.

The most comprehensive and useful assessment procedure for clinical pain appears to be the McGill Pain Questionnaire (MPQ) developed by Melzack and his co-workers (Melzack, 1973, 1975a; Melzack & Torgerson, 1971). Melzack & Torgerson (1971) began collecting 102 adjectives that described pain and grouping them into three main classes and 13 subclasses. The classes were:

> 1) words that describe sensory qualities in terms of temporal, spatial, pressure, thermal and other properties; 2) words that describe affective qualities in terms of tension, fear, and autonomic properties that are part of the pain experience; 3) evaluative words that describe the subjective overall intensity of the total experience of pain. Each subclass, which was given a descriptive label, consisted of a group of words that appeared to be similar in kind. (p. 51)

Twenty subjects indicated whether they agreed or disagreed that each word belonged in its subclass. The eleven words that had less than 65% agreement were reclassified using a forced choice format. After the adjectives were classified, groups of students, patients and physicians rated the relative intensity of pain described by each word. Agreement among the groups was moderate. Two-thirds of the words were ranked in the same order of intensity by all three groups. Melzack & Torgerson (1971) concluded that the many words describing pain could be reliably cat-

gorized into groups representing particular properties of pain. People with divergent backgrounds tended to agree in ranking the intensity of pain described by each word. Melzack and Torgerson (1971) also remarked:

> The fact that there are so many words to describe the experience of pain lends support to the concept that the word 'pain' is a label which represents a myriad of different experiences.
>
> (p.53)

Subsequent research with the MPQ has supported its utility in the assessment of clinical pain (Bailey and Davidson, 1976; Melzack, 1975a; Melzack and Perry, 1975). In a study of 297 patients suffering a variety of painful disorders (e.g. arthritis, cancer, sciatica, etc) Melzack (1975a) demonstrated that the MPQ was sensitive to treatment-related changes in pain. Different scoring techniques correlated highly, and the sensory, affective and evaluative dimensions of the MPQ identified clinically important differences in the pain experience of different disorders. For instance, compared to low back pain the pain of cancer was rated as more intense on the sensory and evaluative dimensions, but less intense on the affective dimension. Thus, the emotional aspect of pain was salient for low back pain patients, but the sensation and meaning of their pain dominated the cancer patients' experience.

In a study of pain control by alpha EEG training, hypnosis, or both (Melzack and Perry, 1975) the MPQ differentiated the effectiveness of the treatment procedures, and as well indicated along which of the sensory, affective and evaluative dimensions the change in pain occured. Bailey and Davidson (1976) factor analyzed intensity ratings of 39 adjectives from the MPQ and found that the first factor was congruent with other measures of intensity, although it accounted for only 15 per cent of the

variance in the ratings. This first 'intensity' factor was best characterized by affective and evaluative adjectives. The MPQ appears to be a reliable and useful instrument for the measurement of clinical pain, and for assessing the effect of treatment. Its ability to separate sensory, affective and evaluative components of the pain experience make the MPQ particularly appropriate to the evaluation of psychosomatic pain such as migraine. To date the factorial structure and sensitivity to treatment effects of the MPQ has not been examined for migraine.

Weisenberg (Note 7) emphasized the need for comprehensive measurement of all the variables related to pain, including:

> social, attitudinal or other psychological factors which go into the predictive assessment of matching person to the optimal pain technique. (p. 10)

The clinician needs to measure both changes in pain over time, and to predict response to treatment. Weisenberg (Note 7) advised researchers to take a broad approach to the prediction of treatment effects:

> The predictive-type assessment is still badly lacking... It would seem desirable to include in any predictive type of instrument some measures of anxiety, attitudes and coping style appropriate for a given treatment. (p. 7)

To the extent that pain is psychologically mediated, personality questionnaires may aid in predicting the response to treatment of chronic pain.

Personality Variables in Migraine:

The published reports concerning personality variables in headache sufferers can readily be grouped into two categories: 1) clinical impressions, and 2 psychometric assessments. Although the clinical reports were generally published earlier than the psychometric results, the findings tend to be congruent.

Following Stenbäch (1954) personality will be discussed as the individual's psycho-physical make-up as it appears at any time. Stenbäch (1954) considered personality to be the product of a hereditarily endowed constitution influenced by physical and psychological environmental factors.

Wolff (1937) offered the original rationale for the relevance of personality variables to headache. He had noted that certain personality characteristics occured among headache sufferers with striking frequency. Not all of his subjects exhibited all of the characteristics, nor were the personality features pathognomic of headache, nor were they found in headache sufferers alone. Nonetheless, he believed that the personality features interacted with certain stressful life situations to create undesirable emotional reactions. Headaches typically occured in predisposed subjects during such emotional reactions. Therefore, Wolff (1937) argued, the personality functions of the headache sufferer play an important role in the syndrome.

Some studies have found differential personality characteristics accompanying migraine and muscle contraction headache types. Other studies have apparently used mixed headache subjects or have not restricted their comments to one headache type. Some personality variables such as depression, have been found with muscle contraction and migraine headache. Substantial gaps exist in our present knowledge of the relationship between headache and personality variables. A particularly strong need exists for controlled treatment studies that attempt to identify the personality (and symptom) correlates separating patients who improve from those who do not.

Clinical Reports:

Wolff (1937; c.f. Dalessio, 1972) used systematic interviews to determine the personality characteristics of the migraine sufferers. He found that more than ½ were 'delicate' children, or treated as such. They had often been shy, withdrawn and extremely obedient. Typically they were polite, well mannered and conscientious at school. Kolb (1963) found that migraineurs reported their parents as nagging, insistent and often punishing. Ordinary expression of assertiveness was not allowed. The families tended to highly value achievement, follow rigid behaviour patterns and suppress any verbal or direct aggression. Kolb (1963) noted that the migraineurs' familial history offered few opportunities for the child to develop non-contingent self-esteem. His subjects reported many doubts about personal adequacy and often felt obliged to repress feelings of resentment. Wolff (1937) noted that the seemingly docile pre-migrainous child occasionally exhibited a contrasting stubbornness and inflexibility in some situations. Nonetheless, migraineurs were often given responsibilities at an early age. These responsibilities tended to generate anxiety, since experience had taught the child to expect an unfavourable estimate of his efforts by his parent. Similar psychodynamics may be characteristic of the development of headache proneness in general. Kolb (1963) speculated that the headache sufferer,

> ...literally uses the muscles of his head and neck in order to maintain an external appearance of composure while concealing the anxiety aroused in connection with taboo feelings. (p. 35)

Wolff (1937) also noted several personality features that tended to appear frequently in adult migraine sufferers. Ninety percent of the 46 subjects he observed were unusually ambitious and pre-occupied with 'success'. They tended to be perfectionistic and efficient, hardworking,

striving, tireless and exacting. Less than perfect outcomes in any activity - research, housecleaning, hairdressing - annoyed the migraine sufferers. They also experienced difficulty allocating responsibility. Ambition and striving made their work extremely important. Anxiety and tension were occasionally associated with interruptions in their work: 'letdowns' or release of tension seemed to precipitate some migraines on weekends and holidays.

Migraine sufferers (Wolff, 1937) tended to have extremely orderly work habits. They often devised complex card index systems. As well they prefered formalistic styles in music and art. Some patients tended to be repetitious and review their work carefully. The repititiousness was often related to fears of failure or not excelling. The migraineurs tried to reach high self-imposed standards. Continual striving was reflected in the migraineur's attitude toward their headaches. Between attacks they generally disregarded even common sense limits on work and pursuit of goals. However; during a headache they often used it to manipulate and dominate their environment.

The migraine sufferer was usually cautious, courteous and gracious in social relations, but often appeared aloof, impersonal and detached, despite his deliberate charm. About 2/3 of Wolff's (1937) patients harboured strong resentments, which often arose from their personal inflexibility. Although patient and tireless with their own tasks, the migraineurs tended to be impatient at any lack of promptness in others. The migraineur's inflexibility occassionally extended to sexual dissatisfaction, often due to an inability to make marital adaptations and compromises. Wolff's (1937) work, then provided a basis for the clinical opinion that migraine sufferers are tense, inflexible and perfectionistic.

Subsequently, clinical studies essentially echoed Wolff's (1937) remarks. Alvarez (1947) examined 500 migraineurs and reported that they tended to be overly sensitive to threat, and resistant to change, as well as anxious and perfectionistic. After studying 2,000 headache patients Friedman, Von Storch and Merritt (1954) concluded that psychological factors were important in 90 percent of chronic headaches. They did not believe that a specific personality pattern typified the migraine sufferer. However, migraineurs often over-reacted to stress and were unusually perfectionistic, intelligent and sensitive. Friedman et al (1954), using a psychodynamic model, suggested that the personality trends in migraine sufferers were due to unconscious conflicts involving dependency, hostility, and guilt. Similarly, psychoanalytic authors (Fromm-Reichmann, 1959; Sperling, 1964) reported finding unconscious hostility toward consciously loved figures and fantasies of attacks to the head in migraineurs during formal psychoanalysis. The effectiveness of psychoanalysis for alleviating migraine has not been systematically evaluated, but clinical opinion generally holds it not to be particularly effective (Fine, 1969).

Kolb (1963) noted that headaches often occured in interpersonal situations,

> in which the sufferer feels compelled to feel comfortable yet at the same time is struggling to repress his anger and resentment toward someone he is expected to love and respect. (p. 35)

Aring (1974) offered clinical comparisons of headache sufferers which were similar to those of Wolff (1937) and Kolb (1963). Aring (1974) briefly described people who suffer muscle contraction headaches as anxious, fearful, overworked and underpleasured. He suggested that migraineurs had difficulty coping with their own and others aggression. They had learned early in life to value other's approval very highly, and spent

considerable energy gaining approval. Like earlier clinicians, Aring (1974) also thought that migraineurs were unusually striving, orderly, conscienctious and meticulous. The migraineurs tended to become immersed in a morass of detail, and as their hard work brought them more and more responsibility, angry, repressed frustration developed, since they could not maintain their conscientiousness except by extreme energy expenditures. Aring (1974) concluded that the migraine attack,

> ...usually erupts in a setting of unconscious angry feelings associated with sustained resentment, anxiety and frustration, in what might be thought of as a state of energy depletion. (p. 192)

Some clinicians have tabulated their impressions of migraineurs' personalities. In a series of 500 migraine sufferers seen at a neurological clinic, Selby & Lance (1960) found obsessional trends in 23% of the patients, tenseness and hyperactivity in 22% and an anxiety state in 13%. The remaining 42% were 'normal'. Selby & Lance (1960) used simple interview questions to reach their psychological impressions. Daalsgaard-Nielsen (1965) believed about two-thirds of his migraine patients had 'vulnerable psychic constitutions'. They tended to suppress their emotions and appeared sensitive and perfectionistic. Emotional stress precipitated a migraine attack in 68% of Daalsgaard-Nielson's (1965) patients, although all of them reported more than one suspected trigger. Similarly, in a study of patients medically disabled by their migraines Klee (1968) found stress-precipitated attacks in 72% of these extreme sufferers, while 60% were notably perfectionistic. Tabulated clinical impressions of migraineurs confirm the more global retrospective impressions, although the tabulations add at best a small increment of accuracy and reliability.

Several clinicians as well as researchers have linked headache and depression. Selby & Lance (1960) reported that depression was 'not uncommon' after headache. Nineteen of the 500 patients reported severe episodic depression. Cassidy, Flanagan & Spellman (1957) found headache to be the most common somatic symptom in a group of manic-depressive psychiatric patients, but did not specify headache type. Kashiwagi, McClure & Wetzel (1972) noted that 50% of a series of 100 patients with various types of headache were depressed. Migraine and mixed headache were present in 69 of the patients, of whom 22 were depressed. The incidence of depression did not differ significantly between migraine, muscle contraction and mixed headache types.

In summary, the clinical features of the migraine sufferer's personality appear to be feelings of tension and insecurity, manifested as inflexibility, meticulousness, perfectionism and repressed resentment. The headache sufferer seeks the approval of others by accepting unreasonably large work loads; frustration, dissatisfaction and resentment often follow. As repeated frustration, sustained resentment and anxiety become very strong, a headache occurs as an adaptive mechanism to temporarily free the sufferer from his overburden of daily routine.

Psychometric Assessments

Psychometric tests have been used in several studies examining the personality characteristics of headache sufferers. Stenbäch (1954) gave the Bernreuter Personality Inventory (BPI) to 58 headache patients, of whom 42 were migraine headaches and 16 were muscle contraction. The BPI yields four scales: neuroticism (B1N), self-sufficiency (B2S) introversion (B3I) and dominance (B4D). As the normative studies of the BPI predicted, Stenbäch (1954) noted considerable overlap between the self-suf-

ficiency and dominance scales. Stenbäch (1954) found that the neuroticism and introversion scores for both muscle contraction and migraine sufferers were equal. However, migraineurs were more dominant and self-sufficient than the muscle tension headache sufferers. Stenbäch's (1954) results suggested that migraineurs and tension headache sufferers differ along a dimension of dominant aggressiveness versus passive dependence.

Martin (1966) gave psychiatric interviews and Minnesota Multiphasic Personality Inventories (MMPI) to 50 muscle contraction headache outpatients, 40 females and 10 males. His interview data was essentially the same as the clinical material already discussed. On the MMPI the patients typically exhibited elevated scores on the hypocondriasis, depression and hysteria scales (the 'neurotic-triad'). Twenty-three of the 40 females showed the 'conversion V' pattern (depression lower than hypocondriasis and hysteria) often found in patients who somatize their emotional problems. Martin's (1966) psychometric findings are in keeping with clinical reports. Muscle contraction headache sufferers tend to be depressed, unusually concerned about their health and are apt to exhibit emotional stress as a physical ailment.

Crouch, Ziegler & Hassanein (1975) assessed the relationship of depression to migraine using the Zung Self-rating Depression Scale (SDS). Nausea, vomiting, photophobia, difficulty thinking, dizziness and throbbing, steady unilateral pain were <u>not</u> significantly related to depression in migraine sufferers. However, sensory disturbance, paresis, speech disturbance and loss of consciousness occuring during migraine were all significantly related to depression. The overall correlation between migraine symptoms and the Zung SDS were .31. Of the 236 migraine subjects, 96 were classified not depressed (SDS <35), 112 borderline (SDS

35-49) and 28 depressed (SDS>49). Couch et al (1975) demonstrated, then, a modest correlation between depression and migraine symptoms. The more severe neurologic symptoms were strongly related to the presence of depression during migraine.

Kudrow (1974) administered Cattell's 16 Personality Factor (16PF) Scale to 13 cluster headache sufferers. (A cluster headache is similar to migraine, except that the attacks strike in short but extremely severe bursts or 'clusters', typically separated by headache free periods of several months to years. The sex distribution of cluster headache is also different from migraine; males represent over 80% of cluster headache sufferers, but less than 40% of migraineurs.) The headache sufferers differed significantly from the expected normative scores on 5 factors. The patients tended to be reserved, detatched, critical and aloof (Factor A), and conscientious, persevering, responsible, staid and moralistic (Factor G). They appeared self-sufficient, resourceful, and prefered to make their own decision (Q2). Other significant descriptors included controlling and socially precise (Q3), tense, frustrated, driven and overwrought (Q4). The congruence between Kudrow's (1974) psychometic findings and clinical opinion is noteworthy.

Not all personality studies of migraine sufferers have yielded clear-cut findings. Davis, Wetzel, Kashiwagi & McClure (1976) gave the California Personality Inventory (CPI) to a group of 74 headache patients, 29 with migraine, 23 with muscle contraction and 22 with mixed headaches. A clinical diagnosis of depression was significantly related to 7 of the 18 CPI scales, but none of the scales separated subjects by type of headache, suggesting that migraine does not usually occur in individuals with a particular personality profile. Lucas (Note 6) used the Maudsley Per-

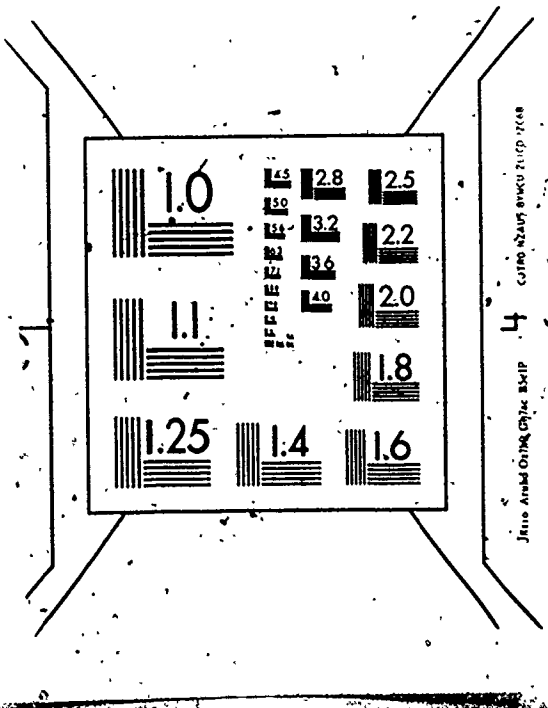
sonality Inventory (MPI) to assess psychoticism, extraversion and neuroticism in MZ and DZ twins. Some twin pairs were both free of migraine, while others were concordant or discordant for migraine. Although Lucas (Note 6) did not analyze his results statistically, he found an overall tendency for the psychoticism score to be lower and the neuroticism score to be higher in migraine sufferers. In MZ twins discordant for migraine the migrainous twin was more extraverted than the migraine free twin, but this relationship was reversed in the DZ twins. Lucas's (Note 6) study, limited in rigour, nonetheless, directs attention to possible complexities in the relationships of personality, heredity and family environment to migraine.

Bihldorff, King & Parnes (1971) compared migraine sufferers to muscle contraction headache and headache free subjects using a check-list of personality characteristics. Of the original 190 adjectives, 23 distinguished the three groups at the .05 level of significance (chi-squared test; not corrected for multiple tests of significance). Migraine sufferers described themselves more frequently as "precise, warm, worrying, tense and sticklers for detail" than the tension headache group, which in turn ascribed those characteristics to themselves more often than the headache free group. Migraine and tension headache sufferers were roughly similar, but significantly lower than the controls, in describing themselves as "carefree, contented, easy going, extravagent, outgoing, stubborn, and trusting". The migraine group said they were the least "irritable, argumentative, adventurous, athletic, happy and lazy". The migraineurs were more "despondent" and "forgetful" than the control group, but less so than the tension headache sufferers. Migraine and tension headache sufferers were both less inclined to express their emotions,

especially anger, than the control group. For example, strong agreement with the statement "If more people expressed their anger when they felt it, there would be less trouble in the world", was endorsed by only 50% of the tension and 9% of the migraine groups, compared to 19% of controls. Bihldorff's et al (1971) findings were in keeping with clinical opinion. The migraine patients tended to be inhibited in expressiveness, particularly of anger, and somewhat compulsive.

Almost all research on personality variables in migraine has involved patients who have come to the attention of a medical specialist. Such patients may not be representative of all migraine sufferers (Rees, 1971). Henryk-Gutt & Rees (1973), in a study of psychological aspects of migraine, attempted to minimize medical selection factors by randomly sampling 50 male and 50 female migraineurs from the British civil service. Non-migraine and headache free groups were also established, and a comparison group of migraine clinic patients was added. A medical interview revealed more frequent emotional difficulties among the migraine sufferers compared to the control groups. Significantly more migraineurs had previously consulted a doctor for emotional symptoms. The migraineurs "bottled up anger and resentment" (p. 148), were unable to express their emotions, lacked assertiveness, and "were subject to excessive mood swings" (p. 148) more than the tension headaches and headache free groups.

Psychometric test results confirmed the findings of the medical interview. On the neuroticism (N) factor of the Eysenck Personality Inventory (EPI) the migraine clinic groups scored significantly higher than the randomly sampled migraine sufferers, who in turn scored higher than the tension headache and headache free groups. The randomly sampled men with classic migraine showed significantly more hostile behaviour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(factor 2, assult, indirect and verbal hostility, irritability) on the Buss-Durkee Hostility-Guilt Inventory (Buss and Durkee, 1957) than the headache free group. The migraine clinic women were significantly higher than all the other groups in both hostile behaviour (factor 2) and hostile attitude (factor 1, resentment, and suspicion). Migrainous women, but not men, scored higher on the "anxiety" and "somatization" scales of the Abridged Minnesota Multiphasic Personality Inventory (MMPI). The female clinic group was significantly higher than the random sample which was in turn higher than the tension headache and headache free groups. Taken together, the psychometric results supports the notion of excessive emotional reactivity, particularly of an anxious, hostile nature, in migraine sufferers. Eysenck (1967) believes, on the basis of a wide spectrum of psychophysiological findings, that high N scores reflect heightened hypothalamic activation. In keeping with Eysenck's (1967) interpretation, Henryk-Gutt & Rees (1973) suggested that their results were:

> ...evidence for increased reactivity of the autonomic nervous system in migraine subjects that may provide a predisposing factor for the development of migraine attacks.
>
> (p. 141)

Schnarch (1977) shared Henryk-Gutt's & Rees's (1973) concern about the effect of sampling on the results of psychological assessments of migraine. In Schnarch's (1977) study:

> ...considerable attention was devoted to avoiding the methodological problems of previous research. The "Clinical Treatment Fallacy" wherein results based on migrainous psychotherapy patients were often generalized into causal inferences for all migraine sufferers, was given particular consideration. (p. 4075)

Schnarch collected headache questionnaire and extensive personality data from 2,306 Michigan State University undergraduates of an original 5,253 contracted. A total of 30 personality scales, developed on the basis of

psychodynamic predictions concerning "migraine personality" were utilized. The scales proved to have good psychometric characteristics. Schnarch's (1977) data did not support the notion of a specific migraine personality. Only 2 of the 30 scales differentiated migrainous from non-migrainous subjects. Migraineurs were more suspicious and more fearful of expressing anger than non-migrainous subjects, although as Schnarch (1977) pointed out, "these differences were actually quite small in an absolute sense" (p. 4706). Schnarch (1977) concluded that his results offered little support for a psychodynamic model of migraine causation or for the suggestion that repression of anger causes migraine.

On the whole systematic research on the role of personality in migraine has had variable results. Further research should attempt to delineate the relationship, if any, between personality variables, symptom patterns, and response to treatment. The effectiveness of such research could be enhanced by using the more recently developed assessment instruments which are less contaminated by response style. Jackson's Personality Research Form (PRF) and Differential Personality Inventory (DPI) would be suitable. The PRF primarily assesses normal functioning (Jackson, 1974) while the DPI assesses psychopathology (Hoffmann & Jackson, 1976). Both are,

> ...based on rational and statistical procedures designed to foster scale independence, internal consistency, and freedom from desirability bias. (p. 862)

The ultimate benefit of linking personality variables with response to treatment could be the development of a convenient pencil and paper test which could predict response to treatment by migraine sufferers.

Behavioural Programs for Migraine:

The many, converging features of migraine make behavioural programs

for alleviating migraine both reasonable and desirable. Migraine is a complex syndrome, diagnostically distinct from muscle contraction headache (Friedman et al, 1962). There may be more than one symptom pattern (Zeigler, 1972). Migrainous pain is due to dilated, hyperpermeable arteries (Dallesio, 1972). The arteries involved are under neural (Edvinson, 1975) and possibly biochemical (Sicuteri, 1972) control. Pharmaceutical treatment often has undesirable side effects (Medina & Diamond, 1977), which are sometimes fatal (Fellner & Tuttle, 1969). Certain personality characteristics may predispose some individuals to migraine (Kudrow, 1974). Drug free behavioural treatments for migraine would seem appropriate to the nature of the complaint, and as well solve some of the problems of existing treatments.

Several behavioural procedures for the control of migraine have in fact been developed. These procedures include relaxation training, hypnosis, biofeedback and behaviour therapy, alone or in various combinations. Historically, the earliest attempts to apply drug-free procedures to migraine involved autogenic training, the mental repetition of 6 physiological self-suggestions related to relaxation (e.g. "my right arm is heavy"). Luthe (1969) reviewed several anecdotal case studies (mostly published in German) and concluded:

> The therapeutic results of the autogenic approach in the treatment of migraine and different forms of headache are variable. In a relatively few cases, no improvement is noted after several months of standard training. The majority of patients, however, report that the frequency and intensity of headache or attacks of migraine is much lower than before therapy. (p. 83)

Luthe (1969) considered autogenic training to be primarily a preventive therapy for migraine. "Mild to moderate diffuse headaches involving large areas of the head" (p. 89) responded particularly well to auto-

Table 1

Summary of Research on the Behavioural Treatment of Migraine without Biofeedback

Authors	Independent Variables	Research Design	Dependent Variables	Subjects Number & Type	Frequency and Duration of Treatment	Results	Comments
Anderson, Basker & Dalton 1975	hypnosis with suggestion, ego-strengthening & autohypnosis versus tranquillizer	2 group outcome study	freq	47, migraine 23 in hypnosis 24 stemetil	6 mo pre baseline, M.D.'s report 6 sessions of hypnosis, 10-14 days apart monthly M.D.'s reports X 12 mo	6 mo pre vs last 6 mo follow: 89% decreased freq for hypnosis (4.5 /mo to .5/mo), 12% decrease for stemetil (3.3/mo to 2.9/mo) complete remission in 44% of hypnosis, 13% stemetil	hypnotherapy vs chemotherapy; "Hawthorne" effect of attention & novelty for hypnosis group
Graham 1975	hypnosis with hand warming suggestion & self-hypnosis	2 systematic case studies	freq intensity duration	2, adults, long history, high Stanford Hypnotic Susceptibility	1 ses/ wk X 5 wks	no migraines at 9 & 12 mo	Ss told to attend to & hand warm at earliest headache cues; subjective hand warming with no FB
Hay & Madders 1971	group relaxation training & discussion	single group outcome study	freq intensity duration IPAT anxiety scale	10 from 98 participants	weekly for 6 wks	3/10 new patients showed "marked improvement". High IPAT anxiety & id pressure	selection of data reported; differences between self-recording & questionnaire report
Lambley 1976	self-monitor (A) + assertiveness training (B) + insight (BC)(cognitive training)	systemmatic case study A, B, BC	freq intensity	1, female age 38, 16 year history of migraine, 3 attacks/wk	weekly X 12 wks, 4 wks/phase of intervention (A, B, BC)	decrease in freq & intensity with B & decrease to Ø with BC; headache free at 9 mo follow-up	note assertive training + modification of cognitive style (insight)
Lutker 1971	Jacobson relaxation, home practice, use at first symptom	anecdotal case report	pain report, medication	1, female student, age 22	4 - 1 hr sessions X 2 wks, + use at first symptom X 1 wk	at 2 mo follow-up no pain and no medication	conditioning self-relaxation & then thought of relaxation, to first symptoms
Maratos, Vallow & Wilkinson Note 9	relaxation vs exercise	2 group outcome study	clinical evaluation of freq, intensity & medication	14 migraine, "precipitated by tension"	1 hr/wk, 6wks	both groups 4/7 improved on clinical migraine, little effect on medication	patients prefered relaxation

Note Abbreviations: EMG=Electromyograph, FB=Feedback, freq=frequency, hr=hour, min=minute, mo=month, S=subject, temp=temperature, wk=week

Table 1 Continued

Author	Independent Variables	Research Design	Dependent Variables	Subjects Number & Type	Frequency and Duration of Treatment	Results	Comments
Mitchell & Mitchell 1971 (study 1)	no-treatment; relaxation application; combined desensitization (CD) (applied relax + SD + assertive)	control group	number duration	17 males & females university students & staff mean age = 22.8	15 sessions, 2/wk 50-60 min each	CD < relaxation < control on both number and duration	possibly a psychologically distressed sample
(study 2)	no-treatment; desensitization alone; CD prior drug failure; CD no prior drugs	control group	number duration	20 males & females university students & staff	15 sessions, 2/wk 50-60 min each	both CD groups significant improvement vs no treatment and relaxation on frequency, but only no prior drugs; CD group significantly superior on duration	possible influence of prior drug failure; results based on analysis of covariance
Mitchell & White 1977	self-recording; self-monitoring; self-management skills (SMS) levels 1 & 2	control group with sequential dismantling to treatments	freq	7 female & 5 male mean age 28.2 mean freq = 14/mo	60 wks continuous self-recording, dismantle to SM SMS1 & SMS2 at 12 wk intervals, + 12 wk follow-up	no effect of self-recording + self-monitoring; 45% reduction with SMS1, 73% reduction with SMS2	elegant design small number long program
Paulley & Haskell 1975	group relaxation training	single group outcome follow-up	self-rating on mailed questionnaire	migraine 51 of 66 returned questionnaire, 800 for clinical impressions	½ hr/wk, 6-8 wks	3/5 "major improvement" 1-8 years later	possible heredity of type O blood in migraine; personality described; psychophysiological group treatment
Warner & Lance 1975	relaxation training with classical conditioning to "relax"; imagery; "hypnoidalization" & self-suggestion	single group outcome study.	freq intensity duration medication	17 tension 12 headaches/mo 12 migraine 8-12 headaches /mo	20 min sessions 1/wk X 4 wks	at 6 mo follow-up 7/17 tension and 8/12 migraine 50% reduction in freq	no self-monitoring self report at start vs questionnaire at 6 mo

genic training, but "moderate to severe attacks of migraine-type headaches were still difficult to control with autogenic training once the initial phase has passed by" (p. 89). As well, autogenic training occasionally initiated localized occipital headaches in individuals previously headache-free. Despite the lack of systematic evaluation, and the equivocal nature of the anecdotal observations, autogenic procedures (and other relaxation techniques) were incorporated into several subsequent behavioural treatments for migraine.

Behavioural programs for migraine that did not use biofeedback are summarized in table 1. These approaches include hypnosis, relaxation training, self-monitoring, and behaviour modification. Table 1 reveals

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Insert Table 1 about here

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seven noteworthy characteristics of these studies. (1) All but the work by Mitchell (Mitchell & Mitchell, 1971; Mitchell & White, 1977) used uncontrolled designs, although the within-subjects systematic case study approach (Blanchard & Young, 1974) was used with three cases (Graham, 1975; Lambley, 1976). Thus, no definite conclusion about the efficacy of the treatments being considered (relaxation training, hypnosis, behaviour modification,etc.) can be drawn from most of the studies reported. (2) A great variety of experimental procedures (independent variables were used. They included relaxation training at home, in weekly sessions alone, or in groups, mental imagery, hypnosis, "hypnoidalization", self-suggestion, assertiveness training, insight, ego-strengthening suggestions, hand warming suggestions, self-recording of headaches, self-monitoring of the situations preceeding headaches, system-

atic desensitization, and behavioural self-management skills training. Some of the treatments were applied concurrently or sequentially. The diversity of treatments alone suggests that no clear-cut, effective procedure has been identified. (3) While procedures have varied, the dependent variables used to measure outcome have been fairly consistent. All but two studies (Paulley & Haskell, 1975; Lutker, 1971) measured headache frequency. Five of the ten behavioural studies also assessed migraine intensity using subject's ratings. Three studies systematically examined medication consumption. Paulley's & Haskell's (1975) study was a single group follow-up that used a self-rating mailed questionnaire to get a global impression of their patients' improvement. Lutker's (1971) anecdotal case study relied on an informal report of symptoms. Since migraine attacks are fairly discrete, frequency and intensity appear to be the most appropriate measures. Medication usage is also a valuable measure, since it too occurs in countable units (number of pills). However, the method of data collection has varied from study to study. Some authors (e.g., Paulley & Haskell, 1975) used a questionnaire, some had their patients report monthly to physicians (Anderson, Basker & Dalton, 1975) and others mixed reporting and questionnaires (Warner & Lance, 1975). Mitchell & Mitchell (1971) used self-recording on take-home forms, and then extended the recording to include self-monitoring of antecedent events (Mitchell & White, 1977), in an attempt to make the record keeping itself a reactive treatment (c.f. Zimmerman & Levitt, 1975). The reliability and validity of these different data acquisition techniques remains controversial. (See Johnson & Bolstad, 1973; Hay & Madders, 1971 who also mentioned this problem.) Self-recording is probably more accurate than questionnaires and interviews, if only because repeated observa-

tions are being made.

A fourth issue raised by the research summarized in Table 1 is the problem of subject selection. All of the subjects in the behavioural studies entered the programs through referral from some sort of clinic. Consequently, the subject selection factors that impunged the epidemiological findings based on clinical populations are also cogent to the behavioural studies. As well, different studies used subjects of different ages. Had the studies used the same or similar procedures, the range of ages would have enhanced the generalizability of the findings. Unfourtunately, each study tended to use a chracteristic type of patient, determined by the population from which referrals were available. The research results therefore, involve to an unknown degree the interaction of subject characteristics and treatment variables. This possibility was made explicit by Graham (1975) whose two (2) cases were selected for high hypnotic susceptibility, and by Maratos, Vallow & Wilkinson (Note 9) whose 14 subjects suffered migraine "precipitated by tension". Even the rigorously designed studies (Mitchell & Mitchell, 1971; Mitchell & White, 1977) probably involved subjects who were particularly psychologically distressed. In Mitchell's & Mitchell's (1971) first study, 13 of the 17 subjects were taking sleeping pills or tranquillizers. Based on a preliminary case report from Mitchell's & White's (1977) study, those subjects may have also suffered psychological problems. Since subject variables such as number, age, selection, headache history and frequency and psychopathology may influence or even determine, the outcome of a study, these variables should be carefully measured, reported and if possible, controlled for, in any systematic research on the behavioural treatment of migraine.

A fifth source of variability in the research in Table 1 is the frequency and duration of treatment. Training ranged from 20 minute sessions each week for 4 weeks (Warner & Lance, 1975) through several studies using ½ to 1 hour sessions for 4 to 8 weeks (Hay & Madders, 1971; Paulley & Haskell, 1975) to a program involving 60 weeks of continuous self-recording and other treatments (Mitchell & White, 1977). The selection of an adequate, but not unnecessarily lengthy, treatment time is of considerable pragmatic importance. Most researchers seem to have arbitrarily selected about 6 weeks.

A sixth feature of the studies summarized in Table 1 is the variability of the results. The lack of a common standard for reporting outcomes make comparison between studies difficult. For example, Anderson et al (1975) noted an 89% mean decrease in number of headaches in their hypnosis group whereas Hay & Madders (1971) reported that 69 of 98 patients "showed a decrease in frequency, severity or duration of attacks" (p. 665). Hay & Madders (1971) also reported some patients' own estimates of their progress; 3 or 10 new patients reported "very marked" improvement, while the remaining 7 changed very little. Warner & Lance (1975) noted that two of their 12 migraine patients were headache free six months after treatment, and another 6 showed more than a 50% reduction in headache frequency. The case studies (Graham, 1975; Lambley, 1976) both reported complete remission of symptoms, which suggests that the case study material may consist of only the instances of best improvement. Given the variability in the type of data reported, all that can reasonably be said about the efficacy of the behavioural treatments for migraine is that some people (perhaps only a few) improve, and that the overall decrease in headaches when all subjects are considered is similar to the

benefits of medication.

A final, seventh, feature of the behavioural studies consists of peripheral issues noted in the "comments" column of Table 1. These issues concern variations in technique that may influence the experimental outcome. For instance, Anderson et al (1975) commented on the possible "Hawthorne" effect of attention and novelty in their hypnosis group. Anderson et al (1975) were also the only researchers to directly compare the effect of medication versus hypnosis, although the tranquillizer that they assessed (Stemetil) was not the most widely used pharmaceutical treatment for migraine. Some researchers met their subjects in groups (Hay & Madders, 1971; Paulley & Haskell, 1975) while others worked with patients individually (Anderson et al, 1975). Several authors openly encouraged subjects to apply whichever technique they were using, either to the beginning of a headache or to their lifestyles in general. All researchers probably encouraged generalization to daily life to some extent. Graham (1975) and Lutker (1971) told their patients to practice hand warming or relaxation respectively as soon as they noticed the first signs of headache. Warner & Lance (1975) attempted to classically condition relaxation to the word "relax", and asked subjects to try brief periods of relaxation throughout their normal day. Lambley (1976) and Mitchell (Mitchell & Mitchell, 1971; Mitchell & White, 1977) emphasized the application of behavioural techniques (assertiveness, applied relaxation, systematic desensitization, self-management skills) to daily life. Two important issues then, are the need to control for the "Hawthorne" or attention-placebo effect, and the problem of generalization of training from the laboratory to daily life. Diamond & Franklin (Note 4) after observing an extensive series of migraine patients

at a residential clinic (which also used biofeedback) emphasized the need for home training:

> Their (migraineurs') activity at home greatly differed from the rigid practice schedule they maintained while at the clinic.... The major problem is incorporating this training into daily life. (p. 4)

Despite the amount of research reported, very little can be reliably concluded about the efficacy of behavioural treatment for migraine (Table 1). With the exception of Mitchell's & Mitchell's (1971) work, and to a lesser extent (due to small sample size) Mitchell's & White's (1977) study inadequate research design precludes any definite conclusions about the specific effectiveness of the behavioural interventions. Nonetheless, the effect of behavioural programs for migraine appears to be roughly similar to that of pharmaceutical treatments. The need for further, more rigorously designed research is self-evident.

In addition to relaxation and behavioural training, procedures for controlling migraine without drugs have recently been developed from biofeedback research. In human biofeedback, information about one or more of an individual's on-going physiological processes is returned to him as an overt sensory representation (Birk, 1973). Biofeedback has been applied to a number of clinical problems such as neuromuscular re-education (Johnson & Garton, 1973), anxiety (Raskin, Johnson & Rondestvedt, 1973), hypertension (Benson, Shapiro, Tursky & Schwartz, 1971), muscle contraction headache (Budzynski, Stoyva, Adler & Mullaney, 1973) as well as migraine (Sargent, Walters & Green, 1973). The alleged benefits of biofeedback programs must be judged carefully however, since the majority of studies in clinical biofeedback applications are methodologically flawed (Blanchard & Young, 1974).

Biofeedback treatment of migraine originated at the Menninger Foundation in the early 1970's. The initial observation was serendipitous. During a psychophysiological study unrelated to headaches a subject with a migraine experienced spontaneous remission and showed a concomitant rise in finger temperature. Sargent et al (1973) began to deliberately train migraine sufferers to warm their hands and cool their heads, using a temperature feedback meter in conjunction with autogenic phrases. Autogenic phrases were used because they included physiological suggestions of hand warming and forehead cooling. Sargent et al (1973) developed a three step rationale for using hand warming biofeedback to reduce migraine. (1) They postulated that migraine may be a type of stress response due to excessive sympathetic arousal, (2) since the sympathetic nerves to the hands constrict blood flow, making the hand colder, hand temperature can be used as a single-variable index of sympathetic arousal. (3) Training migraine sufferers to warm their hands should reduce sympathetic outflow, and thereby, reduce migraine attacks.

Sargent's et al (1973) procedure was straight-forward. Daily, at home, patients tried to warm their hands by watching the temperature meter and practicing autogenic relaxation. The patients kept detailed headache records, including a one month pre-treatment baseline. On the basis of Dr. Sargent's "global clinical judgement", aided by the patients' records of headache intensity and analgesic use, 81% improved. The 81% showing improvement were equally divided between "slight" or "moderate" and "good" or "very good" improvement.

Unfortunately, the Menninger study suffered serious flaws in methodology, data reporting, and possibly rationale. In design it was a single group outcome study. It mixed, in unknown degrees, hand temperature

Table 2

Summary of Research on the Biofeedback Treatment of Migraine

Authors	Independent Variables	Research Design	Dependent Variables	Subjects Number & Type	Frequency and Duration of Treatment	Results	Comments
Adler & Adler 1976	interpretive psychotherapy + EMG & temp FB with passive concentration	single group 5 year follow-up	freq success = 75% decrease	22 migraine 19 muscle contraction 12 mixed 5 cluster	5 to 60 sessions based on individual; at least 2 sessions/wk	success rates: muscle contraction 88%, migraine 81%, mixed 60% cluster 60%	integration of psychtherapy & FB; possible physiological changes during therapy
Andreychuk & Skriver 1975	handwaming FB; alpha EEG FB; self-hypnosis; high vs low hypnotizability (HIP)	3 groups outcome	self-monitored headache index (HI) = duration X intensity	33 migraine volunteers	10 wks; 1 session of 45 min/wk	pre to last 5 wks decreased in density hand 79%, alpha 43% hypnosis 36% - all significant	HIP correlated .48 with HI; higher HIP in temp FB, trend for temp FB to be best treatment; suggestability effect
Beasley 1976 (Abstract)	relaxation exercises + autogenic suggestion + FB (finger temp?); FB only; relaxation & autogenic suggestions; no treatment	control group	freq, EMG, intensity, finger temp	37 female, migraine	10 ½ hr sessions	less freq & intensity for group 1 only; trend for group 2	only combined FB, relaxation & autogenic suggestions effective.
Bild 1976 (Abstract)	Cephalic vasomotor response FB (CVMR); Frontalis EMG FB; waiting list	control group	temporal artery CVMR frontalis EMG --- freq, intensity, duration, medication, headache record 45 days pre & post	19 migraine	10 sessions of FB	CVMR FB reduced temporal artery size; EMG FB decreased EMG; CVMR > EMG waiting list for reducing duration; no effects on intensity; only CVMR reduced medication	migraineurs learned CVMR; CVMR > EMG, but some EMG effect
Diamond & Franklin Note 4	handtemp FB; simultaneous frontalis FB	2 group outcome	freq, severity success = dec-rease in both	93 migraine & mixed	intensive = 12 days X 2 sessions/day; routine = 4-6 wk, 2 sessions/wk	intense overall = 50%; vascular = 90%; mixed = 40%; routine overall = 72%; vascular = 76%; mixed = 68%	vascular response best; careful diagnosis important; routine procedure best; need for home practice

Note Abbreviations: EMG=Electomyograph, FB=Feedback, Freq=frequency, hr=hour, min=minute, mo=month, S=subject, tempt=temperature, wk=week

Table 2 Continued

Author	Independent Variables	Research Design	Dependent Variables	Subjects Number & Type	Frequency and Duration of Treatment	Results	Comments
Diamond & Franklin Note 3	handtemp FB; simultaneous EMG FB	5 group outcome	ffeq, severity success = decrease in both	107; vascular, psychogenic & mixed	4-6 wks, 2 sessions / wk	successes: vascular 12/17; mixed 32/81; psychogenic 5/9 - 40% overall success	no improvement in patients with depression
Drury, DeRisi & Liberman Note 10	reading headache article; relaxation training; autogenic phrases; fingertip temp FB; self-charting	multiple baseline	intensity, duration, freq, & medication	4, migraine carefully screened	5 mos, daily monitoring; treatments introduced sequentially; 4 mo baseline for 1 S	treatment package related to intensity & medication; idiosyncratic variations; monitoring alone reduced headache for the S with long baseline	problem of idiosyncratic responses of the four Ss
Friar & Beatty 1976	pulse amplitude FB, temporal artery vs finger	2 group outcome	freq, intensity, medication, temporal artery pulse	19, frequent ergot responsive migraines	45 min, 8 sessions in 3 wks	larger decrease in temporal artery group for freq of migraine 3 hrs more temporal artery constriction in temporal artery group; finger constriction in both	relationship of temporal artery constriction to headache change not reported
Feuerstein, Adams & Beiman 1976	EMG FB, cephalic vascomotor response (CVMR)	systemmatic case study; multiple baseline; ABACA design	freq, duration EMG, CVMR, number of vasospasms	1 female age 67, 62 year history of migraine, "extremely anxious"	5 wks baseline, 6 wks EMG FB, 8 wks baseline, 6 wks CVMR FB, 8 wk follow-up	no effect of EMG FB on EMG, or CVMR FB on CVMR; but freq decreased while EMG FB; fewer vasospasms during EMG FB, tranquillizer use decreased	older S; increase at follow-up; decrease during EMG FB with no control suggests placebo effect
Graham 1974 (Abstract)	hypnoses only; finger temp FB only; hypnosis + FB	3 groups outcome study	freq, duration intensity, & medication	30, migraine, susceptible to hypnosis	to criterion of +2°F finger temp change in 1 min	all 3 groups improved on all variables; no difference between groups; hypnosis + FB group reached criterion faster	hypnosis = FB = hypnosis + FB on migraine variables
Kentsmith et al 1976	relaxation + medication + finger temp FB; headache diary	anecdotal case study	density, medication, plasma depamine beta hydroxylase (DBH: index of sympathetic activity)	1 female migraine age 45, 31 year history; 1 sex & age matched control	daily home practice	50% decrease in density over 4 mos 71% over 9 mos; 25% decrease in DBH at 4 mos but back to near baseline at 7 mos.	DBH as measure of sympathetic activity

Table 2 Continued

Authors	Indpendent Variables	Research Design	Dependent Variables	Subjects Number Type	Frequency and Duration of Treatment	Results	Comments
Medina Diamond & Franklin 1976	self-relaxation, then 10 min EMG FB then 20 min autogenic phrases with hand temp FB	single group outcome	self-recording of freq intensity & medication	14 migraine, 13 mixed	½ hr, 2/wk, 4 wks + daily home practice with temp FB meter	½ at least a 30% reduction in intensity & medication; 9/14 migraine, 4/13 mixed	year long pre & post records; better for migraine than mixed all Ss elevated hand temp 2°F in first min no effect of age
Mitch McGrady & Iannone 1976	finger temp FB + autogenic phrases	single group outcome	freq, duration intensity, & medication	8 male, 12 female volunteers 60% migraine, 40% mixed mean age 34, mean history 10 years; 10 followed-up	2 months daily home practice then 1-2 sessions /wk & with headache	of 20 patients: 9 had less freq, 12 had less duration, 15 had less intensity; 11 had less medication	4 patients (20%) recieved "minimal counselling"
Pearse, Walters, Sargent & Meers Note 11	hand bloodflow FB; autogenic phrases; body stress scanning; relaxation	follow-up of single group outcome	self-rated improvement & drug reduction	23 migraine &/or tension	5 days, 2hr/day	19 (82.5%) "successful"	multiple treatments
Peper & Grossman Note 12	handwarming FB practice with & without machine at home & school	2 unsystematic case studies	freq & medication	2 childhood migraine; 2 girls age 9 & 13	3 wks daily practice, 2 training sessions	4 mos symptom free no medication	fast handwarming in two children
Sargent, Walters & Green 1973	handwarming FB; autogenic relaxation & handwarming training	single group outcome	intensity total analgesics, analgesic potency	42 no psychological disorders; all migraine	150 days, daily home practice with FB meter	81% some success; 9/42 slight, 25/42 moderate to very good	selection of data reported
Thompson 1977 (Abstract)	positive expectancy vs neutral expectancy, autogenic FB vs no autogenic FB	2X2X12 ANOVA (expectancy XFBXsessions) control group (neutral expectancy no FB)	freq, duration intensity, finger temp baseline to highest value within session change	16 female, 4 male volunteers migraine	12-30 min sessions over 4 wks	FB better control of finger temp (highest value); no finger temp effect of expectancy; all groups headaches improve over sessions, FB X expectancy interaction on intensity	FB not better than no FB for reducing migraines; overall placebo effect; expectancy not significant

Table 2 Continued

Authors	Independent Variables	Research Design	Dependent Variables	Subjects Number Type	Frequency and Duration of Treatment	Results	Comments
Turin Note 1	Finger temp FB: stage 1= 4 Ss warm, 3 Ss cool then 3 cool Ss warm	systemmatic case studies	freq + duration	7 migraine	"short term"	headache activity same or worse during cooling; decreases only during warm	small sample; controll for expectancy only if experimenter gave equal cues
Turin & Johnson 1976	Fingerwarming FB Fingercooling FB	3 systemmatic case studies --- 4 unsystematic case studies	within session finger temp change, freq, duration & medication	1 older adult 6 younger, of whom 4 were students	1 session/wk for 19 wks, 2-5 min baseline, 20 min treatment, 10 sessions down then 9 sessions up finger temp --- 9 sessions finger temp up	improvement during warming only; finger temp up slightly; freq decreased 59%, duration decreased 46%, medicaion decreased 40%	long baseline at each session; very small actual within session change in temp; errors in data table
Weinstock 1972	psychological history; functional analysis of behaviour, behaviour modification, EMG FB, handwarming FB, relaxation, handwarming FB follows EMG FB	single group outcome	freq	7 undifferentiated tension & migraine, 2-35 years duration	4-5 mos, 2 taped relaxation instruction/day	all Ss headache free	emphasises incorporation of headache into lifestyle of sufferers
Wickramaskera 1973	frontalis EMG FB 2-3 wks break, then 3 wks baseline, 10 wks fi-fingerwarm FB, 3 mo follow up; temp FB follows EMG FB	2 systemmatic case studies	freq, finger temp, analgesic use, intensity	2 severe migraine, 15 years duration, psychological problems	16 wks EMG FB, 10 wks finger temp FB; 1 session/wk	no effect of EMG FB; reduction of headache & analgesics to virtually nil with finger temp FB; still effective at 3 mo follow-up; within session finger temp change increases as headaches decrease	lack of effect during EMG FB supports temp effect vs placebo; specific treatment for type of headache; FB free practice at lab & home
Zamani 1974	temporal artery pulse amplitude FB alone (8 Ss) vs relaxation tape (6 Ss)	2 group outcome	freq, duration, peak pain rating, & medication	14 frequent migraine	½ hr, 2/wk, 4 wks + daily home practice without instruments	8 wks pre vs 8 wks post: freq, duration intensity & medication all significantly decreased for FB only	pulse amplitude data not usable & not recorded

biofeedback, self-monitoring, diary keeping, non-specific attention-placebo effects, and the passage of time. As well, only a selection of the data was reported, and that data was a global clinical judgement rather than the actual number of headaches. Sargent's et al (1973) rationale may be suspect in the light of the physiological findings introduced earlier (see pages 41 & 56) in that sympathetic innervation to the head constricts blood flow, so that increased sympathetic activity might actually decrease migraines rather than increase them. On the other hand, some evidence for a reciprocal relationship between head and hand blood flow has been reported (e.g. Sovak et al, 1976), and the issue is certainly far from resolved (c.f. Lance, 1973). Nonetheless, the Menninger Clinic study formed the basis for a great deal of similar work on the use of relaxation training and biofeedback for the treatment of migraine.

The 22 studies that use some form of biofeedback procedure for migraine, with or without other treatments, are summarized in Table 2.

Insert Table 2 about here

As was the case with the purely behavioural studies, all aspects of the research varied enormously from project to project. Only three studies used an adequate control group research design (Beasley, 1976; Bild, 1976; Thompson, 1977). Two authors (Friar & Beatty, 1976; Turin, Note 1) used a bidirectional design, in which one group receives the opposite reinforcement contingency to the other. Thus, Friar & Beatty (1976) trained one group to dilate their temporal arteries while another group learned to constrict the same arteries. Similarly Turin (Note 1) had 3 subjects attempt to cool their fingers while another 4 attempted finger warming.

Although these bidirectional studies appear to be control group designs, they do not meet the minimum requirement for an adequately controlled design, which is "an untreated group of comparable patients who are assessed at the same time as the experimental group" (Blanchard & Young, 1974, p. 574). Lacking a no treatment or attention placebo (Paul, 1968) control group, the bidirectional design studies cannot detect spontaneous remission and other non-specific treatment effects. An apparent superiority of one of the bidirectional groups may simply reveal which group suppresses spontaneous remission less. The plague of methodological inadequacies which rendered most of the behavioural studies at best ambiguous (or at worst completely uninterpretable) are also apparent in the research on biofeedback treatment of migraine. Only 3 studies of 22 meet acceptable standards of methodological rigour.

Great variation among the independent variables (experimental procedures) is also apparent in Table 2. Six studies used hand warming feedback alone (Andrechuk & Skriver, 1975; Beasley, 1976; Graham, 1974; Peper & Grossman, Note 12; Turin, Note 1; Turin & Johnson, 1976). Four studies utilized hand warming biofeedback in conjunction with autogenic training (Beasley, 1976; Mitch et al, 1976; Sargent et al, 1973; Thompson, 1977). Two other researchers used the combined autogenic feedback technique, but added reading an article on the approach (Drury et al, Note 10) or scanning one's body for stress during the day (Pearse et al, Note 11). Treatment at the Diamond Headache Clinic in Chicago (Diamond & Franklin, Note 3, Note 4; Medina et al, 1976) incorporated relaxation electromyograph (EMG) feedback and hand temperature feedback into each session. Weinstock (1972), Wickramaskera (1973) applied EMG feedback for

several weeks before instituting hand warming training. Weinstock (1972) also used behaviour modification technique. Direct operant conditioning of the temporal artery pulse volume was used by Bild (1976), Friar & Beatty (1976), Feuerstein et al (1976), and Zamani (1974). The use of learned vasoconstriction of the temporal artery for alleviating migraine is particularly noteworthy in that it could imply an increase in sympathetic tone, contrary to Sargent's et al (1973) rationale. Other biofeedback approaches to migraine include those of Adler & Adler (1976) who integrated hand temperature feedback with interpretative psychotherapy, Andreychuk & Skriver (1975) who assessed the benefit of alpha EEG feedback and Kentsmith et al (1976) who added both relaxation and meditation training to hand temperature feedback. No consistent relationship between procedure and results appeared in the 22 studies. All the studies reported at least some success, suggesting the possibility of a general placebo effect, rather than specific treatment-related effects, although the two systematic case studies of Wickramaskera (1973) and the control group study of Bild (1976) offer some support for treatment-specific benefits.

Dependent variables and procedures for all but four researchers reported the frequency of headaches (Andreychuk & Skriver, 1975; Kentsmith et al, 1976; Pearse et al, Note 11; Sargent et al, 1973). Of these four exceptions, the "headache index" measures of Andreychuk & Skriver (1975) and Kentsmith et al (1976) implicitly involved frequency, since the index consisted of duration times intensity (which could be called "density") averaged each week. The Menninger studies (Pearse et al, Note 11; Sargent et al, 1973) used a more complex overall rating form that relied heavily on self-rated improvement (Pearse et al, Note 11) or clinical judgement

(Sargent et al, 1973). The self-rating approach may be less reliable (i.e. accurate and reproducible) than self-recording (Johnson & Bolstad, 1973). As well as monitoring headache frequency, 11 studies used subjective intensity ratings, 8 measured duration, and 10 assessed changes in medication use. Some authors groupdd their measures of medication according to potency, which often corresponded to prescription regulations (e.g. Medina, Diamond & Franklin, 1976). Several studies also reported physiological measures. Bild (1976), Friar & Beatty (1976) and Feuerstein et al (1976), who used temporal artery pulse amplitude for feedback, reported changes in that measure. Only three of the authors who fed back finger temperature reported their finger temperature data (Thompson, 1977; Turin & Johnson, 1976; Wickramaskera, 1973). Three studies monitored forehead EMG (Beasley, 1976; Bild, 1976; Feuerstein et al, 1976) and one study (Kentsmith et al, 1976) measured plasma dopamine beta hydroxylase, an index of sympathetic nervous system activity. The most common dependent variable in the biofeedback studies was headache frequency. Intensity, duration, density (duration times intensity rating) and medication were also common measures. Few of the biofeedback studies reported physiological data supporting their hypothesis that headache reduction was mediated by specific learned physiological control. The procedures used for data collection varied from study to study. Most studies utilized a standardized card or diary on which subjects recorded their headaches, but a few studies used more global evaluations (e.g. Pearse et al, Note 11).

The number, age and symptom characteristics of the experimental subjects differed substantially across projects. Some studies relied on

volunteers from the general population (e.g. Tompson, 1977) while other studies used volunteers from medical practices (Andreychuk & Skriver, 1975). Headache Clinic patients were the data base in Diamond's work (Diamond & Franklin, Note 3, Note 4 ; Medina et al, 1976). Most researchers attempted to insure that their subjects were accurately diagnosed, although not all studies were restricted to people suffering from clear-cut migraine. The subjects in Pearse's et al (Note 11) study had "migraine and/or tension" headaches, and Weinstock's (1972) subjects showed "undifferentiated tension and migraine headaches". Other authors, in contrast, "carefully screened" their migraine sufferers (Drury et al, Note 10) to insure a purely migrainous sample, or added additional diagnostic requirements, such as response to ergotamine (Friar & Beatty, 1976). Diamond's group (e.g. Medina et al, 1976) separated their subjects into migraine and mixed headache samples and studied both. Sample size also varied widely, from case studies (Kentsmith et al, 1976) through large single group reports (Diamond & Franklin, Note 3) to moderately sized control group projects (Beasley, 1976). Subjects varied in age from children (Peper & Grossman, Note 12) to the elderly (Reuerstein et al, 1976), although most were adults in their 20's, 30's and 40's (e.g. Mitch et al, 1976). As with the non-biofeedback behavioural studies then, age, number and diagnosis of the subjects in the biofeedback studies varied widely. The only possibly reliable observation relating subject variables to outcome was the tendency for subjects with mixed headaches or depression to not improve as much as clearly diagnosed migraine sufferers (Adler & Adler, 1976; Diamond & Franklin, Note 3; Sargent et al, 1973). More systematic evaluation of psychological and symptom variables

in relation to the alleviation of migraine by biofeedback techniques is needed before a definite conclusion can be made.

Frequency and duration of treatment varied widely in the biofeedback studies summarized in Table 2. Most programs involved between 8 to 10 weekly sessions of ½ to 1 hour duration. Two attempts were made at intensive training (Diamond & Franklin, Note 4.; Pearse et al, Note 11), which consisted of approximately two hours of training daily for 12 or 5 days respectively. At the other extreme Adler & Adler (1976) worked with their patients for up to 60, one (1) hour sessions at the rate of 2 per week, the duration of treatment being based on the individual's needs. No clear relationship appeared between duration of treatment and improvement. Pearse et al (Note 11) claimed 83% "successful" improvement in a followup of their 5 day intensive program, but their results involved patients' subjective judgements. Diamond & Franklin (Note 4) concluded that, overall, their improvement rate was 50% for the intensive program versus 72% for the routine 4 to 6 week program. However, the improvement rate for vascular (migraine) headaches was higher with the intensive program than with the routine one (90% versus 76%). Neither of the intensive programs included control groups. The three control group studies used 10-12 training sessions.

The subjects in several studies practiced daily at home, in some studies with a temperature feedback meter (Medina et al, 1976: Sargent et al, 1973), and in others with taped relaxation instructions (Weinstock, 1972). Zamani (1974) urged her subjects to practice pulse amplitude control daily at home by monitoring their temporal pulse with the fingers of one hand. Home practice should enhance treatment benefits through respon-

se generalization. However, the precise role of home practice is still unclear from the studies to date. Diamond & Franklin (Note 4) emphasized that failure to generalize biofeedback training to daily life was the "major problem" faced by the technique.

The results of the studies on the biofeedback treatment of migraine (Table 2) varied widely, and were not systematically related to the procedures used. For instance, among the studies that examinined hand temperature feedback alone as one of their conditions, Andreychuk & Skriver (1975) reported a 79% decrease in headache density (duration times intensity rating) whereas, Beasley (1976) found no significant decreases in the number or intensity of headaches. Andrechuk & Skriver, (1975) reported a significant superiority of hand temperature feedback over both alpha EEG feedback and hypnosis, but Graham (1974) found no difference between hand temperature feedback, hypnosis and both combined. Beasley (1976) observed significant improvement only in his group that received hand temperature feedback with relaxation training and autogenic suggestions; feedback alone, autogenic relaxation alone and no treatment all failed to generate improvement. Turin & Johnson (1976) found a decrease in the number and duration of migraines during finger warming training but not during finger cooling. The results of hand warming training alone appear equivocal.

The benefits of combined hand warming feedback and autogenic trainings are also uncertain. Beasley (1976) found only the combined autogenic feedback group improved, while autogenic relaxation and feedback alone were ineffective. Sargent et al (1973) reported that 81% of their patients showed at least some improvement, although this result was based on

clinical opinion. Of the 20 subjects in Mitch et al (1976) study, 9 had fewer and 15 had less intense headaches. However, Thompson (1977) showed that the manipulation of positive versus neutral expectancy and autogenic feedback in a rigorous 2 X 2 design had no differential effect on migraine: all 4 groups improved equivalently, suggesting an overall "attention placebo" effect (see Paul, 1969). While the single group outcome studies (Mitch et al, 1976; Sargent et al, 1973) both showed encouraging results, the rigorously controlled studies (Beasley, 1976; Thompson, 1977) were split in their support of combined autogenic feedback as a treatment for migraine.

The addition of EMG feedback to a program of autogenic feedback or hand temperature feedback alone appeared to offer no increment in benefits, even though muscle tension might contribute to the pain in mixed headaches. Diamond & Franklin (Note 3, Note 4) and Medina et al (1976) consistently reported more improvement in migraine rather than mixed headache patients, even with the addition of EMG feedback to their program. The success rate for migraine averaged about 70%. Weinstock's (1972) 7 subjects who had undifferentiated mixed headaches all became headache free after a program of hand temperature and EMG feedback, relaxation training and behaviour modification. Wickramaskera's (1973) 2 subjects did not improve during EMG feedback, but did improve with hand warming feedback. Electromyogram biofeedback certainly is not necessary for the biofeedback treatment of migraine, and may add very little or nothing to the overall treatment package.

Temporal artery pulse volume has been used as a physiological measure for the biofeedback treatment of migraine (Bild, 1976; Friar &

Beatty, 1976; Zamani, 1974). In pulse volume (PV) feedback the subject attempts to learn direct vasoconstrictive control of his temporal artery in order to reduce or eliminate the painful vascular dilation characteristic of migraine. Bild (1976) found that PV feedback was significantly superior to EMG feedback which was in turn better than a waiting list control for decreasing the number and duration of migraines. Intensity was unaffected by any of the procedures, and only PV feedback reduced medication consumption. In Friar's & Beatty's (1976) study subjects who practiced temporal artery constriction reduced their number of major headaches by 46%, compared to a 14% reduction in the finger vasoconstriction group used as a control. Zamani (1974) reported significant reductions in number, duration and intensity of headaches, and medications used, in her PV feedback group that did not appear for a relaxation training comparison group. The number of headaches declined by 66% for the PV feedback group, versus only 2% in the relaxation group. Pulse volume feedback appears to offer a potentially beneficial technique for the control of migraine. It is limited somewhat, however, as a research procedure by the inability of PV transducers to be calibrated from session to session. Only the change from each session's baseline can be measured, which confounds of course baseline shifts with treatment effects. As well, PV feedback equipment is more complex than simple temperature feedback meters.

The overall pattern of results (Table 2) suggests that PV feedback and hand temperature biofeedback, with or without autogenic training may be beneficial for migraine. The addition of EMG feedback does not seem necessary. The control group studies (which separate specific from non-specific effects) were split on the effectiveness of autogenic feedback:

Beasley (1976) found combined autogenic relaxation and feedback to be superior to feedback alone, autogenic relaxation alone and no treatment, but Bild (1976) reported that subjects improved whether or not they had postitive or neutral expectancy about the project, and whether or not they practiced autogenic feedback. As was the case with purely behavioural programs for migraine, further controlled evaluation of the biofeedback treatment of migraine is clearly necessary.

Theoretical and Methodological Issues

The theoretical issue most frequently discussed by biofeedback researchers is whether or not biofeedback represents "pure" operant conditioning of the autonomic nervous system (ANS), free of somatic or cognitive mediation. Kimmel (1967) expressed concern that the reported instances of operant ANS conditioning "may be artifactual consequences of some somatic responses" (p. 343). However, he noted that somatic responses that can reduce an autonomic response are unlikely to increase it as well:

> ...it is one thing to assert that an experimental S (subject) may learn to make some somatic response which, incidentally, elicits a GSR or a change in heart rate, but it is quite something else to claim that in the same experiment Ss learn to not make some somatic response and thus emit fewer GSRs. This is particularly cogent in those studies showing both directions of change within the same Ss. Nevertheless, the more obvious potential somatic mediators should undoubtably be examined, observed or controlled. (p. 343)

Kimmel (1967) seems to have ignored the possibility that subjects could learn different somatic or cognitive manoeuvers to develop spurious ANS control. Nonetheless, Kimmel's (1967) positive evaluations of bidirectional control designs and somatic monitoring seem sound.

Katkin & Murray (1968) discussed the possibility of classical ANS conditioning occuring within operant procedures. Following Skinner (1968)

they noted four ways in which apparantly operant ANS conditioning could be classically mediated. The ANS changes could be unconditioned or conditioned responses to external or internal stimuli. The situation in which an ANS response is classically conditioned to an internal source of stimulation is particularly pertinent when biofeedback is used for therapy:

> ... an individual may engage in subvocal activity (thinking) and this activity may elicit a previously conditioned response pattern. (p. 53)

However, Katkin & Murray (1968) went on to note:

> ... at a practical level a distinction must be drawn between conditioning the ANS and controlling it (Black, 1966). For those researchers whose primary goal is to gain control over ANS function... it may be unnecessary to demonstrate the pure phenomenon of instrumental conditioning... the desired control of autonomic activity might be more efficiently produced by proper reinforcement of both the somatic and the cognitive mediators. (p. 66)

Katkin & Murray (1968) offered "minimal criteria for the acceptability of evidence for instrumental conditioning" (p. 53). For applied researchers Katkin's & Murray's (1968) criteria are useful guidelines for evaluating biofeedback processes:

> First there should be some demonstration that the response being reinforced shows an increase in frequency or amplitude, or probability of occurence over the level shown in a free-operant period. Second, the experimental design should allow comparisons between experimental groups and appropriate control groups. Finally, the data should be, within reasonable limits, free of obvious alternative explanations. (p. 53-54)

Crider, Schwartz & Shnidman (1969) criticized Katkin's & Murray's (1968) theoretical remarks and interpretations of the operant ANS conditioning reports. Crider et al (1969) argued that classical conditioning interpretations of the operant results are weakened by:

> ... findings that the same S^r (reinforcer) can be used either to increase or to decrease the frequency of a given automatic response. (p. 456)

Crider's et al (1969) response is in keeping with Kimmel's (1967) earlier remarks and again emphasizes the utility of the bidirectional research design as a control for classical conditioning effects within the operant paradigm. Crider et al (1969) also questionned the importance of cognitive mediation. They offered two observations that they believed weakened the cognitivists' position:

> ...operant autonomic conditioning seems to be specific to the reinforced response. (p. 457)

> ... it is a currently debatable question that cognitive activity per se produces any marked autonomic effects at all. (p. 458)

Crider's et al (1969) observations, although empirical questions and still far from resolved also underscored the importance of assessing possible somatic and cognitive processes in biofeedback studies. In a rejoinder to Crider et al (1969), Katkin, Murray & Lachman (1969) urged experimenters to attempt to adequately describe,

> ...relationships between verbal reports of mediation and the dependent variables under investigation. (p. 465)

Schwartz (1973) adopted a similar position and stated that researchers should "analyze carefully what else is simultaneously being 'reinforced'" (p. 668).

> The question of mediation is complex, especially at the level of the brain, and one must evaluate (a) relations between responses, (b) the nature of the reinforcement contingency and (c) natural biological environmental and 'state' constraints in order to understand and predict exactly what is being learned. (p. 669-670)

> Patients may be able to learn certain cognitive or somatic strategies which they can use without feedback. (p. 671)

Theoreticians concur then, (Crider et al, 1969; Katkin & Murray, 1968; Katkin et al, 1969; Kimmel, 1967; Schwartz, 1973) in the need to control for and evaluate cognitive and classical conditioning factors in biofeedback research.

Another more concrete way of thinking about the problem of experimental control is to try to operationally define "self-regulation". The operational definition can then be used to evaluate whether or not a particular study is an instance of "self-regulation". Stoyva (1976) "broadly defined" self-regulation as:

> ... the endeavour to modify voluntarily one's own physiological activity, behaviour or processes of consciousness. (p. 2)

Stoyva (1976) did not further refine or operationalize his definition, but from his context he would apparantly accept any change that is not obviously an unconditioned response as an instance of voluntary self-regulation. The least stringent definition of physiological self-regulation then requires two events to be present: a discriminitive stimulus (S^D) and a significant physiological change that occurs when the S^D is present, the physiological change not being an obvious unconditioned response to the S^D. Trivial instances of self-regulation, e.g. holding ice to cause vasoconstriction, are eliminated by this definition. However, it paradoxically admits classically conditioned responses as instances of self-regulation, while excluding the processes that led to their classical conditioning in the first place. For example, if the S^D "imagine yourself lying on a warm beach" was followed by vasodilation, the definition would accept the vasodilation as an instance of self-regulation. However, vasodilation due to actually lying on a warm beach would not be accepted.

Incidently, classical conditioning as a self-regulatory technique has its supporters. Furedy & Paulos (Note 13), in a study of classically conditioned cardiac deceleration remarked:

> The lack of attention to the feasibility of using such Pavlovian behavioural control is probably due to a confluence of factors. First, while the power of operant experimental procedures have been recently emphasized (e.g. Miller, 1969), the parallel power of many Pavlovian procedures (e.g., c.f. Gormezano, 1966) have failed to be as widely noted. Secondly, the very notion of behavioural "control" implies some kind of direct alteration of the target behaviour, while the clinical aspects of the Pavlovian strategy would involve indirect control in the sense of pitting an antagonistic response (i.e. a decelerative CR) against the target response (i.e. stress-elicited acceleration). Needless to say, indirectness of this sort does not entail less behavioural control. (p. 32)

Reberg (Note 14) concured with the potential value of the classical approach noting:

> ... where practical control is the goal, one should certain be prepared to consider any working method. (p. 51)

Miller (1969) took particular care to control for classical mediation in his studies of operant ANS conditioning. He used bidirectional groups to insure that any observed physiological changes had not been inadvertantly elicited as unconditioned responses by the experimental situation (i.e. the S^D) or by reinforcement (S^r). Miller's (1969) remarks about the bidirectional design offered a more stringent definition of voluntary control than Stoyva's (1976) since the possibility of unconditioned classical mediation was more rigorously controlled:

> ... we used the experimental design of rewarding dogs in one group whenever they showed a burst of spontaneous salivation, so that they would be trained to increase salivation and rewarding dogs in another group whenever there was a long interval between spontaneous bursts, so that they would be trained to decrease salivation. If the reward had any unconditioned effect, this effect might be classically conditioned to the experimential situation and therefore produce

> a change in salivation that was not a true instance of instrumental learning. But in classical conditioning the reinforcement must elicit the response that is to be acquired Therefore, conditioning of a response elicited by the reward could produce either an increase or decrease in salivation, depending upon the direction of the unconditioned response elicited by the reward, but it could not produce a change in one direction for one group and in the opposite direction for the other group. The same type of logic applies for any unlearned cumulative after effects of the reward; they could not be in opposite directions for the two groups. With instrumental learning, however, the reward can reinforce any response that immediately preceded it; therefore, the same reward can be used to produce either increases or decreases. (p. 436-437)

Miller's (1969) bidirectional approach can be used as a criterion for judging the rigour of experiments that attempt to demonstrate self-regulation.

Vanderwolf (1971) also struggled with the problem of defining "voluntary" behaviour. He commented:

> There is no generally accepted objective definition of "volution" or of "voluntary movement" and the difference between it and "automatic movement". Some possibilities are that voluntary acts are those that can be brought under the control of many different drive states and can be performed in a variety of combinations and sequences. (p. 83-84)

In the context of physiological self-regulation Vanderwolf's (1971) remarks offer two other, even more rigorous criteria for the demonstration of voluntary physiological self-regulation: the physiological response should at least be able to be turned on and off to more than one reinforcer.

The definitions of voluntary self-regulation provided by Stoyva (1976) Miller (1969) and Vanderwolf (1971) form a hierarcy of increasingly rigorous operational criteria. The minimum criterion is a significant physiological change in the presence of a discriminitive stimulus. This criterion can only generate a list of S^D's that result in physical change.

The appropriate control at this level of description consists of the same stimulus array as the experimenatal condition, except for the particular S^D under study. A unidirectional response to verbal instructions is an example meeting this criterion. The second criterion requires proof that the change is not an unconditioned response. The bidirectional design (e.g. Miller, 1969) appears to be the most effective technique of controlling for at least overt unconditioned responses. The possibility of classically conditioned mediation in humans - e.g. by cognition - cannot be completely eliminated. Bidirectional change to verbal instructions with different meanings (e.g. "Now warm your hands", "Now cool your hands") mixes classical and operant control in unknown proportions, but may be of practical convenience in the development of clinical control (c.f. Katkin & Murray, 1968). Rigorous control for classical mediation would require bidirectional control to one stimulus, for example, both hand warming and cooling after appropriate reinforcement to the stimulus "Now warm your hands". Finally, a further dimension of voluntary self-regulation is demonstrated when a physiological response can be turned on and off for different types of reinforcement (Vanderwolff, 1971). The rigour of attempts to demonstrate voluntary self-regulation can be gauged by the level of criteria they meet.

When the research on voluntary self-regulation of peripheral blood flow and migraine symptoms is evaluated within the framework of Stoyva's (1976), Miller's (1969) and Vanderwolff's (1971) remarks, several features of the work become apparant. (1) First of all, very few of the clinical studies on the voluntary control of migraine, with or without biofeedback, used rigorous designs. In the non-biofeedback behavioural approaches to migraine (Table 1) only Mitchell's (Mitchell & Mit-

chell, 1971; Mitchell & White, 1976) work used control groups. Most of the behavioural studies used a group outcome design, or were case studies. The level of rigour in the biofeedback studies of migraine (Table 2) is also low. Of the 22 studies cited, only 3 used a control group design (Beasley, 1976; Bild, 1976; Thompson, 1977) and none used a bidirectional design sensitive to mediation. Most of the studies were group outcome designs. Three reports used the systematic case study approach (Drury et al, Note 10; Feuerstein et al, 1976; Turin & Johnson, 1976). Among the 3 controlled group studies, results were mixed but generally encouraging. Beasley (1976) found that relaxation training, autogenic suggestions and (finger temperature) biofeedback were effective only in combination and not separately. The data on physiological measurements was not presented in the dissertation abstract. Bild (1976) found that temporal artery pulse volume feedback was more effective than EMG feedback or being on a waiting list for alleviating migraine, although some benefit accrued to the EMG feedback group. In Thompson's (1977) study, autogenic feedback led to the highest finger temperature, but physiological change was unrelated to headache improvement. All four of Thompson's (1977) treatment groups improved, including the neutral expectancy no feedback group, probably indicating an overall placebo effect. Thus, the controlled studies suggest that autogenic feedback (autogenic phrases with feedback) and temporal artery pulse volume feedback can alter peripheral blood flow, but the relationship between learned physiological control and the alleviation of migraine is equivocal. Only 3 biofeedback studies of migraine met even the minimum criterion (c.f. Stoyva, 1976) for demonstrating learned physiological control.

If the applied studies on learned control of peripheral blood flow

Table 3

Summary of Basic Research on the Biofeedback Control of Peripheral Blood Flow

Authors	Independent Variables	Research Design	Dependent Variables	Subjects Number Type	Frequency and Duration of Treatment	Results	Comments
Alberstein 1977 (Abstract)	finger temp FB increase; finger temp FB decrease; relax only (no treatment); EMG FB decrease	bidirectional + control group	finger temp; frontalis EMG	56 male undergraduates	5 sessions: 1 30 min baseline; 4 session consisting of a 10 min wait, 15 min baseline, & 15 min FB	temp increase not different from baseline & relax; temp decrease significant vs baseline & relax; EMG FB decreased EMG vs baseline & relax	EMG & vasoconstriction learned, but not vasodilation
Bloom, Houston & Burish 1976	threat vs no threat of shock	control group	Finger pulse volume (FPV), Heart rate (HR), affect adjective check list (AACL)	192 female undergraduates	1 session, 10 min	residualized score (base free) higher HR, anxiety, lower FPV under threat. Correlations: FPVrHR=-.41;FPV rAACL=-.24;HRrAACL=.25	residual scores; very brief intervention.
Boudewyns 1976 Study 1	sit for 3 min	baseline only (normative data collection)	finger temp	57 male & 76 female, normal adult volunteers	3 min	possible bimodal distribution of initial finger temp	rare bimodal distribution of finger temp; female fingers cooler; higher baseline in summer
Study 2	relaxation tape, 4 shocks vs no shock, relaxation	control group	finger temp, arousal rating (ALR) gain scores	10 males & 11 females from study 1	1 session, 10 min; relax, 7 min stress vs no stress, 15 min relax	finger temp increased with both relaxations; decreased with stress, corresponding changes in rated arousal	
Study 3	same as study 2	control group	finger temp; arousal rating; heart rate (HR) skin conductance (SC) gain scores	20 males & 20 females from study 1	1 session; 10 min; relax, 15 min stress, 15 min relax	decreased temp & increased SC under stress, compared to control group; during second relaxed phase finger temp correlated with arousal -.46	baseline SC differences between experimental and control groups larger than experimental effect itself; finger temp suitable index of arousal for FB
Carlton, M. 1974 (Abstract)	control self-relaxation; autogenic; finger temp FB	control group	finger temp Eysenek Personality Inventory	36 female hospital employees, ages 20-56	3X90 second base trials; 20X90 second trials, 2/day	FB no different from no FB on finger temp; no effect of personality	trend for less temp increase in stable introverts; very brief sessions

Note Abbreviations: EMG=Electomyograph, FB=feedback, freq=frequency, hr=hour, min=minute, mo=month, S=subject, temp=temperature, vs=versus wk=week

Table 3 Continued

Authors	Independent Variables	Research Design	Dependent Variables	Subjects Number Type	Frequency and Duration of Treatment	Results	Comments
Carlton, P 1974 (Abstract)	relaxation - instructed; autogenic; autogenic + finger temp FB	control group	finger temp	30 normal adults	same as Carlton, M.	no differences between the 3 groups	very brief sessions
Gardner & Keefe 1976 (Abstract)	informed vs uninformed of target response by increase vs decrease forehead skin temp	2X2, bidirectional 4 independent groups	forehead skin temp	40 undergraduates	12X30 min; consisting of: 10 min adaptation, 10 min baseline, 10 min FB	increase & decrease in correct direction; mean = ± 1.5°-2.0 F; knowledge of target made acquisition faster but not larger	
Keefe 1975	increase vs decrease finger temp referenced to head, auditory and visual FB	2 bidirectional groups	finger temp change referenced to head	8 male undergraduates	12 daily sessions; 5 min rest, 10 min FB	at session 12 mean increase 1.9°F, mean decrease 1.5°F p .01	small sample; no control at session 4, control appeared at session 8; room temp 21°C; lack of an "instructions only" group noted
Koppman, McDonald & Kunzel 1974	Blood, Volume Pulse (BVP) FB; mental imagery	9 systematic case studies with bidirectional control	temporal artery pulse amplitude (BVP)	9 migraine	1hr, 2-3/wk, for 4 wks	7/9 learned significant control of constricting vs dilating temporal artery	includes critical evaluation of instrumentation
Leeb 1974 (Abstract)	positive, neutral or negative instructional set about autogenic FB hand temp training	control group	finger temp	15 female normals, age 18-35	12 - ½ hr sessions; 1/day	main effect of instructional set; need for positive set for hand temp control supported	instructional not analagous to placebo effect; young normal Ss
Maslach Marshall & Zimbardo 1972	10 hrs hypnosis + 10 min hypnotic suggestion vs none; 1 hand warm other cold	bidirectional (opposite hands) + control	10 sites of hand & forearm temp temp difference between hands	9 paid undergraduates	10 hrs hypnotic training for hypnosis group; then 2 - 10 min testing sessions	hypnotic Ss averaged over 3°C difference between hands; no difference for others; temp increase larger	small sample size; specificity of temp change to hand vs forearm

Table 3 Continued

Authors	Independent Variables	Research Design	Dependent Variables	Subjects Number Type	Frequency and Duration of Treatment	Results	Comments
Ohno, Tanaka, Takeya & Ikemi 1977	finger temp increase; finger temp decrease; false FB; no FB	bidirectional + control group	finger temp; Cornell Medical Index; Manifest Axiety Scale	40 healthy adults	30 min/day, 3 days; 5 min baseline, 17 min training, 5 min post	only 3rd session increase vs decrease; changes significantly different; no pre-post changes significant; increase = $1^{\circ}F$ decrease = $-1.5^{\circ}F$ false FB = $-1^{\circ}F$	no relation of personality scales to finger temp control; small absolute change
Price & Tursky 1976	finger blood volume FB; yoked FB; relaxation tape; neutral tape	control group	finger blood volume; forehead blood volume	40 migraine; 40 non-migraine female volunteers	1 session, 16X2 min periods	no overall effect of groups or treatments; groups and treatments interaction with periods; correlation between finger and forehead pulse volume change = .67 for migraine & = .7 for non	FB no better for finger dilation than yoked control or relaxation tape; positive head vs. hand blood flow correlation contradicts Sargent's et al (1973) rationale
Roberts, Kewman & MacDonald 1973	warming & cooling hands with hot & cold pads; hypnotic training	bidirectional (opposite hands) single group	temp difference between hands	4 female, 2 male undergraduates, "selected", age 20-24, "extensive hypnotic training and experience"; high hypnotic susceptibility	5 to 9 X 1hr training then 9 trials in 3 separate sessions of 10 min hypnosis, 5 min rest & relax, 8 min test trial	3 Ss show more than $1^{\circ}C$ difference between hands	room temp = $22.5^{\circ}C$
Roberts, Schuler, Bacon, Zimmerman & Patterson 1975	warming & cooling opposite hands by audio FB (high vs low susceptibility + absorption)	bidirectional (opposite hands)	difference in finger temp of opposite hands	7 high, 7 low on hypnotic susceptibility + absorption paid undergraduates	16X1 hr sessions; 10 min relax, then 3X8 min trials of temp difference between hands, reverse direction on alternate trials	$1^{\circ}C$ average difference between hands on sessions 8-16; no effect of hypnotic susceptibility + absorption; 4/14 showed mean overall temp difference $1^{\circ}C$	small value of absolute temp difference ($1^{\circ}C$); 4 response patterns: wrong direction, same direction different rates of change, correct change in 1 finger only finger temps diverged or converged; high positive correlation of absolute temp between hands in poor responders & early sessions

Table 3 Continued

Authors	Independent Variables	Research Design	Dependent Variables	Subjects Number Type	Frequency and Duration of Treatment	Results	Comments
Sheridan, Boehm, Ward & Justesen 1976 (Abstract)	control; autogenic phrases; finger temp FB; autogenic phrases + finger temp FB for hand warming	control group	hand temp	40 undergraduates	15 min after stabilizaion, daily for 5 sessions	autogenic phrases alone effective for both male and female Ss; autogenic + FB effective for female Ss only	"these findings may not generalize to patients"; "simplicity & economy of autogenic phrases makes it worthwhile to assess the effectiveness of mere autogenic phrases"
Snyder & Noble 1968	reinforcement (light on) for finger vasoconstriction vs vasostability vs no reinforcement instructions	control group	number of vasoconstrictions/ 5 min	27 male & 27 female undergraduates	1 session; 5 min baseline, 25 min conditioning, 10 min extinction	reinforcement for vasoconstriction caused 5.5 times more vasoconstrictions than vasostability or baseline control which did not change	controlled for body & finger movement, EMG, HR & respiration; vasoconstriction not mediated by these systems
Surwit, Shapiro & Feld 1976 Study 1	increase vs decrease finger skin temp	bidirectional	finger skin temp (& other blood flow measures for comparison)	8 male & 8 female normal volunteers from newspaper ad, ages 18-30	2 baseline & 5 training days; 20X75 second trials/session in 4 blocks of 5 trials	only decrease significant across trials; no effect of days; warm vs cool differed on trials; mean decrease = $-2^{\circ}C$, mean increase = $+25^{\circ}C$, first to last trial block	9 days no more effective than 5 days reflectance plethysmograph & strain gage finger volume & temp all correlate .87 to .96, therefore, increased superficial blood flow
Study 2	as in Study 1, but with cooler ambient temp	as in Study 1	as in Study 1	4 males & 4 females	as in Study 1	lower room temp decreased baseline from $33.3^{\circ}C$ to $29.9^{\circ}C$; training to increase finger temp in cool room led to actual decrease	change in room temp a larger effect than FB training; suggested "arousal" behaviour easier to condition than relaxation
Thompson & Russel 1976 (Abstract)	finger temp FB; yoked no FB controls; relaxation tape	control group (yoked)	finger temp	35 undergraduates	4 sessions; 15 min	only FB Ss increased finger temp	high reported anxiety related to low initial finger temp; visual imagery of heat scenes best technique

Table 3 Continued

Authors	Independent Variables	Research Design	Dependent Variables	Subjects Number Type	Frequency and Duration of Treatment	Results	Comments
York 1975 (Abstract)	autogenic classical vs operant finger temp training (+ migraine vs non-migraine Ss; prefered vs non prefered hand)	counter balanced repeated measures with 2 populations 2 group outcome	hand skin temp	10 migraine 10 non-migraine	6 weekly sessions; 3 operant/ 3 classical	all Ss warmed hand; faster with initial classical conditioning; no differences between migraine vs non migraine Ss; 1 wk cooling enhanced Ss hand warming ability	classical conditioning with heating pad potentialted operant conditioning (autoshaping); anecdotal support for migraine relief

lacked rigour, there were basic studies of good quality. Table 3 summarizes 19 experiments that attempted to train voluntary self-regulation of

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Insert Table 3 about here

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peripheral blood flow. Excepting York's (1975) study, the research is equally divided between control group and bidirectional control studies. Thus, the basic studies are notably more rigorous than the applied. Unfortunately only two of the studies utilized migraineurs as subjects (Price & Tursky, 1976; York, 1975), while the others relied on non-clinical populations, usually undergraduates. Neither Price & Tursky (1976) nor York (1975) found a difference between migrainous and non-migrainous subjects in their response to the blood flow training procedures. Price's & Tursky's (1976) study did show a groups by periods interaction in which non-migrainous subjects exhibited more finger volume dilation than migraineurs. However, for both groups finger dilation was not significantly different under finger volume biofeedback, relaxation or yoked control, indicating that the changes were not treatment specific anyway.

Possible thermal reflex abnormalities in migraineurs may limit the clinical generalizability of biofeedback results based on normal subjects (c.f. Downey & Frewin, 1972; Morley, 1977). Consequently, the basic research findings in Table 3 may not be entirely pertinent to the question of learned control of peripheral blood flow in migraineurs. Nonetheless, their rigour permits an overall conclusion about self-regulation of blood flow, even if the conclusion may only be true for undergraduates and healthy adults. The results in Table 3 show that learned control of

peripheral blood flow has been demonstrated and independently replicated. However, the changes are typically of small magnitude, usually no more than $1^{o}C$ or $1^{o}F$ in either direction when measured as skin temperature. Furthermore, skin temperature decreases, representing increased arousal, are easier to learn and of larger magnitude than skin temperature increases. As well the changes from baseline temperatures are sometimes statistically non-significant (e.g. Ohno et al, 1977); Surwit et al, 1976) or only significant in the decrease direction (e.g. Alberstein, 1976). Often statistical significance is only attained when comparing groups trained to change their hand temperature in opposite directions. Such an approach is apt to generate statistical significance more often than the small changes from baseline actually warrant. Given the small absolute changes in temperature, particularly increases, in normal subjects, voluntary control of hand blood flow in migraineurs may not be a very robust phenomenon. The relationship between voluntary control of blood flow and alleviation of migraine symptoms is another question that requires further elucidation.

The need for rigorously evaluated relaxation and biofeedback treatments for migraine is underscored by the potent placebo effects in migraine therapy noted by virtually all investigators. Placebo, meaning literally "I will please" has come to refer to the non-specific demand characteristics, attention and suggestion effects that are part of research or therapy. The placebo effect has most frequently been documented in drug studies. For instance, in three studies by Friedman & Merritt (1957) totalling 5,237 cases, improvement to placeboes occured for 25%, 50% and 55% of the patients involved. Similarly, Jellinek (1946) observed a success rate (the number of headaches relieved divided by the

number treated) of .81 for various formulae of headache medication compared to .52 for placebo. Waters (1970) compared the widely prescribed migraine medication ergotamine tartrate to placebo pills in a double blind cross over design study. He found that 51% of his subjects improved with ergotamine versus 58% with placebo. Significantly more subjects were made worse with ergotamine, due to side effects. At the end of the trial subjects were offered a small supply of their preferred medication. Of the 43 who accepted the offer, 21 chose ergotamine and 22 chose placebo. Hubbe (1975) believed the placebo effect to be so pervasive in migraine treatment that, in a discussion of controlled clinical trials of migraine medication he concluded:

> On comparison with placebo they (drugs) can be shown to have some effect, but this is generally less the more efficiently the study is carried through. (p. 95)

Careful control for placebo effects in the study of biofeedback treatment of migraine appears mandatory, particularly in light of the lack of conclusive documentation relating symptomatic improvement to physiological change.

Even attempting to measure migraine attacks may influence them. Aside from the issue of reliability in subjective reports, such as ratings of headache intensity, headache diary keeping itself may be a reactive measure. Several authors have discussed and provided data supporting the notion that self-recording of a behaviour can lead to change in that behaviour, without the addition of other interventions (Johnson & White, 1971; McFall & Hammen, 1971; Kazdin, 1974; Lipinski & Nelson, 1974). McFall (1970) studied two groups of cigarette smoking students. Students who recorded the number of cigarettes that they smoked in class increased thier cigarette consumption, whereas, students who recorded

the number of times that they considered smoking but did not tended to decrease cigarette consumption. Zimmerman & Levitt (1975), through 14 psychotherapists, had 22 clients simply count the frequency of a symptom or behaviour using a wrist golf counter. Behaviour modification occured in 8 of the 22 clients "merely by virtue of using the counter and counting" (p. 337), and 14 of the clients reported increased understanding of their symptom. The most direct evidence, however, that headache diary keeping may itself be a reactive measure is an unelaborated remark in an epidemiological study by Waters & O'Conner (1971);

> There is evidence that progressively fewer episodes were recorded during the six months that diaries were kept and there was selective drop out. (p. 150)

Whether the reported decrease in reported headaches accurately reflects an actual change in headache frequency, or instead is simply an artifact of recording in a moot point. The need for careful measurement and design is self-evident.

Hypotheses

The present study attempted to assess whether handwarming training, with or without biofeedback, was a specific, effective technique for the alleviation of migraine. A bidirectional plus control group design was used, so that handwarming autogenic training, with and without hand temperature biofeedback, could be compared to the (counter-theoretical) effects of hand cooling, and an attention-placebo control (c.f. Paul, 1967). Three additional topics were also examined: (1) The relationship of headache symptoms to treatment outcomes. This was followed by (2) the assessment of personality variables in migraine and their relationship to treatment outcome, and (3) the systematic description of migraine pain using the McGill Pain Questionnaire (Melzack & Torgerson, 1971),

measurement of change in the pain experience during treatment, and the relationship of pain characteristics to improvement. The specific hypotheses that followed from the experimental design and the research literature were: (1) If handwarming training is a specific, effective treatment for migraine, subjects practicing hand warming will have significantly fewer headaches than those in the hand cooling or attention placebo groups. (2) If the process by which hand warming is effective is altered blood flow, hand warming subjects who learn to increase their hand temperature more will show the largest decrease in headache symptoms. (3) If biofeedback is an effective technique for learned physiological control, the addition of hand temperature biofeedback to autogenic training will result in larger temperature changes in the desired direction than autogenic phrases alone. (4) If the placebo effect is the main factor operating in the treatment program, improvement in headache symptoms will occur acoss the duration of the experiment, independent of experimental conditions.

METHOD

Subjects:

Sixty volunteer migraine sufferers from the university and general public in London, Ontario, Canada served as subjects. They were solicited by advertisements in the campus and local newspapers and by subsequent publicity concerning the project in the same papers. To be included in the study volunteers had to be over the age of 18, suffer at least two migraines per month, and exhibit at least three migraine symptoms. Acceptable symptoms included unilateral onset, throbbing pain, prodromata, nausea, and a family history. Subjects were initially screened by telephone to insure that they met these criteria, and subsequently examined

and diagnosed independently by Dr. John Brown, a London Neurologist.

Apparatus:

The facilities of the Psychophysiological Laboratory, University Hospital, London, Ontario, Canada were used for physiological measurement during the study. A comfortable reclining chair and electrode connections were in one room while the monitoring equipment was in another. The rooms were separated by a one-way mirror and connected by an intercom. Frontalis EMG was monitored with Hewlitt-Packard (HP) 22mm. silverplated electrodes, amplified by a HP8811A bioelectric amplifier and integrated by a HP8815A integrating pre-amplifier. Modifications to the pre-amplifier enabled it to produce a continuous reading of the preceding .5 second integral of the raw EMG signal. Forehead and fingertip skin temperature were measured by HP8805A carrier pre-amplifier and an HP14060F temperature bridge and thermister. The 3 physiological measures were recorded on a HP7700 heatwriting polygraph, and scored manually.

Other experimental apparatus included a Sony model TC-353D tape recorder and a Sony TA88 audio amplifier, used to present autogenic phrases to the subject in the lab. At appropriate parts of the study volunteers were loaned for home practice, Futura tape cassette players with a 15 minute recording that included either handwarming or handcooling autogenic phrases. Volunteers were also loaned custom built hand temperature meters. The meters consisted of a thermister similar to the one used in the psychophysiology lab, and a meter display with adjustable zero point, designed so that one inch deflection of the meter corresponded to $1^{o}F$ change in the temperature at the thermister.

Formal consent to participate in the study was obtained on the con-

sent form (Appendix I) worded to meet ethical standards of informed consent. At the end of the study each volunteer was debriefed and received a debriefing form (Appendix V). Headache symptoms were measured by a 45 item headache symptom questionnaire constructed for the project (Appendix II). Subjects recorded individual headaches on a headache diary sheet (Appendix III). Personality was assessed by Cattell's 16 Personality Factor Test Form A (16PF: Institute for Personality and Ability Testing, 1967), Jackson's Personality Research Form (PRF: Jackson, 1974) and Jackson's Differential Personality Inventory (DPI: Jackson & Carlson, 1973; Jackson & Messick, 1975). Subjects described their headache pain by completing the McGill Pain Questionnaire (Appendix IV). The pencil and paper test material was supplied to each volunteer at his/her first session in a randomly arranged packet.

Procedure:

Volunteer migraine sufferers, having passed the initial telephone screening, were seen individually at the Psychophysiology Lab, either during the day or in the evening. At the first meeting the project was briefly outlined to the volunteer and consent was obtained. The volunteer received the packet of headache diary sheets and questionnaires, and sat through a sample physiological monitoring to habituate him/her to the lab.

During the sample monitoring and the 12 subsequent weekly monitorings the subject sat in an easy chair in one room of the lab while the experimenter recorded the physiological measures in the adjacent room. The frontalis EMG reference electrode was affixed one inch above the nasion and the active electrodes two inches bilaterally from the reference (Lippold, 1967). The forehead skin temperature thermister was

Figure 1

Experimental Design

W1	X	I	I+FB
W2	X	I+FB	I
C3	X	I	I+FB
C4	X	I+FB	I
X5	X	X	X
WK	1-4	5-8	9-12

EXPERIMENTAL DESIGN

Note Abbreviations: W = finger warming, C = finger cooling, I = Autogenic Instructions, FB = finger temperature feedback, X = baseline (headache record keeping and weekly lab visits), Wk = weeks.

taped to the forehead on the more painful side of the headaches, between the EMG electrodes. The finger temperature thermister was taped to the palmar surface of the middle finger tip of the subject's left hand, where cutaneous blood flow is greatest (Hertzman, Randall & Jochim, 1946). All monitoring sessions lasted 15 minutes.

Figure 1 shows the experimental design. All subjects began with a

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Insert Figure 1 about here

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4 weeks baseline (marked "X" in the first column of Figure 1). During the baseline, volunteers were asked simply to "let yourself relax as deeply and as completely as possible" when they were being monitored in the lab. The personality, symptom and pain questionnaires were also completed and collected during the 4 weeks of baseline. At the end of week 4 subjects were randomly assigned to treatment conditions. Until the end of week 4 neither the subjects nor the experimenter knew which group the subject would be in. The "attention-placebo/self-monitoring" group 5 continued to follow the baseline procedures. Groups W1 and W2 (warming) were given 15 minute cassette tapes of autogenic instructions (I) that included suggestions of hand warming and forehead cooling. The tapes were augmented for 4 of the 8 treatment weeks with a hand temperature biofeedback meter (FB). Groups W1 and W2 differ only in the order in which they used the feedback meter. Groups C1 and C2 (cooling) followed procedures identical to those of W1 and W2 except that their autogenic instructions included suggestions of hand cooling and forehead warming - thus creating the bidirectional design. Subjects were asked to practice their assigned procedure daily at home. During the weekly lab visits

participants practiced in the lab whichever procedure they were using at home. At the end of week 12, subjects completed a second form of the MPQ, were debriefed (Appendix V), and were offered the loan of any tapes or meters for their own personal use. The autogenic text is in Appendix VI.

Six months later follow-up headache diary sheets were mailed to the participants. They were asked to record their headaches for one month and then return the diary in an enclosed return envelope.

Dependent Variables:

From each subject's headache diary the weekly number, intensity, duration and density of headaches was tabulated, along with prescription and non-prescription drug use. Headache "density" is simply the duration of each headache times its one to three intensity rating, summed for the week. The three physiological measures were scored at minute 1 and minute 15 of each weekly session. Physiological change scores (minute 15 minus minute 1) were calculated for the three physiological measures. Thus, a total of 15 weekly physiological and headache diary variables were analyzed: the minute 1, minute 15, and within session change values for forehead temperature and EMG, hand temperature, headache number, intensity, duration, density, and prescription and non-prescription drug use.

The 45 items of the headache questionnaire and the raw scale scores of the 16PF, PRF and DPI were treated as individual dependent variables. The McGill Pain Questionnaire was scored by calculating each subject's mean adjective endorsement, ranging from 1 ("never" characteristic of their pain) to 5 ("always" characteristic of their pain), on the 14 MPQ subscales.

Data Analysis:

The physiological and headache diary data were analyzed in a 5X3X4

(treatments X blocks X sessions within blocks) analysis of variance (ANOVA), and further examined by factor analysis. The fourteen subscales were analyzed in a 5X2 (treatments X pre/post) ANOVA. All ANOVAs were tested at the $p < .003$ level of significance to maintain an analysis wise error rate of $p < .05$. Pair-wise comparisons were made with Duncan's New Multiple Range Test (MRT: Duncan, 1955).

The descriptive data, consisting of the 15 block 1 physiological and headache diary means, the 14 MPQ pretest subscales, the 45 items of the headache questionnaire, and the scales of the 16PF, PRF, and DPI, were each factor analyzed separately. These 6 factor analyses, as well as elucidating the underlying structure of the data, decomposed it to a manageable number of factor scales for a final discriminant function analysis that related descriptive features to improvement.

After consideration of the ANOVA results, and the raw data, all 60 subjects were divided into 3 improvement groups, irrespective of their original treatment group. The "best improvers" consisted of subjects who had more than a 50% reduction in headache frequency from block 1 to block 2. The "moderate improvers" were people whose decrease in headache frequency was from 1% to 50%. The "non-improvers" were people whose headaches did not change or got worse. Multivariate discriminant function analyses (MDFA: Veldman, 1967) were used to separate the three improvement groups and to establish their defining characteristics. A MDFA distinguishes known groups on the basis of a set of variables measuring the groups. The MDFA establishes (1) if the groups are significantly separate in multidimentional space and (2) which defining characteristics significantly contribute to the separation of the groups. The 6 sets of

Table 4

ANOVA Summary Table for Minute 15

Forehead Temperature

Source	Degrees of Freedom	Mean Square	F Ratio
Treatments (A)	4	1.12	.23
Subjects (S)	55	4.86	
Blocks (B)	2	8.27	7.61*
A X B	8	1.21	1.12
B X S	110	1.09	
Sessions (C)	3	.67	.86
A X C	12	.49	.62
C X S	165	.78	
B X C	6	.92	.97
A X B X C	24	.92	.98
B X C S	330	.94	

*$p < .001$

Table 5

ANOVA Summary Table for

Headache Frequency

Source	Degrees of Freedom	Mean Square	F Ratio
Treatments (A)	4	43.85	1.38
Subjects (S)	55	31.69	
Blocks (B)	2	10.40	8.18*
A X B	8	.99	.78
B X S	110	1.27	
Sessions (C)	3	3.41	2.55
A X C	12	2.33	1.74
C X S	165	1.34	
B X C	6	1.54	1.36
A X B X C	24	1.23	1.09
B X C X S	330	1.13	

*p < .0008

Table 6

ANOVA Summary Table for

Headache Intensity

Source	Degrees of Freedom	Mean Square	F Ratio
Treatments (A)	4	5.89	1.64
Subjects (S)	55	3.59	
Blocks (B)	2	6.21	7.77*
A X B	8	.33	.41
B X S	110	.80	
Sessions (C)	3	2.05	1.82
A X C	12	.91	.81
C X S	165	1.13	
B X C	6	.90	.97
A X B X C	24	1.21	1.31
B X C X S	330	.93	

*p<.001

factor scales from the factor analysis were examined separately by MDFA. Thus, the MDFA identified which, if any, features of the baseline physiological and diary variables, MPQ pre-test, headache symptom questionnaire, 16PF, PRF and DPI distinguished the best improvers and moderate improvers from the non improvers.

RESULTS

In overview, the headache diary variables decreased during the course of the experiment, but treatment specific physiological learning did not take place. Reduction in the number and intensity of headaches was unrelated to physiological change or to assigned treatment, but was related to several personality and symptom variables. A systematic examination of the results follows.

Insert Tables 4, 5 and 6 about here

Physiological Variables: EMG

The minute 1, minute 15, and within session change (minute 15 minus minute 1) values for forehead EMG did not attain significance in the 5X3X4 ANOVAs (treatments X blocks X sessions within blocks). No main or interaction effects were present for the 3 EMG variables. The average reduction in EMG over the 720 sessions monitored was 1.1 microvolts, or about a 1/6 decline from the minute 1 mean.

Forehead Temperature

Minute 1 and within session change values for forehead temperature were non-significant in the ANOVAs, but minute 15 forehead temperature decreased significantly across blocks (Table 4, $F(2,110)=7.6$, $p < .001$), suggesting that volunteers had less forehead blood flow at the end of each

lab session as the experiment progressed. The mean forehead temperatures across blocks were 33.23°C, 32.87°C, and 33.0°C. In the pair-wise comparisons performed with Duncan's MRT (1955), accepting throughout $p<.05$, blocks 2 and 3 were significantly cooler than block 1 (blocks 1 versus 2 observed difference, OD=.37, critical difference, CD=.197; blocks 1 versus 3, OD= +.24, CD=.186). Blocks 2 and 3 did not differ significantly (OD=.13, CD=.197). Although the decrease in minute 15 forehead temparature across blocks was statistically significant, in absolute terms it was relatively small. The decrease did not exceed .37°C or about 1% of the block 1 mean. Cell data is in Appendix VII.

Hand Temperature

The ANOVAs did not attain significance for minute 1, minute 15 or within session change values of hand temperature. The mean within session change was +1.6°C. Mean hand temperature increased within sessions in all 60 cells (5X3X4) of the experimental design, although individual subjects would occasionally decrease their hand temperature during a session. Clearly, subjects did not learn voluntary control of hand temperature. Cell data is in Appendix VIII.

Headache Diary: Frequency

The number of headaches per week decreased significantly across blocks (Table 5, blocks $F(2,110)= 8.18$, $p<.0008$) but was unaffected by treatments or sessions within blocks. The mean number of headaches per week across blocks was 1.85, 1.50 and 1.49, again with the last two blocks being significantly lower than block 1 (block 1 to block 2 OD= .35, CD=.20; block 1 to block 3 OD=.37, CD=21; block2 to 3 OD=.10, CD= .20, non-significant, NS). The overall decrease in the weekly number of headaches from block 1 to block 3 was 20%. Cell data is in Appendix IX.

Intensity

Intensity, like frequency, decreased significantly across blocks but was unrelated to differential treatments or sessions (Table 6, blocks $F(2,110)=.77$, $p<.001$). The block means for intensity, rated on a 1 to3, mild to severe, scale were 1.44, 1.27 and 1.12, with blocks 2 and 3 significantly lower than block 1 (block 1 to block 2 OD=.17, CD=.16; block 1 to block 3 OD=.32, CD=.17; block 2 to block 3 OD=.15, CD=.16, NS). The mean overall decrease in rated intensity was 22%. Cell data is in Appendix X.

Other Headache Diary Variables

The findings for headache duration and density and prescription and non-prescription drug use were congruent with those for frequency and intensity - decreases across blocks only - but did not attain significance. The effect of blocks on weekly headache duration approached significance ($F(2,110)=5.61$, $p<.005$). The mean weekly duration of headaches across blocks was 15.62, 12.72 and 12.51 hours. The long duration of pain they typically suffered each week emphasized the severity of the volunteers' migraines. While the decline of 3 hours in mean weekly head pain from block 1 to block 3 was statistically non-significant, it represented a 20% decrease from baseline.

Headache density was simply the duration of each headache times its 1 to 3 intensity rating, summed each week. Since density was directly related to intensity and duration, it too declined somewhat across blocks but did not change systematically with treatments or sessions. The mean densities across blocks were 32.26, 26.83 and 25.38, representing an overall decrease of 21%

Both prescription and non-prescription drug use tended to decline over the course of the study, although no significant effects for treat-

ments, blocks or sessions were present in the ANOVAs. The mean number of prescribed tablets consumed per week decreased across blocks from 2.95 in block 1 to 2.15 in block 2 and 2.20 in block 3. Thus, prescription drug consumption declined by 25% over the course of the study. Non-prescription drug use declined by 20% over the course of the study, from a mean of 3.08 tablets per week in block 1 to 2.73 in block 2 and 2.54 in block 3.

The results of the 5X3X4 (treatments X blocks X sessions within blocks) ANOVAs of the physiological and headache diary variables can be summarized as follows. The 5 treatments did not differentially affect significantly any of the 15 dependent measures. Sessions within blocks did not systematically affect any of the 15 dependent variables, nor did any of the possible interactions attain statistical significance for any of the variables. Over the course of the study, however, the end of session (minute 15) head temperature, and the frequency and intensity of headaches decreased significantly.

Analysis of Variance of the McGill Pain Questionnaire: Experimental Effects

Experimental effects on the 14 MPQ subscales were analyzed in a 5X2 (treatments X pre/post) ANOVA. Each variable was assessed at an alpha level of .003, to maintain an analysis-wise Type 1 error rate of .05. Neither the treatment effects, nor the pre/post effect, nor the interaction attained statistical significance for any of the 14 subscales. Nearly significant trends for a pre-to-post effect on the "temporal" ($F(1,55)=8.00$, $p<.007$) and "fear" ($F(1,55)=8.78$, $p<.005$) subscales did, however, appear. The subjects' mean characterization of their pain as "temporal" during baseline was 2.9 on a 1 to 5 scale, and 2.7 after the study. The mean characterization of the headaches as "fearful" was 3.0 during baseline and 2.7 after the study. No treatment or interaction effects

Table 7

Varimax Factor Matrix of the Physiological and Diary Variables Based on Data From All 3 Blocks

Measure	Factor					
	1	2	3	4	5	6
Minute 1 EMG	-.025	-.022	-.118	.435	.876	.033
Minute 1 Head Temperature	-.065	-.232	.835	-.029	-.024	.350
Minute 1 Hand Temperature	-.019	.969	.086	-.012	-.069	.118
Minute 15 EMG	-.070	-.034	-.045	.869	.110	-.185
Minute 15 Head Temperature	.055	.220	-.023	-.069	.013	.930
Minute 15 Hand Temperature	.066	.015	-.313	.025	.006	.134
EMG Change	-.005	.077	.011	.269	-.911	-.038
Head Temperature Change	-.021	.083	-.822	.059	.014	.253
Hand Temperature Change	.098	-.471	-.576	.049	.138	.020
Frequency	.890	-.059	.055	.067	-.021	-.045
Intensity	.670	-.036	-.155	-.136	.652	.119
Duration	.885	-.006	.095	.361	-.079	.050
Density	.892	-.004	-.024	.204	-.037	.076
Prescription Medication	.691	.276	-.070	-.275	.077	-.155
Non-prescription Medication	.420	.050	-.087	.665	-.153	.176

approached significance. Thus, the trends in the MPQ experimental results were in keeping with the findings on the physiological and headache variables: improvement occured over time, unrelated to treatment.

The overall pattern of experimental results on the physiological diary and MPQ variables suggested a non-specific "placebo" effect that, while beneficial to many of the participants, did not support the unique effectiveness of relaxation training with or without hand temperature biofeedback in alleviating migraine. The ramifications of these findings and their relationships to descriptive findings (personality, symptom, and pain questionnaires) were examined by further analyses.

Factor Analysis of Physiological and Diary Variables Across All 3 Blocks

One interesting feature of the results was the significant decrease in both headaches and minute 15 head temperature across blocks. At first examination these findings suggested that possibly lower forehead temperature was related to decreased head pain. Such a finding would be in keeping with the well established observation that dilatation of arteries at the head causes pain (Dalessio, 1972). However, under further analysis the end of session forehead temperature and headache frequency and intensity proved to be unrelated.

Insert Table 7 about here

Minute 15 forehead temperature correlated negligibly with the number of headaches and their mean intensity each week (Pearson r=.02 and .12 respectively). Secondly, factor analysis of the 15 physiological and headache diary variables across all 3 blocks clearly separated the two types of measures (Table 7). The first rotated factor (varimax criter-

Table 8

Varimax Factor Matrix

of the

McGill Pain Questionnaire Subscales

SCALE	FACTOR 1	FACTOR 2
Temporal	.63167	.22246
Spatial	.35572	.55716
Punctate	.54169	.63907
Incisive	.23593	.71592
Constrictive	.35055	.75246
Traction	.25020	.75596
Thermal	.02173	.80118
Brightness	.11714	.75225
Dullness	.62243	.42043
Tension	.74543	.09042
Autonomic	.67908	.21388
Fear	.52957	.19124
Punishment	.78702	.30460
Evaluative	.83002	.16969

Table 9

Varimax Factor Matrix of the Headache Symptom Questionnaire

ITEM	FACTOR 1	FACTOR 2	FACTOR 3	FACTOR 4	FACTOR 5	FACTOR 6	FACTOR 7	FACTOR 8	FACTOR 9	FACTOR 10
1	-.03553	-.01710	.04432	-.07762	.02067	-.11100	.07154	-.05582	-.10809	-.03656
2	.26468	.05565	.27010	.07704	.06084	.16541	.15674	-.27672	-.17643	-.23473
3	-.20873	-.12821	.15202	-.06612	.09617	.12927	-.06344	.20569	-.22351	.15459
4	.28008	.60974	-.08317	-.08212	-.17558	.11590	-.29999	-.04467	-.06229	-.27598
5	-.16147	-.12026	-.04860	.18589	-.77434	-.04670	-.23506	.00855	-.15071	.04393
6	.14614	-.09173	.04214	-.16499	-.03305	.27621	.21011	-.30009	-.10760	-.07102
7	.08951	-.05771	.05490	-.08281	.02191	.08510	-.17497	.05636	-.03028	-.45772
8	-.10388	-.03858	.37701	.18400	.12639	-.41417	.37614	.06644	-.21866	.08628
9	-.20057	-.01072	.23303	-.01496	.01564	.06690	.02169	.00174	.02207	-.06626
10	-.07661	.69017	-.04752	-.02466	-.01172	.15259	-.18786	-.09250	-.13399	.10794
11	.01922	-.00896	-.01907	-.06520	.07140	.03305	.03429	.02299	-.03319	.02711
12	.31652	.09888	.10070	.16370	.48037	-.00175	.01521	.15912	.07230	-.30971
13	-.03011	.03152	.08483	-.07060	.03704	-.03172	.13115	.12151	.16922	-.07085
14	.13940	.61950	.15550	.17790	-.02025	-.05147	.02935	.12343	-.03535	-.02079
15	.41533	.16789	-.02456	.13140	.03204	-.02281	.27071	-.17118	.52944	.20017
16	.07439	-.06992	-.06835	-.12575	.08699	.05421	.79439	.08268	.07246	.04157
17	-.08937	-.03226	-.17056	.12508	-.05567	.12049	.18075	-.24110	-.02758	-.14570
18	.19543	.26172	.68745	.22049	.04272	.27655	.18181	.19594	.23616	.32072
19	.16404	.26951	.04645	.06717	.20686	.28141	.00796	.51926	-.00583	-.26598
20	.07944	-.06671	.23274	.09768	-.17890	.17903	-.06377	.27666	-.12171	-.19290
21	.13138	-.01567	.04254	.05847	.11623	.14906	.07780	.79484	-.00916	.06178
22	.77443	.16436	.12985	-.01920	.07229	-.02720	-.02295	.30821	-.09325	-.02179
23	.65435	.09019	-.08088	.10095	-.06256	.08151	.01536	-.06495	-.09225	.00094
24	.08905	.13131	.01181	.50458	.18798	.28756	.07394	.40024	.21570	.10334
25	.08812	.00135	.14019	.19323	-.20685	.03340	.11113	.52498	-.19494	-.08902
26	.17703	-.21866	-.07103	.12263	.04667	.15678	.11255	.09232	-.05941	.08788
27	.01942	-.01346	.05285	.07194	.03299	.60047	.18366	.16161	.04365	.09668
28	.05885	.31806	-.17149	.47598	.10217	.51264	-.02549	.12599	-.05681	.14634
29	.23182	-.05836	-.11155	.37865	-.04207	.25935	.23850	.39057	.01734	.21466
30	.17910	-.04231	.04998	.19369	.02781	.31718	.14526	.24554	.20446	.18274
31	-.05953	.15486	.49804	.40416	.15454	.12422	.05476	-.27655	-.23902	.17069
32	-.04048	-.23052	.06542	.06154	-.17073	-.09589	.03564	-.02539	.10251	-.17948
33	.15670	-.12056	.11443	.62195	-.09881	.00996	-.15953	.07279	-.08261	.00456
34	-.09600	-.19754	.72594	.01307	-.04518	-.01873	.09412	.12823	.11314	-.10325
35	.18447	.04774	.75347	.14798	.15003	.02170	.04903	.10359	.04610	-.07566
36	.14842	-.18970	.74365	.13977	.08592	-.04681	-.16020	.03404	.31053	.04600
37	-.03167	-.18720	.17854	.14107	.54266	-.21223	-.16753	.01356	.10448	.28480
38	.58303	-.19163	.54303	.09316	.03814	.13387	.11784	.06472	.11344	-.04443
39	.21428	.15369	.12334	.20729	.27245	.00787	-.17436	.07321	.26172	.01399
40	-.12366	-.76961	.17744	-.03273	-.03958	.04900	-.13164	-.03972	-.13259	.07227
41	-.01260	-.53450	.10338	.32187	-.17338	-.06397	-.05151	.09433	-.12987	.02987
42	.26486	.03764	.29445	-.06296	.46430	.09712	.49241	.11116	.07526	.05395
43	-.06248	.02670	.16464	.10065	-.05226	.06694	.22722	.01141	-.02309	.02098
44	.02245	.66288	.06356	-.17482	.03803	-.09965	.10477	.23422	.09274	.07708
45	-.25775	-.09109	.13261	-.16942	.24588	.07548	.43359	-.13486	-.03056	-.13434

Table 9 Continued

ITEM	FACTOR 11	FACTOR 12	FACTOR 13	FACTOR 14	FACTOR 15
1	.06102	-.08038	.11500	.01977	.84399
2	-.12719	.09132	-.57774	.12024	.05971
3	-.15455	.14029	.13992	-.02495	-.02021
4	-.16871	.09277	.05068	.09595	-.10041
5	-.05050	-.03078	.09200	.08480	.01934
6	.20703	.06182	-.10696	.00557	.17601
7	.07166	.02008	.22830	.24972	-.04025
8	-.11413	.10538	.21205	.21215	-.00674
9	.03888	.02669	.02679	.02754	-.04690
10	.04540	-.04647	.20284	.11190	-.04501
11	-.11649	.01685	.04277	.15287	-.10764
12	-.01456	-.02993	-.10639	.31205	.16122
13	.61365	.13123	.02650	.10198	-.10999
14	.11641	.74756	-.12504	.05033	-.05219
15	.16041	.15151	.21756	-.14553	.02352
16	.08845	.01033	.04648	-.14994	.14424
17	.10876	-.01706	.07825	-.74105	-.02076
18	.42173	-.18484	-.02136	.03570	.20941
19	-.02664	.15499	.24735	.16215	.02276
20	.61663	.08179	.15995	-.20605	.31818
21	.21192	.04618	.04375	.09732	.05006
22	.00091	.22315	-.05445	.04077	-.04847
23	.05165	.07751	-.06167	.07419	.06041
24	-.11015	-.03718	-.25245	.11132	-.02391
25	-.01478	-.56504	-.02419	.08703	-.00625
26	.11099	.33331	-.56887	.26975	-.05866
27	.00017	-.01147	-.10343	-.09153	.01126
28	.08076	-.04094	.08165	-.13161	-.02766
29	.21187	.08677	.22812	-.26004	.20584
30	-.15881	.02829	.07043	.11446	.60229
31	.00682	.04149	.13730	-.13960	.13940
32	.11931	.08075	.17865	-.14285	-.23955
33	-.08061	.02778	.00222	.12956	.13334
34	.15847	.00261	-.07339	.16272	.13147
35	.04142	-.12224	.22682	.15456	-.09362
36	.09436	.24202	.08077	-.03478	.02816
37	.07127	-.43882	.03330	.00006	.16736
38	-.09681	-.34829	-.03918	.04146	.01268
39	.32646	.15550	.52253	-.04405	.17647
40	-.02622	-.02486	-.02871	-.19623	.09139
41	.09908	-.01995	.10541	.69748	-.01454
42	-.31059	.12730	.08329	.06073	.07336
43	.03939	-.04783	.01413	.08526	.10566
44	.07075	.03803	-.11232	-.26163	.22425
45	.33736	-.27258	.02496	.15907	-.15539

Table 10

Varimax Factor Matrix of the

Block 1 Physiological and Diary Means

MEASURE	FACTOR 1	FACTOR 2	FACTOR 3	FACTOR 4	FACTOR 5	FACTOR 6
Minute 1 EMG	-.03600	.19384	-.02673	.39113	-.88283	-.12448
Minute 1 Head Temperature	-.19554	-.83157	-.29082	.03061	.05065	.25444
Minute 1 Hand Temperature	.03406	-.14179	.96726	-.06070	.10962	.12060
Minute 15 EMG	-.02237	.11763	-.03836	.88765	-.20053	-.26287
Minute 15 Head Temperature	.07126	.82440	-.17974	-.11421	.02248	.94686
Minute 15 Hand Temperature	.13999	.35129	.87780	.01271	.01215	.12427
EMG Change	.07162	-.05356	.10156	.23576	.92945	-.96800
Head Temperature Change	-.03408	.79289	.16719	.03711	-.05372	.42867
Hand Temperature Change	.11313	.79290	-.01137	.16713	-.18962	-.02688
Frequency	.90267	.01214	.07324	.10768	.02826	.01621
Intensity	.61409	.27966	.11281	-.20237	.05509	.06989
Duration	.84252	-.09270	-.10590	.40054	.08959	.04878
Density	.87051	.14413	-.03315	.28437	.04210	.08156
Prescription Medication	.70318	.03028	.32245	-.35165	-.12075	-.18557
Non-prescription Medication	.40928	.00017	-.05383	.70327	.20358	.13214

Table 11

Varimax Factor Matrix of the 16PF

SCALE	FACTOR 1	FACTOR 2	FACTOR 3	FACTOR 4	FACTOR 5	FACTOR 6
A	.10291	.75939	.15721	.05805	.03840	.00164
B	.00749	-.05198	-.02124	.07508	.50903	.14746
C	-.71021	-.05911	.23518	.01577	.32476	-.05398
E	.09227	.19708	.88613	-.14615	.18952	.25871
F	-.04646	.69078	.30729	-.20681	-.02310	.10102
G	.18999	.15168	-.07016	.69038	.10245	-.08169
H	-.20548	.54638	.61537	.02308	.18105	.00535
I	.13329	.22976	-.03006	-.38937	.46968	-.35548
L	.58613	.24132	.27084	-.12622	-.11011	.30120
M	-.26384	.06724	.16335	-.57912	.42560	.11453
N	.03606	-.07615	-.58747	.15107	.15484	.03281
O	.80035	-.20133	-.00411	.02779	.14894	-.23921
Q1	.00251	-.03354	.08733	-.10886	.16967	.73390
Q2	-.01979	-.79239	.03305	-.12201	.06734	.12291
Q3	-.30142	-.06625	-.34208	.58791	.10016	-.02770
Q4	.77517	.05311	.01756	.23045	.13282	.01207

Table 12

Varimax Factor Matrix of the PRF

SCALE	FACTOR 1	FACTOR 2	FACTOR 3	FACTOR 4	FACTOR 5	FACTOR 6	FACTOR 7
Abasement	-.80229	.03795	.18759	-.06599	-.11996	.08301	.19371
Achievement	.08586	.66007	.15915	-.40344	-.05814	.25419	.27938
Affiliation	.46211	-.14371	.28875	.60878	-.16994	-.02508	.33620
Aggression	.52599	-.02535	-.02864	.06527	.58440	.16162	.09991
Autonomy	.11657	-.08394	-.09779	-.61459	.01171	.50248	-.15509
Change	-.04061	-.19196	.57041	-.07768	-.02019	.48585	.32857
Cognitive Structure	-.00851	.81922	-.07282	.15532	.20694	-.08314	-.11672
Defendence	.53008	.04183	.02206	-.09413	.59255	-.08836	-.26756
Dominance	.71668	.16181	.18078	-.29742	-.07116	.28497	.06925
Endurance	-.03522	.65274	.14314	-.14882	-.15523	.05499	.21977
Exhibition	.81472	-.11837	.25725	.19183	.02294	.09388	.04189
Harmavoidance	-.03108	.65970	-.04478	.23163	-.14586	-.60450	-.01252
Impulsivity	.17656	-.59935	.46425	-.06856	.41291	-.15370	-.05960
Nurturance	-.06992	.39411	.67803	-.05189	-.20267	-.29396	.00492
Order	.04746	.52784	-.08325	.16172	-.10269	-.42112	.01482
Play	.25472	-.32921	.69929	.18513	.67225	.25284	.01028
Sentience	.06175	.14379	.79722	.18994	-.00162	.20552	-.03600
Social Recognition	.26883	.49735	.12906	.38766	.59337	-.03440	-.06660
Succorance	.02390	-.01789	.10188	.90490	.09633	-.11088	-.09800
Understanding	.13779	.12270	.15521	.91238	-.20269	.77193	-.10815
Infrequency	-.07132	.18624	.00508	.03657	.12519	-.09960	.85766
Desirability	.17079	.14960	.12100	.02221	-.78960	.00770	-.19632

Table 13

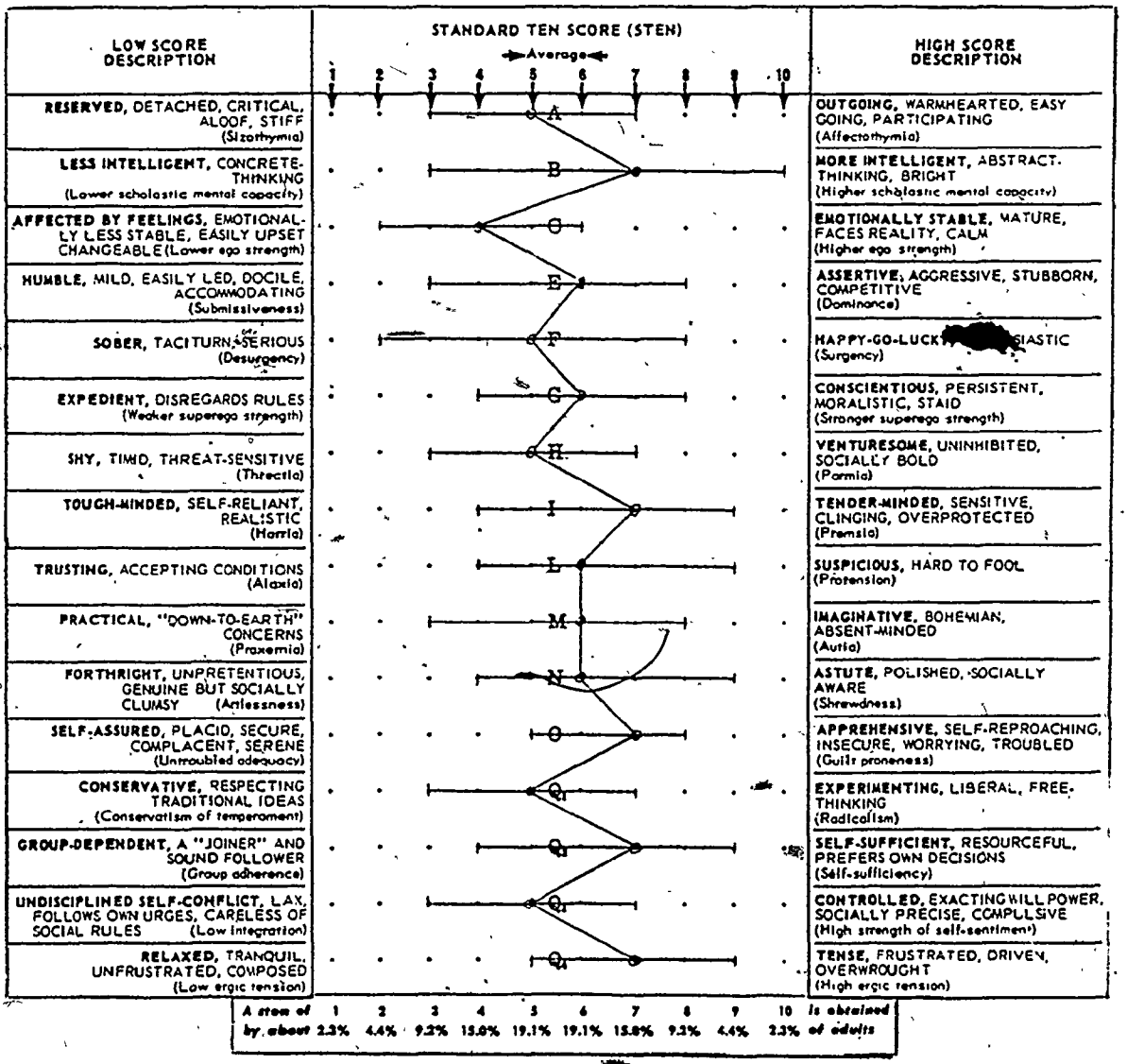
Varimax Factor Matrix of the DPI

SCALE	FACTOR 1	FACTOR 2	FACTOR 3	FACTOR 4	FACTOR 5	FACTOR 6	FACTOR 7
Insomnia	.55717	.05212	.11158	.01700	.47422	.02948	-.13953
Headache Proneness	-.10022	-.10804	.21828	.31354	.47514	.14532	.08245
Brooodiness	.41398	.10079	.55180	.34520	.14586	.46857	.03579
Cynicism	-.06109	.23264	.80371	-.06074	.09222	.14363	-.16191
Depression	.15612	-.15703	.72767	.19118	.27669	.05912	.06404
Desocialization	.19864	-.07679	.51199	.04010	.07417	.54887	.17264
Disorganization of Thinking	.50498	.01108	.06594	.11202	.40673	.08020	.30869
Famial Discord	.06671	-.03164	.25262	.26372	.15377	.56713	-.19151
Feelings of Unreality	.46911	-.02698	.37039	.11328	.15798	.51906	.34096
Health Concern	.50279	-.03816	.07718	.20522	.45022	.27945	-.04986
Hostility	.66194	.47596	.28580	.23664	-.02771	-.00526	.03596
Hypochondriasis	.11163	.10286	.25907	-.12344	.74592	.27159	.06374
Ideas of Persecution	.43299	.02981	.64925	.15003	.28254	.25901	.01097
Impulsivity	-.04255	.53217	-.04283	.01875	.07598	-.42364	.43102
Irritibility	.00855	.31837	.38225	.58766	.22803	.04238	-.29420
Mood Fluctuation	-.09096	.19029	.25768	.67912	.22694	-.03134	.20103
Neurotic Disorganization	.15253	.10345	-.00928	.14769	.21858	.02890	.61418
Panic Reaction	.22161	-.15270	.34565	.60487	.26083	.22156	.15074
Perceptual Distortion	.10360	.08231	.09985	-.07753	.33855	.06047	.08664
Rebelliousness	-.31403	.52034	.00955	.06591	-.26217	.16578	.52654
Repression	.46011	-.58035	.25613	-.16009	-.08054	-.29147	.29677
Sadism	-.17843	.68977	.13714	.13405	.20525	.01341	.10755
Self Depreciation	.48926	-.08956	.55369	.01863	.15413	.24844	.08969
Shallow Affect	-.03553	-.09523	.24563	-.83643	.19818	-.06700	-.06714
Socially Deviant Attitudes	.32248	.71881	.07046	-.21662	.06917	.05352	.17897
Somatic Complaints	.20337	.23660	.15527	.14019	.73016	.11634	.13569
Defensiveness	-.27063	-.73124	.15947	-.33880	.06188	.12056	.10231
Infrequency	.87223	.30271	.08885	-.15059	.00558	.24276	.06116

Figure 2

Mean 16PF Profile,

± 1 Standard Deviation



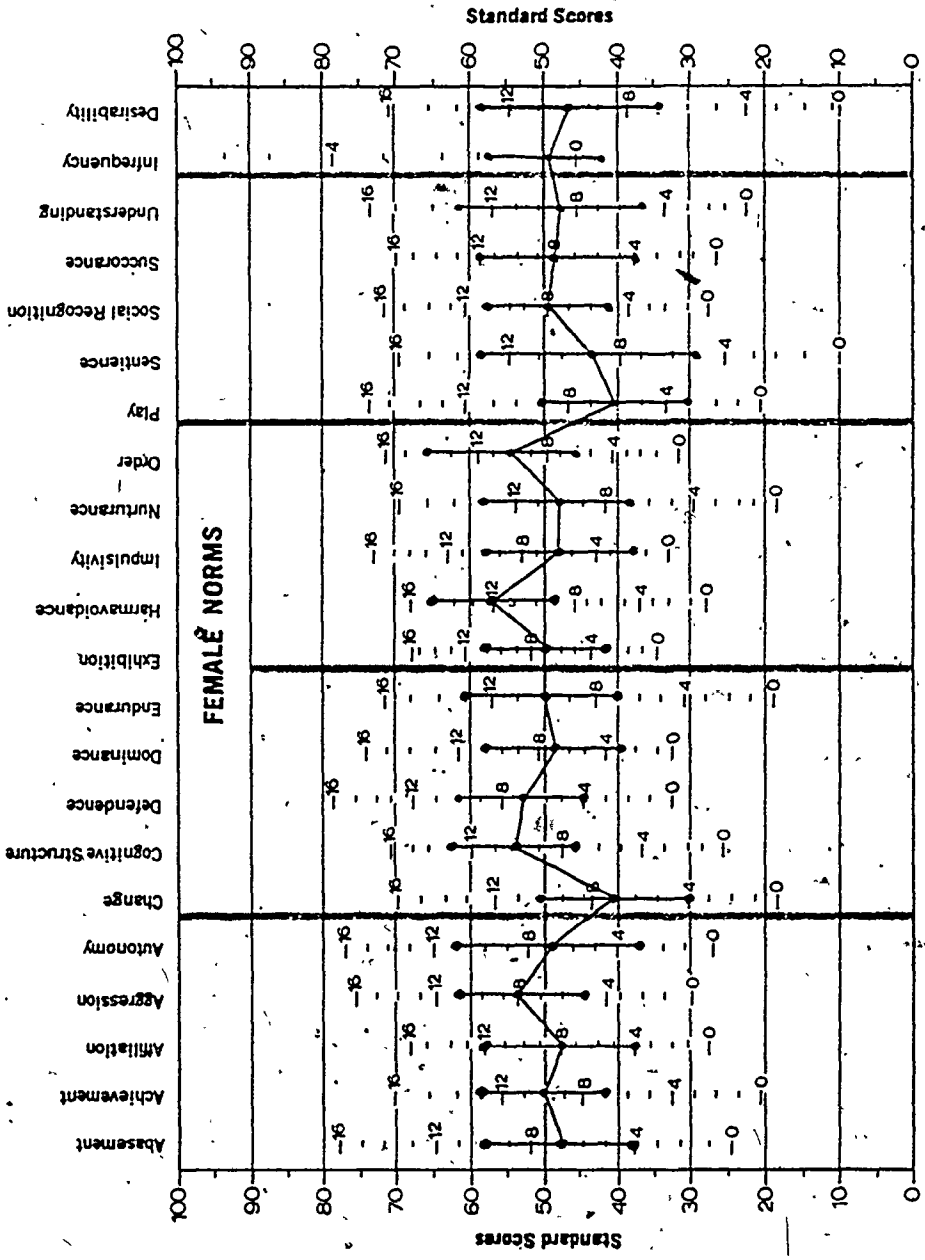


Figure 3

Mean PRF Profile, ± 1 Standard Deviation

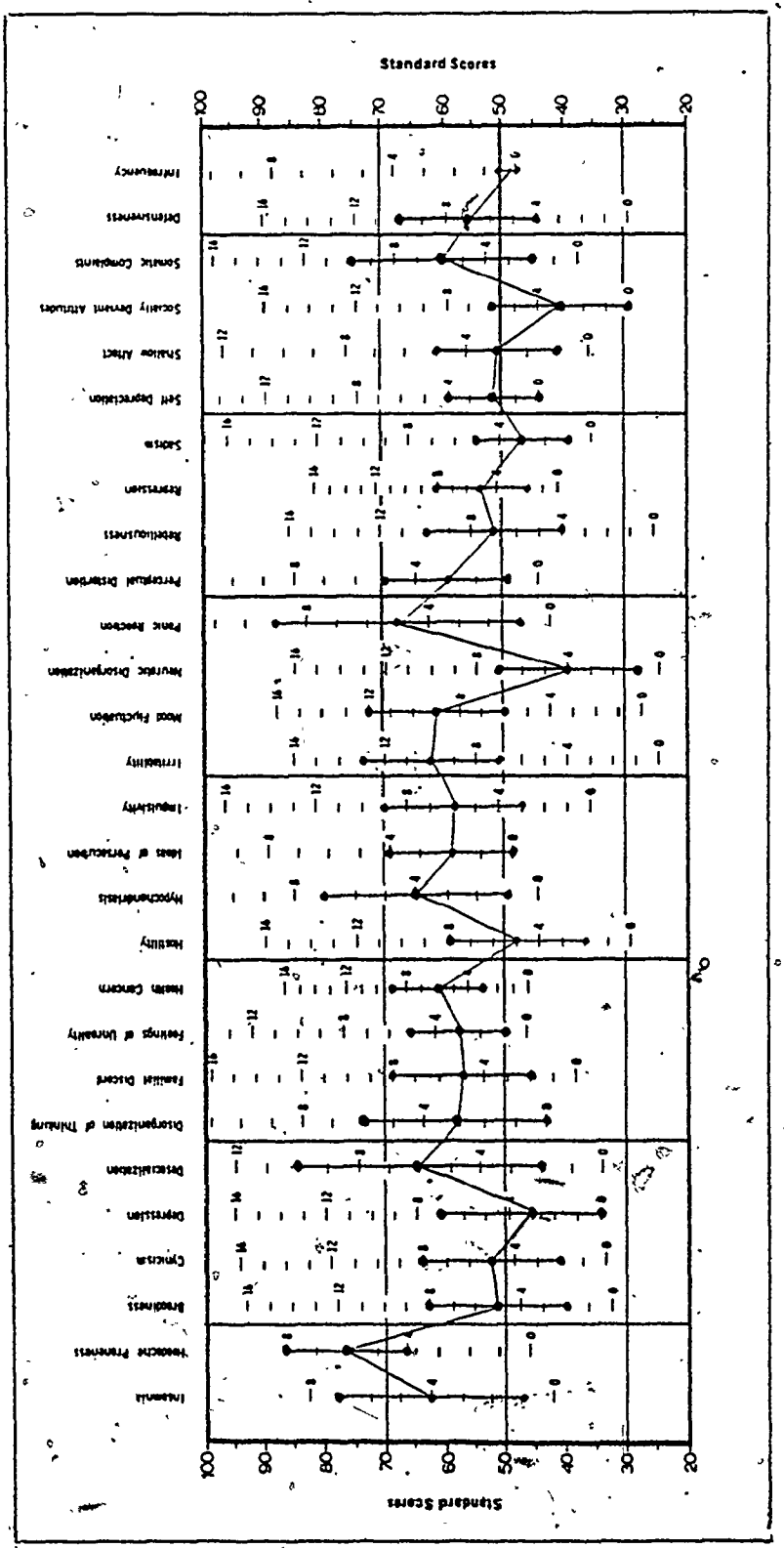


Figure 4

Mean DPI Profile, ± 1 Standard Deviation

ion) was associated with headache diary variables, while physiological measures loaded on other factors. The separation of headache diary and physiological variables was notably clear. The headache variables' loadings on the first factor averaged .74 whereas, the highest physiological loading was .10. Apparantly then, the improvement in migraine headaches across the experiment was entirely unrelated to the physiological variables measured.

Factor Analyses of Descriptive Data

The MPQ test subscales, Symptom Questionnaire, 16PF, PRF, DPI and Block 1 physiological and headache diary variables were each separately factor analyzed, with factor rotation to the varimax criterion of factors with eigenvalues greater than 1. The block 1 pre-experimental baseline physiological and diary variables were considered descriptive data in the sense that they presumably represented the naturally occuring characteristics of the subjects on these variables, before intervention, affected only by measurement itself. The Varimax factor matrices from these analyses are presented in Tables 8 through 13.

Insert Tables 8 to 13, and Figures 2 to 4 about here

Two rotated factors were present in the 14 subscales of the MPQ from this sample of migraine sufferers (Table 8). The factors clearly separated an affective-evaluative component of migraine pain (as described on the MPQ) from a purely sensory one. The first factor was identified most strongly with the "evaluative" and "fear" subscales of the MPQ (both loadings=.83 on factor I). Other scales with high loadings on factor I (and low loadings on factor II) were "punishment" (.79), "ten-

sion" (.75), and "autonomic" (.68). The first sensory scale, "temporal" pain, was also more related to factor I (loading=.63), than to factor II (loading=.22). The sensory factor II was characterized by high loadings with the "thermal" (.80), "traction" (.76), "constrictive" (.75) and "brightness" (.75) subscales. "Punctate" and "dullness" pain tended to be associated with both factors, although "dullness" was slightly more related to factor I than II (loading .62 versus .42). Two main components then, one affective-evaluative, and the other sensory, accounted for the way in which the volunteers described their head pain using the MPQ.

Fifteen rotated factors underlay the 45 items of the Headache Symptom Questionnaire (Table 9). These factors were probably somewhat unstable due to the small ratio of variables to subjects (45 to 60). Taken at face value, however, the simple fact that 15 factors emerged from the analysis suggested that a wide diversity of headache symptom patterns were present among the 60 volunteers.

The 15 factors represented clusterings of related headache symptoms. The symptoms best describing the 15 factors were, in descending order of loadings greater than .50: I, questionnaire items 23, 22, 38, vomiting, nausea and food triggers; II, items 40, 10, 44, not present on awakening, occuring in clusters, helped by sleep; III 35, 34, 36, occuring when under stress, tired or depressed; IV 33, 32, 24, eyes water, face flushes, ptosis; V 5, 3, 37, headaches began at low age, more than one type of headache, occur when hungry; VI 27, 7, 28, part of body becomes numb, long duration of attack, accompanied by tingling-prickling sensations; VII 16, other family members have headaches with similar characteristics; VIII 21, 19, 25, unusually sensitive to odours, miosis, aggravated by light or sound; IX 9, 15, worse during weekends and holidays; other

family members have headaches as frequently; X 11, more of head involved in pain; XI 20, hands or feet get cold; XII 14, 28, one side of face hurts, no tingling-prickling sensations; XIII 43, 26, 39, difficulty falling to sleep, nose runs during headache, stomach upset after eating; XIV 17, 41, 6, can identify causes of headaches, awoken by headaches; long history of headaches; XV 1, 30, past head injury, speech slurred during headache. These factors should be interpreted with caution. They may suggest a substantial diversity of headache "syndromes" in this particular sample of volunteer, chronic migraine sufferers.

The factor structure of the 15 physiological and headache diary variables during block 1 (Table 10) did not differ substantially from the factor structure for the same variables over all three blocks (Table 7). During the block 1 baseline factor I was entirely characterized by diary variables (except non-prescription medication) while the physiological variables formed other factors. Non-prescription medication loaded (.70) with minute 15 EMG (.85) to characterize factor IV. Factor II consisted chiefly of low minute 1 forehead temperature (loading -.83) and large within-session increases in forehead and hand temperature (both loadings .79). Factor III reflected a congruence of minute 1 and minute 15 hand temperature, loading .97 and .88 respectively. Factor V underlay within session EMG change (loading .93) and minute 1 EMG (loading -.88). This factor requires some care in interpretation, since the within session EMG change was typically negative. What the two factor loadings show is that low minute 1 EMG is related to little (i.e. zero or slightly positive) within session change. Minute 15 forehead temperature (loading .95) described factor VI.

The most interesting feature of the factor matrix of the block 1

physiological and diary variables is the clear separation of headache experience from the physiological measures. Beyond that separation most of the factors seemed to arise simply from self-evident features of the data, such as the consistency of each subject's relative hand temperature from minute 1-15 (factor III), or the basement effect of low minute 1 EMG on within session EMG change (factor V).

The mean profile of the 60 volunteers on the 16PF scales is illustrated in Figure 2. One standard deviation is marked above and below the mean profile. The factor analysis of the 16PF data rotated 6 factors (Table 11). Factor I was primarily formed by 16PF scales Q4, O, L and on the negative pole, scale C. Thus, factor I represents an underlying theme of tension, frustration (Q4), apprehension, guilt proneness (O), suspiciousness (L) and emotional lability (C). Factor II was characterized by outgoing group dependency (A, Q2) enthusiasm (F) and absence of shyness (H). Factor III was formed by expediency (G), imagination (M), lack of self-discipline (Q3) and tender-mindedness (I). Only scale B, intelligence, loaded highly on factor IV. Factor V represented forthright (N), assertive (E) and socially bold (H) personality features. Factor VI chiefly represented scale Q1, liberalism and free-thinking. The 6 factors of the 16PF appeared internally consistent. The emergence of a first factor involving tension, apprehension, suspiciousness and emotional lability suggested that considerable emotional distress was present among the volunteers. Whether this pattern of emotional distress caused, was caused by, or co-existed with migraine cannot of course be answered by a descriptive analysis.

The mean PRF profile with standard deviation is illustrated in Figure 3. The factor analysis of the PRF data yielded 7 factors (Table

12). Possible summary labels, and the scales best describing the factors (in descending order of loadings to a minimum of .50) were: I "aggressive attention seeking", Exhibition and (low) Abasement, Dominance, Defendence and Aggression; II "rigid determination to achieve", Cognitive structure, Achievement, Endurance, Order and (low) impulsivity; III "exuberance" Sentience, Play, Nurturance and change; IV "group mindedness" Succorance, (low) Autonomy, Affiliation; V "social recognition" (low) Desirability, Social Recognition, Defedence, Aggression; VI "intellectually venturesome" (low) Harmavoidance, Understanding, Autonomy; VII "infrequency" Infrequency.

The mean DPI profiles with standard deviations is illustrated in Figure 4. The factor analysis of the DPI rotated 7 factors (Table 13). Possible summary labels and the scales best describing the factors (in decending order of loadings to a minimum of .50) were: I "hostility/confusion", Infrequency, Hostility, Insomnia, Disorganizaion of thinking, Health Concern; II "social deviance", (low) Defensiveness, Socially Deviant Attitudes, Sadism, (low) Repression, Impulsivity, Rebelliousness; III "depression", Cynicism, Depression, Self Depreciation, Ideas of Persecution, Broodiness, Desocialization; IV "emotional reactivity" (low) Shallow Effect, Mood Fluctuation, Panic Reaction, Irritability; V "bodily concern", Hypochondriasis, Somatic Complaints; VI "perceptual distortion" Perceptual Distortion, Familial Disorder, Desocialization, Feelings of Unreality; VII "disorganization, Rebelliousness.

Overall, the factor analysis of the MPQ, Symptom Questionnaire, block 1 physiological and diary variables, 16PF, PRF and DPI successfully reduced the measures to a smaller number of underlying factors. Typically these factors were readily interpretable. The factors of the Symptom

Questionnaire should be considered cautiously, since the number of variables entering that analysis was large relative to the sample size. The rotated factors that emerged from the factor analysis were used to obtain factor scores for final analyses of the descriptive data by multivariate discriminant function analysis (MDFA).

Multivariate Discriminant Function Analysis

Since there was significant improvement in the study, and some people improved but not others, an attempt was made to discover any variables that distinguished improvers from non-improvers. To do this the change in headache frequency from block 1 to block 2 was used as an index of improvement. Fifteen subjects had more than a 50% decrease in headaches from block 1 to block 2 and were termed the "best improvers". Twenty subjects with from 1% to 50% improvement were the "moderate improvers", and 25 subjects whose headaches did not change or got worse were the "non-improvers". This division was not due to regression toward the mean: the block 1 mean headache frequency did not differ significantly among the 3 groups (3 level, one way ANOVA, $F(2,177)=1.07$, $p > .5$, NS).

Each set of factor scores - from the MPQ, Symptom Questionnaire, block 1 pysiological and diary variables, 16PF, PRF and DPI - were analyzed in separate MDFA's. The Headache Questionnaire and DPI factors significantly discriminated the 3 improvement groups. The factors from the MPQ, block 1 variables, 16PF, and PRF did not significantly discriminate the 3 groups, although trends for some of the factors in these non-significant MDFA's tended to corroborate the stronger findings in the significant MDFA's

As mentioned in the introduction, MDFA separates known groups on the basis of a set of dependent variables. MDFA is analogous to factor

analysis, but is applied to a matrix resembling a series of F-ratios (i. e. ratios "of terms representing within groups and among-groups variation" Veldman, 1967, p. 270). The MDFA is also constrained to producing factors which maximally separate the pre-defined groups. The vectors (discriminant axes) obtained from these operations are also analogous to factors in that they are independent and define a multidimensional (discriminant) space.

> The nature of this space, however, is such that when points representing the groups and located within it, these points are separated from each other to a maximum degree.
> (Veldman, 1967, p. 270)

The position of each subject in this space can also be established by calculating his discriminant score. The discriminant score is the weighted sum of the subject's scores on the dependent variables, the weights having been established in determining the maximal separation of the groups.

For analytical purposes, however, another feature of MDFA is particularly important: each dependent variable can be correlated with each discriminant axis. These discriminant loadings are used to interpret the discriminant space. For example, if group 1 in an MDFA is positioned near the end of discriminant function I, dependent variables loading highly on function I also describe subjects in group 1. The unique contribution of each dependent variable to the separation of the groups can be assessed by univariate F-ratios, once the significance of the overall multivariate separation of the groups has been established.

Typically an intermediate stage of significance testing is also performed between the overall multivariate assessment and each variable's univariate F-ratio. Each discriminant function is tested for its indi-

vidual contribution to group separation. If for instance three groups were significantly separated in the space defined by two discriminant axes, the first axis could account for the largest proportion of the variance separating the groups. If the second discriminant axis did not contribute significantly to group separation, the variables correlating primarily with it would be much less informative for describing the separation of the groups than those loading primarily on the first axis. Three steps then, are taken in the significance testing of an MDFA. First, the overall multivariate separation of the groups is assessed. Then the significance of each discriminant axis is tested. Finally, individual variables are examined by univariate F-ratios.

Following the recommendations of Olsen (1976) all MDFA's were tested for overall significance using the Pillai-Bartlett V test (Pillai, 1969; Timm, 1975). This test is notably resistant to inflation of the Type I error rate by violations of the assumptions of normality and homogeneity of variance-covariance. The reduction of the number of variables entering the MDFA's (by the prior factor analysis) also tends to reduce the risk of type 1 error (Olson, 1976, p. 583). Thus, the V test provides a robust test statistic sufficiently powerful to detect true effects, but not apt to generate Type 1 errors. All 6 MDFA's were tested at an alpha level of .05. The two MDFA's attaining significance proved to be also significant at $p < .01$. If an MDFA significantly separated the 3 groups, the significance of each latent root (i.e. discriminant function, discriminant axis) was tested by Rao's chi-squared test (Rao, 1952; see Woodward & Overall, 1975). The significance of each (factor) scale's contribution to the separation of the groups was assessed by univariate F ratios.

The MDFA revealed that the fifteen factors of the Headache Questionnaire significantly separated the three improvement groups (Critical V, CV=.70, Observed V, OV= .81). Only the root corresponding to the first discriminant function, accounting for 66% of the explained variance, was statistically significant ($x^2(16)=33.97$, $p < .006$). Three of the fifteen symptom questionnaire factors contributed significantly to discriminating the three groups; Factor II, $F(2,57)=8.11$, $p < .001$; Factor III, $F(2,57)=3.07$, $p < .05$; Factor VIII, $F(2,57)=3.83$, $p < .03$. The factor score means for group 1 (best improvers), group 2 (moderate improvers) and group 3 (non-improvers) were respectively; Factor II, .81, -.25, -.28; Factor II, 0, .40, -.32; Factor VIII; .50, .06, -.35. Factors II and VIII loaded primarily with the first discriminant function (.65 and .48 respectively). Factor III was associated with the second discriminant function (.52). Since the first discriminant function accounted for 66% of the explained variance (and only it attained significance), the separation of the improvement groups by the Headache Questionnaire factors was best accounted for by Factors II and VIII. Factor II represented headaches that were helped by sleep, were not present on awakening, and occured in clusters. Factor VIII was described by the symptoms of enchanced sensitivity to odours, aggravation by sound and light, and miosis. Group one scored highest on Factor II, while group 2 and 3 scored equally low. The scores on Factor VIII decreased consistently from group one to group three. Thus, the MDFA of the Headache Questionnaire factors shows that the volunteers who best improved had headaches that were helped by sleep, were not present on awakening and tended to occur in clusters (Factor II). The best improvers were unusually sensitive to odours, light and

sound and reported miosis during their headaches. The non-improvers were not characterized by these neurological signs.

A further trend in the Questionnaire data, represented by Factor III, was of interest when considered in conjunction with the DPI results. Factor III reflected headaches that were precipitated by fatigue, stress, or depression. The factor scores for groups one to three were 0, .40, and -.32 respectively. Thus, according to the volunteers' self-report, stress precipitated headaches occasionally for the best improvers, more frequently for the moderate improvers, but seldom for the non-improvers

The MDFA of the DPI factors also showed significant separation of the three groups (CV=.38, OV=.61). Both latent roots attained significance (root I, $X^2(8)=18.05$, $p<.02$; root II, $X^2(6)=12.80$, $p<.05$). Root I accounted for 60% and root II 40% of the explained variance. Two of the 7 DPI factors contributed significantly to separating the three groups, Factor III ($F(2,57)=3.24$, $p<.05$) and Factor IV ($F(2,57)=3.28$, $p<.04$). Factor II approached significance ($F(2,57)=2.57$, $p<.07$). The factor score means for groups 1, 2 and 3 respectively were: Factor III, .26, .28, -.37; Factor IV, .49, .03, -.32; Factor II, -.21, .42, .21. Factor III was mainly associated with the first discriminant function (loading .53) while Factor IV was associated with the second discriminant function (loading .62). Nearly significant Factor II correlated most highly with the first discriminant function (loading .51: This was the only other loading between factors and discriminant functions greater than .50).

Factor III was a depression factor characterized by the DPI scales Cynicism, Depression, Self Depreciation, Ideas of Persecution, Broodi-

ness and Desocialization. The best and moderate improvers (groups one and two) scored high on this factor, while the non-improvers (group three) scored low. Factor IV was an emotional reactivity factor characterized by the DPI scales (low) Shallow Affect, Mood Fluctuation, Panic Reaction, and Irritability. The best improvers scored highest on this factor, the moderate improvers in between and the non-improvers lowest. Factor II was a social deviance factor underlying the DPI scales (low) Defensiveness, Socially Deviant Attitudes, Sadism, (low) Repression, Impulsivity and Rebelliousness. The moderate improvers scored high on this factor, while the best and non-improvers both scored low.

The MDFA of the DPI factors described the best improvers as showing signs of moderate depression, and high emotional reactivity. The moderate improvers appeared moderately depressed, somewhat emotionally reactive, but high in social deviance. The last observation possibly suggests a type of bitter anger directed against society at large or other individuals. The non-improvers were lowest on all three factors, depression, emotional reactivity and social deviance. Thus, the non-improvers were characterized by an absence of psychological distress. The best improvers showed some psychological distress in that they were emotionally reactive and moderately depressed. The moderate improvers were the most psychologically distressed, since they were both moderately depressed and angry or "socially deviant".

The MDFA's of the MPQ, block 1 physiological and headache diary variables, 16PF and PRF did not attain significance. The critical ($p<.05$) and observed Pillai V's for the four MDFAs were: MPQ, CV=.16, OV=.11;

block 1, CV=.34, OV=.22; 16PF, CV=.38, OV=.25; PRF, CV=.38, OV=.33. Three factors within the non-significant MDFAs could be considered trends since their univariate F ratios were significant at $p < .05$. These trends are not strictly interpretable. They tend, however, to support the significant MDFA results found on the Symptom Headache Questionnaire and DPI factors, and are reported simply to show that any non-significant trends in the data were congruent with the significant findings.

The first factor of interest, Factor II of the block 1 physiological and headache diary variables ($f(2,57)=3.22$, $p < .05$) showed that group 2 had the least forehead and hand temperature change within sessions, and a high minute 1 forehead temperature. The mean factor scores for groups 1 to 3 were: .11, -.44, and .28 respectively. Perhaps group 2 could be considered the "hot-heads" of the sample. Factor I of the 16PF data also attained univariate significance ($F(2,57)=5.06$, $p < .01$). Groups 1 and 2 scored moderately high on this factor, with group 1 slightly higher than group 2, and group 3 scored quite low. The factor score means for groups 1 to 3 were .39, .27 and -.45 respectively. Factor I of the 16PF represented a theme of tension, frustration, apprehension, guilt proneness, suspiciousness, and emotional lability. Thus, the univariate trend for Factor I in the MDFA suggests the preseence of psychological distress in the best and moderate improvers, and its absence in the non-improvers. Similarly, a univariate trend for PRF Factor V underscored the notion of "social deviance" or bitterness among the moderate improvers. The means for groups 1 to 3 on Factor V of the PRF ($F(2,57)=3.47$, $p < .04$) were .02, .41, and -.35 respectively. Factor V was described by the PRF variables (low) Desirability, Social Recognition, Defendence and Aggression.

This "asocial recognition" or "chip-on-the-shoulder" factor was similar to the "social deviance" Factor II of the DPI that tended to discriminate the groups in the same way. "Asocial recognition" strongly characterized the moderate improvers, the best improvers only somewhat, and the non-improvers hardly at all. Thus, trends in the non-significant MDFAs were congruent with the statistically significant findings. The best and moderate improvers were tense, apprehensive and emotionally labile. The moderate improvers were also salient on a factor of "asocial recognition". The non-improvers scored lowest on these factors of psychological distress.

Taken as a whole, the MDFAs indicated some psychological problems among the best improvers, a slightly more disturbed pattern among the moderate improvers, and an absence of psychological distress among the non-improvers. The best improvers were more depressed and emotionally reactive than the other two groups. The moderate improvers were both depressed and "socially deviant", the latter probably reflecting a form of bitter, anti-social anger. The moderate improvers also tended to report that fatigue, stress or depression precipitated their headaches. The best improvers had headaches which were helped by sleep, were not present on awakening, and tended to occur in clusters. The best improvers also reported being unusually sensitive to odours, light and sound and having constricted pupils (miosis) during their headaches. The MDFAs suggested that emotionally distressed subjects improved from Block 1 to Block 2 of the study, while emotionally well adjusted subjects did not.

DISCUSSION

The results of the present study do not support the specific effectiveness of autogenic training with or without hand temperature biofeedback for the treatment of chronic migraine. The mean reported

frequency and intensity of the volunteers' headaches decreased during the study, but this decrease was unrelated to treatment. The improvement was almost certainly not due to the passage of time, since subjects typically reported long histories of severe headaches. Improvement was not related to learned physiological control: the physiological training procedures (autogenic phrases and biofeedback) did not systemmatically affect finger temperature, the training parameter. The only systemmatic physiological change to occur was a decrease across blocks of minute 15 forehead temperature. Subsequent analysis revealed that this measure too was unrelated to the reduction in headaches. The improvement in the "attention/placebo" control group did not differ significantly from the improvement in the other 4 groups. The most reasonable conclusion appears to be that improvement was due to a non-specific "placebo" effect.

Changes in hand (and forehead) skin temperature did not mediate the reduction in migraine, contrary to hypotheses made in the past (e.g. Sargent et al, 1973). Conceivably improvement was due to change in a physiological parameter not monitored in the present study. (In the strictly reductionist sense this must of course be true.) Further research is needed to identify which parameter(s) might be important. Some suggestions have already been made. In particular, direct measurements of temporal artery activity by the pulse volume technique of Friar and Beatty (1976) or its variations (Bild, 1976; Feuerstein et al, 1976) appears to be a promising approach. Biochemical techniques such as analysis of plasma dopamine beta hydroxylase (Kentsmith et al, 1976) may also be able to link symptomatic and

physiological change. Nonetheless, on the basis of the present study changes in hand or forehead skin temperature do not appear necessary for migraine to improve.

Since physiological learning did not occur, the argument could be made that the present study did not in fact test voluntary hand warming as a migraine treatment. If the volunteers in the first two treatment groups had learned hand warming perhaps its specific benefits would have become apparant. The research to date however does not sustain such an argument. Only 4 of the 18 studies using hand temperature biofeedback actually monitored hand temperature. (See Table 2: This is in itself somewhat astonishing since the use of hand warming biofeedback for migraine presumably implies that increased hand temperature is related to decreased headaches.) The majority of studies to date do not offer data pertinent to the mediation issue. Of the studies that did measure hand temperature, data was not available from one (Beasley, 1976). Turin and Johnson (1976) though, found small hand temperature changes, usually in the appropriate direction, during training in hand warming and cooling. Headache improvement occured during the warming phase of treatment in their 3 systematic case studies. Two cases reported by Wickramaskera (1973) also showed within-session increases in hand temperature as their migraines improved. The data from Turin and Johnson (1976) and Wickramaskera (1973) appeared to offer some weak support for the mediation of headache improvement by hand warming. However Thompson's (1977) more systematic study contradicts the earlier observations. In Thompson's (1977) study autogenic feedback increased hand temperature more than no feedback, but the improvement in migraine was equivalent between

the two groups. Headache improvement was unrelated to finger temperature change. Thompson (1977) concluded that "support cannot be claimed for the effectiveness of feedback" (p. 3636-B). The present study then, rather than failing to replicate other studies, may in fact be representative of their actual (but unexamined) physiological processes. This view is in keeping with Taub's (Note 15) summary of a symposium entitled "Some methodological issues in the training of self-regulation of skin temperature" held at the 1975 annual meeting of the Biofeedback Research Society. "The experimenters in this symposium report varying degrees of success in obtaining operant control of skin temperature" (p. 103).

Data presented by Friar and Beatty (1976) also supports the hypothesis that migraine improvement is unrelated to finger temperature increase during biofeedback. Friar and Beatty (1976) found the largest symptomatic improvement in their temporal artery constriction group. Finger blood flow, and therefore finger temperature, also decreased during temporal artery constriction. Apparantly finger temperature decreases can also be associated with migraine improvement.

The present study and some earlier reports suggest that during biofeedback training symptomatic improvement in migraine can occur in the absence of hand temperature warming. These findings contradict the sympathetic mediation hypothesis put forward by Sargent et al (1973). Sargent et al (1973) theorized that migraine was caused by excessive sympathetic arousal. Since sympathetic activation constricts finger blood vessels, making the fingers colder, hand temperature was used as an index of sympathetic arousal. Hand warming training was thought to be a technique for reducing sympathetic arousal, and thereby

migraine symptoms. The present study fails of support Sargent's et al (1973) theory. Hand temperature was unrelated to improvement. The hypothesis of an overall placebo effect seems to best account for the data.

The placebo effect consists of several features, such as demand characteristics (Orne, 1962), attention or the "Hawthorne effect" (Dalessio, 1974; Roethlisber & Dickson, 1939), and the "effect of the doctor (or experimenter) himself" (Shapiro, 1960, p.130). The number of reported migraines declined simply during self-monitoring in an epidemiological study by Waters and O'Connor (1971). The decline may have reflected increasing disinterest by the participants, a direct self-monitoring effect (Zimmerman and Levitt, 1975) or perhaps even "compliant responses arising from the therapeutic context" (Evans, 1974, p. 296). Further research specifically geared to the placebo issue is necessary to separate the relative effects of the components.

Not all subjects respond equally to placebo. Jellinek (1946) conducted a very careful examination of the placebo effect in a trial of analgesics among frequent headache sufferers. His findings were remarkably similar to those of the present study:

> The success rate of .52 on placebo was due to 120 out of 199 subjects. No relief whatever was reported by 79 subjects although they had three to ten headaches treated with placebo. Examples of the rare U shaped distribution are seen here. Thus there are individuals who definitely tend to respond and individuals who definitely do not tend to respond to placebo. This difference in response to placebo must reflect a difference in the nature of headaches. The sample is drawn from at least two broad populations of sufferers from headaches. If subjects never report relief through a pharmacologically inactive substance but always report at least some attacks relieved through bona fide analgesics, it must be assumed that they represent a "pure culture" of physiological headaches not accessible to suggestion, while the 120 subjects who either always or most of the time respond to placebo

> represent, perhaps predominantly, psychogenic headaches and to some extent also milder physiological headaches coupled with a tendency toward suggestibility. (p. 288)

Several studies have identified personality variables that distinguish placebo responders from non-responders. Gliedman (1958) found placebo responders to be more depressed and suggestible than non-responders. Fisher and Dlin (1956) described platebo responders as mildly neurotic. High anxiety was also a typical feature (Evans, 1974; Tibbets & Hawkings, 1956). Non-responders have been characterized as more rigid and emotionally controlled (Lasagna, Mosteller, Von Felsinger & Beecher, 1954), and somewhat hostile and antagonistic toward authority (Muller, 1965). In what appears to be the most pertinent study to date Shipman, Greene & Laskin (1974) found that, based on MMPI scores, "hypochondriacally oriented depressives" (p. 481) with myofascial pain dysfunction consistently experienced reduced pain with placebo treatments. Patients who did not improve to placebo tended to by psychopathic. The placebo effect was enhanced in neurotics when it was accompanied by a strong suggestion of effectiveness. Depression and anxiety apparantly characterize placebo responders, while emotional control and hostility to authority interfere with the placebo effect.

In the present study the best, moderate and non-improvers were separated (independently of their original treatment groups) and analyzed by MDFA. The results of the MDFA were congruent with the earlier descriptions of placebo responders. The best improvers were notably depressed and emotionally over-reactive (i.e. probably "anxious"). The moderate improvers were also depressed, but more "socially deviant" or angry at life and society, and less emotionally reactive. The non-im-

provers were least depressed, emotionally reactive and "socially deviant". Depression and emotional reactivity in the two improvement groups may have mediated a non-specific, placebo effect, while the anti-social anger ("psychopathy") of the moderate improvers could have attenuated their placebo improvement. The non-improvers were emotionally stable, in keeping with earlier findings on placebo non-responders. The congruence between the personality features characterizing the improvement groups in the present study and the personality features found in placebo responders further supports a placebo effect interpretation of the results.

More generally, improvers were characterized by emotional distress, while non-improvers were not. Emotional distress was relatively mild in the best improvers, but more marked in the moderate improvers. Thus, a unimodal improvement profile was present, from the least emotionally distressed members of the total sample, who did not improve, through somewhat distressed individuals who improved the most, to the most distressed volunteers, who improved moderately. Migraine sufferers who are mildly emotionally distressed seem to be most apt to benefit from participation in a largely autogenic and biofeedback based research project. Since the improvement was independent of the physiological changes allegedly mediating it, but was related to emotional and symptomatic variables, those variables may describe migraine sufferers who are sensitive to the placebo effect.

Even if the placebo effect is the main cause of improvement in the present study, the development of predictors of that improvement still has clinical utility. Hubbe (1975) emphasized the need to "establish what it is that differentiates a patient who responds to a given treat-

ment from one who does not" (p. 96). Clinicians may want to use biofeedback, self-monitoring and autogenic phrases for their placebo effect in the treatment of migraine until better techniques are developed.

Even apparatus has been claimed to have a placebo effect. Lipkin, McDevitt, Schwartz & Duryee (1945), treating patients with Raynaud's syndrome, noted:

> Suggestion in the form of impressive but pharmacologically impotent apparatus with or without additional verbal suggestion of impending therapeutic benefit caused pronounced subjective and slight objective improvement in the majority of vasospastic disorders studied. (p. 157)

An overall placebo-medication response rate of 25% to 50% for migraine has been long established (e.g. Friedman & Merritt, 1957). Possibly the placebo improvement in the present study was mediated by a reduction in depression and emotional reactivity, brought about by participation in the experiment and relaxation. The placebo effect fell in the lower portion of the typical drug-placebo results. This feature of the data may be due to variation in the way treatments are presented. Drug studies often include suggestions by the researchers that the "new medication" will be helpful. In the present study, a carefully neutral stance was taken to the presumed effectiveness of the experimental procedures. The consent form the volunteers read and signed before the study included the statement; "Based on published research, the probability that the procedures in this study will reduce headache frequency, duration or intensity is approximately fifty percent" (Appendix I). The experimenter maintained a similarly cautious position in answering the volunteer's questions. Since strong suggestion

has been shown to enhance the placebo effect in neurotics (Shipman et al, 1974), the mild suggestion in the present study may have weakened its placebo effect.

Weak transfer of training (generalization) may also have reduced any effectiveness of the training procedures. In discussing biofeedback techniques, Knapp & Peterson (1976) remarked, "the biggest problem is that no studies have demonstrated generalization from the laboratory to the natural environment" (p. 268). Diamond & Franklin (Note 4) emphasized that migraine sufferers should practice biofeedback at home as well as in the lab, but they did not present data to show that home practice increased therapeutic benefits. Budzynski et al (1973) treated tension headaches with two laboratory EMG biofeedback sessions per week, and attempted to enhance trasfer of training by asking subjects to practice relaxation twice a day at home. The combined lab training and home relaxation benefited headache sufferers more than no treatment, but the relative effects of biofeedback and relaxation training was not assessed. Pearse et al (Note 11) trained their subjects to practice "body stress scanning" - taking a few moments at different times of the day to notice the build up of physical tension. Body stress scanning was intended to encourage patients to practice relaxation at the first sign of headache when, presumably, the pain was most susceptible to control. Similarly, Graham (1975) instructed his two cases to practice hand warming at the first signs of a migraine. Neither the Pearse et al (Note 11) nor the Graham (1975) study presented data on the effectiveness of their attempts to generalize to daily life.

In the present study volunteers typically practiced their relaxation and biofeedback at home in the evening. No particular attempt was

made to encourage volunteers to practice relaxation at the onset of a migraine, or at different places and times throughout the day. Several volunteers mentioned that they could not effectively perform the relaxation once a migraine had begun, because they could no longer concentrate on the autogenic phrases. Procedures designed to generalize training to whenever and wherever a migraine begins may potentiate relaxation and biofeedback programs for migraine. On the other hand, home practice in the present study failed to yield any demonstrable benefits.

The mean personality profiles of the volunteers were in keeping with past reports of the "migraine personality" (c.f. Dalessio, 1972). Taken as a whole the volunteers tended to be more intelligent, affected by feelings, apprehensive, tenderminded, self-sufficient, controlled and than males and females in the general population. They were more apt to over-react to small problems and were less disorganized than college undergraduates. However, the deviation about each mean weakens the notion of a "typical" migraine personality. The significant separation of the improvement groups on personality factors by the MDFA also showed that the present sample was heterogeneous with respect to personality.

Overall, the results of the present study failed to support the specific effectiveness of autogenic training, with or without hand temperature biofeedback, as a treatment for migraine. The amelioration of headaches that did occur in the study is interpreted as a placebo effect, possibly mediated by personality features that discriminated two levels of improvers from non-improvers, based on self report.

Significance

This study submitted increasingly popular relaxation and biofeedback treatments for migraine to rigorously designed experimental evaluation. The physiological, experiential, symptomatic and personality variables of chronic migraine were assessed and related to improvement. The results suggested that, contrary to current theorizing, autogenic training and biofeedback are placeboes rather than physiologically specific treatments.

APPENDIX I

CONSENT FORM

I, _____, consent to particpate in an experiment studying three techniques which may be effective in alleviating migraine headache. Based on published research, the probability that the procedures in this study will reduce headache frequency, duration or intensity is approximately fifty (50) per cent. My participation will involve one or more of the following: 1) keeping careful records of my headache, 2) listening to taped relaxation instructions for 15 minutes each day, 3) hand temperature training by biofeedback. The experiment will run for approximately 12 weeks. I understand that I will be asked to fill out personality and headache questionnaires and to be medically examined by a neurologist. I also understand that my performance will be monitored in a psychophysiological laboratory each week. During the laboratory sessions metal recording disks will be taped to my skin.

It has been explained to me that this study is a research project only, not a medical treatment. I understand that all communication between myself and Mr. Barton Jessup, the experimenter, is strictly confidential, and that I may withdraw from this study at any time.

Dated at London, this _____ day of _____, 1975.

_____ _____

Witness Signature

APPENDIX II

HEADACHE QUESTIONNAIRE

Date: _____

Name: _____

Sex: _____

Hair Colour: _____

Eye Colour: _____

Height: _____

Weight: _____

Age: _____

Blood Type: _____

Smoker: (Quantity) yes ( ) no ( ) _____

Drinker: (Quantity) yes ( ) no ( ) _____

Medication: (Including non-prescription analgesics, and birth control pills)

Clinical Assessment: (M.D.)

PLEASE PLACE THE NUMBER OF THE ANSWER THAT MOST ACCURATELY DESCRIBES YOUR HEADACHES BETWEEN THE BRACKETS AT THE RIGHT SIDE OF THE PAGE.

1. Have you had an injury to your head within the past 10 years? ( )

(1) no (2) very slight (3) a moderate injury (4) was briefly unconscious (5) required hospitalization

2. Have you had surgery in the past? ( )

(1) no (2) minor (e.g. a mole removed) (3) tumor removed from part of body (4) major surgery (5) cranial surgery

3. Some headache sufferers may report suffering more than one distinct type of headache. How many do you suffer? ( )

(1) 1 type (2) 2 types (3) 3 or more types

4. How frequently do you have headaches? ( )

(1) daily or almost daily (2) weekly (3) monthly (4) less often than monthly (5) only during certain times of the year (indicate which times)

---

5. How old were you when your headaches began? ( )

(1) 0-10 years (2) 10-20 years (3) 20-30 years (4) 30-40 years (5) over 40

6. How long have you been having headaches? ( )

(1) 6 months or less (2) 6 months to 1 year (3) 1-2 years (4) 2-5 years (5) more than 5 years

7. When you get a headache it usually lasts for: ( )

(1) 20 min. - 2 hours (2) 2-5 hours (3) 6 hours to 1 day (4) more than one day (5) constantly for many days

8. What time of the day are your headaches worst? ( )

(1) cannot say (2) evening (3) afternoon (4) noon (5) morning

9. Are your headaches worse during weekends and vacations? ( )

(1) never (2) seldom (3) occasionally (4) frequently (5) always

10. Do your headaches occur frequently for 1 week to 3 months, separated by longer headache free periods? ( )

(1) never (2) seldom (3) occasionally (4) frequently (5) always

11. How much of your head is involved in your headache? ( )

(1) Eye(s) only (please indicate left/right) (2) one side of head (please indicate left/right) (3) back of head (4) front of head (5) the whole head (like a "hat band") _____

12. Do the veins at the painful area of your head swell and throb when you have a headache? ( )

(1) never (2) seldom (3) occasionally (4) frequently (5) always

13. Do you have a tight, stiff feeling in your lower neck or shoulders when you have a headache? ( )

(1) never (2) seldom (3) occasionally (4) frequently (5) always

14. Does one side of your face hurt during a headache? ( )

(1) never (2) seldom (3) occasionally (4) frequently (5) always

15. Have other members of your family had headaches as <u>frequently</u> as you? ( )

(1) none (2) 1 family member (3) 2 family members (4) 3 family members (5) more than 3

Which other family members? (mother, brother, aunt etc.) _____

_____

16. Have other members of your family had headaches <u>with similar characteristics</u> to yours? ( )

(1) none (2) I family member (3) 2 family members (4) 3 family members (5) more than 3

Which other family members? (mother, brother aunt etc.) _____

_____

17. Can you identify any cause(s) of your headaches? ( )

(1) always (2) frequently (3) occasionally (4) seldom (5) never

Cause(s) _____

18. Do you have unusual visual experiences (e.g. flashing lights, jagged or wavy lines, etc)? ( )

(1) never (2) before some headaches (3) before and during a headache (4) before a headache/sometimes with out a headache (5) often

19. When you get a headache, does light or sound make it worse? ( )

(1) never (2) seldom (3) occasionally (4) frequently (5) always

20. Do your hands or feet get cold during headaches? ( )
(1) never (2) seldom (3) occasionally (4) frequently (5) always

21. When you get a headache, are you unusually sensitive to odours? ( )
(1) never (2) seldom (3) occasionally (4) frequently (5) always

22. Do you feel nauseous when you have a headache? ( )
(1) never (2) seldom (3) occasionally (4) frequently (5) always

23. Do you vomit when you have a headache? ( )
(1) never (2) seldom (3) occasionally (4) frequently (5) always

24. Does your eyelid(s) droop during a headache? (Indicate which one) ( )
(1) never (2) seldom (3) occasionally (4) frequently (5) always

---

25. Does the pupil of your eye get smaller during a headache? ( )
(1) never (2) seldom (3) occasionally (4) frequently (5) always

26. Does your nose run when you get a headache? ( )
(1) never (2) seldom (3) occasionally (4) frequently (5) always

27. Does part of your body become numb when you have a headache? ( )
(1) never (2) seldom (3) occasionally (4) frequently (5) always

28. Do you experience tingling-prickling sensations in part(s) of your body when you get a headache? ( )
(1) never (2) seldom (3) occasionally (4) frequently (5) always

29. Does any part of your body become limp or lose strength when you have a headache? ( )
(1) never (2) seldom (3) occasionally (4) frequently (5) always

30. Does your speech become slurred when you have a headache? ( )
(1) never (2) seldom (3) occasionally (4) frequently (5) always

31. Do you hear buzzing or ringing in your ears during a headache? ( )
(1) never (2) seldom (3) occasionally (4) frequently (5) always

32. Does the skin on your face or head flush at the painful areas of your headache? ( )

(1) never (2) seldom (3) occasionally (4) frequently (5) always

33. Do your eyes water when you have a headache? ( )

(1) never (2) seldom (3) occasionally (4) frequently (5) always

34. Do you get a headache when you are already tired or irritable? ( )

(1) never (2) seldom (3) occasionally (4) frequently (5) always

35. Do you get headaches when you are under stress? ( )

(1) never (2) seldom (3) occasionally (4) frequently (5) always

36. Do you get headaches when you are depressed? ( )

(1) never (2) seldom (3) occasionally (4) frequently (5) always

37. Do you get headaches when you are hungry? ( )

(1) never (2) seldom (3) occasionally (4) frequently (5) always

38. Do you get headaches when you eat certain foods? ( )

(1) never (2) seldom (3) occasionally (4) frequently (5) always

Which foods? _____

39. Does your stomach become upset after eating? ( )

(1) never (2) seldom (3) occasionally (4) frequently (5) always

40. Do you have a headache when you wake up in the morning? ( )

(1) never (2) seldom (3) occasionally (4) frequently (5) always

41. Do headaches wake you up? ( )

(1) never (2) seldom (3) occasionally (4) frequently (5) always

42. Do you wake up early in the morning (e.g. 3-5 a.m.) even when you don't have a headache? ( )

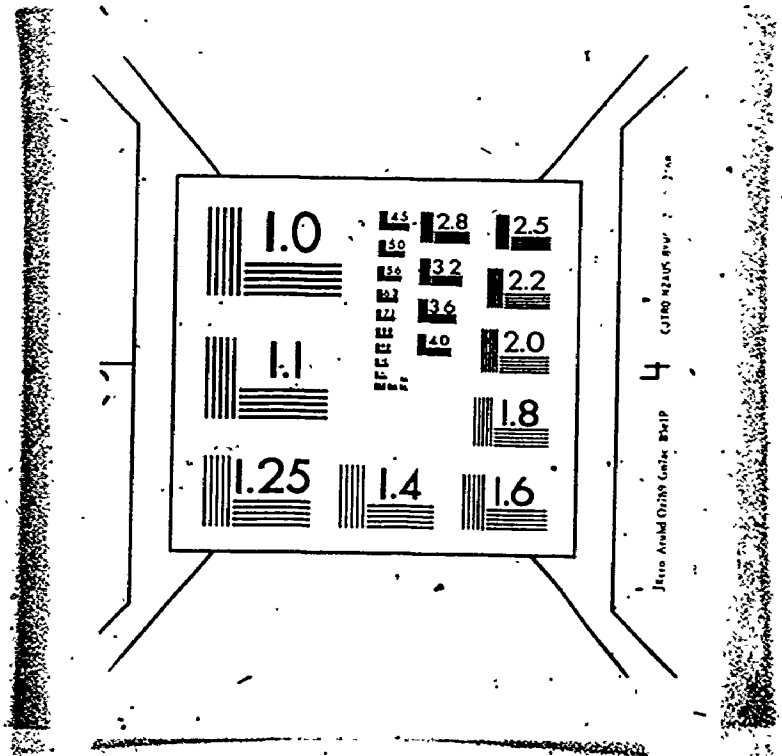
(1) never (2) seldom (3) occasionally (4) frequently (5) always

43. Do you have difficulty falling asleep? ( )

(1) never (2) seldom (3) occasionally (4) frequently (5) always

3 3

OF/DE



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5

44. Are your headaches helped by sleep? ( )

(1) never (2) seldom (3) occasionally (4) frequently (5) always

45. Do you get headaches after you have slept more than usual? ( )

(1) never (2) seldom (3) occasionally (4) frequently (5) always

THE FOLLOWING WORDS DESCRIBE DIFFERENT FEATURES OF HEADACHE PAIN. INDICATE HOW CHARACTERISTIC OF YOUR HEADACHES EACH WORD IS BY PLACING A 1-5 RATING BESIDE EACH WORD.

1 = never 2 = seldom 3 = occasionally 4 = frequently 5 = always

Beating	( )	grinding	( )	awful	( )
flickering	( )	pulling	( )	dreadful	( )
pounding	( )	tugging	( )	fearful	( )
pulsing	( )	wrenching	( )	frightful	( )
quivering	( )			terrifying	( )
throbbing	( )	burning	( )		
thumping	( )	hot	( )	cruel	( )
		scalding	( )	gruelling	( )
darting	( )	searing	( )	killing	( )
flashing	( )			punishing	( )
jumping	( )	itchy	( )	racking	( )
radiating	( )	rasping	( )	torturing	( )
shooting	( )	smarting	( )	vicious	( )
spreading	( )	stinging	( )	wicked	( )
		tickling	( )		
boring	( )	tingling	( )	agonizing	( )
drilling	( )			annoying	( )
lancinating	( )	aching	( )	bearable	( )
penetrating	( )	blinding	( )	discomforting	( )
piercing	( )	blurred	( )	distracting	( )
pricking	( )	drawing	( )	distressing	( )
stabbing	( )	dull	( )	excruciating	( )
		heavy	( )	horrible	( )
cutting	( )	hurting	( )	intense	( )
lacerating	( )	numbing	( )	intolerable	( )
sharp	( )	sore	( )	mild	( )
tearing	( )	splitting	( )	miserable	( )
		steady	( )	savage	( )
binding	( )	tender	( )	troublesome	( )
biting	( )			ugly	( )
cramping	( )	exhausting	( )	unbearable	( )
crushing	( )	fatiguing	( )	violent	( )
gnawing	( )	nagging	( )		
gripping	( )	tiring	( )		
nipping	( )				
pinching	( )	choking	( )		
pressing	( )	nauseating	( )		
squeezing	( )	sickening	( )		
taught	( )	suffocating	( )		
tight	( )	wretched	( )		

THANK YOU FOR YOUR CO-OPERATION AND ASSISTANCE.

APPENDIX III

Headache Diary Sheet

PATIENT NAME: _____ START DATE: _____

	MIGRAINE ATTACKS				GENERAL SYMPTOMS				DIET AND ACTIVITIES		
Date	Severity of Migraine* "A"	Duration of Migraine (mins hrs)	Treatment of Attack (Product & Dose)	Degree of Relief*** "B"	Quality of Sleep "C"	Quality of Appetite++ "D"	General Well-Being++ See "E"	Other Symptoms "F"	Off Work Because of Migraine Yes / No	Social Activities Missed Due to Migraine Yes / No	Special Foods Ingested# "G"

A * 0 No Migraine
1 Mild
2 Moderate
3 Severe

B *** 0 No relief
1 Mild relief
2 Moderate relief
3 Marked relief

C + 0 Slept very well
1 Slept fairly well
2 Sleep not as good as usual
3 Sleep markedly disturbed and/or waking up with migraine

D ++ 0 Appetite very good
1 Appetite average
2 Appetite not as good as usual
3 Appetite poor

E +++ 0 Feeling exceptionally well
1 Feeling fairly well
2 Not as good as usual
3 Bad

F ** 0 No other symptoms
1 Tired and lacking in energy
2 Mentally alert and full of energy
3 Other (please state)

G # 0 Alcohol
1 Beans
2 Cheese
3 Chocolate
4 Coffee
5 Dairy Produce
6 Eggs
7 Fatty Fried food
8 Fish
9 Fruit (Citrus)
10 Meat (Pork)
11 Nuts
12 Seafoods
13 Tea
14 Tomatoes
15 Vegetables (onions)
16 Wheat

APPENDIX IV

McGill Pain Questionnaire

THE FOLLOWING WORDS DESCRIBE DIFFERENT FEATURES OF HEADACHE PAIN. INDICATE HOW CHARACTERISTIC OF YOUR HEADACHES EACH WORD IS BY PLACING A 1-5 RATING BESIDE EACH WORD.

1 = never 2 = seldom 3 = occasionally 4 = frequently 5 = always

Beating	( )	grinding	( )	awful	( )
flickering	( )	pulling	( )	dreadful	( )
pounding	( )	tugging	( )	fearful	( )
pulsing	( )	wrenching	( )	frightful	( )
quivering	( )			terrifying	( )
throbbing	( )	burning	( )		
thumping	( )	hot	( )	cruel	( )
		scalding	( )	gruelling	( )
darting	( )	searing	( )	killing	( )
flashing	( )			punishing	( )
jumping	( )	itchy	( )	racking	( )
radiating	( )	rasping	( )	torturing	( )
shooting	( )	smarting	( )	vicious	( )
spreading	( )	stinging	( )	wicked	( )
		tickling	( )		
boring	( )	tingling	( )	agonizing	( )
drilling	( )			annoying	( )
lancinating	( )	aching	( )	bearable	( )
penetrating	( )	blinding	( )	discomforting	( )
piercing	( )	blurred	( )	distracting	( )
pricking	( )	drawing	( )	distressing	( )
stabbing	( )	dull	( )	excruciating	( )
		heavy	( )	horrible	( )
cutting	( )	hurting	( )	intense	( )
lacerating	( )	numbing	( )	intolerable	( )
sharp	( )	sore	( )	mild	( )
tearing	( )	splitting	( )	miserable	( )
		steady	( )	savage	( )
binding	( )	tender	( )	troublesome	( )
biting	( )			ugly	( )
cramping	( )	exhausting	( )	unbearable	( )
crushing	( )	fatiguing	( )	violent	( )
gnawing	( )	nagging	( )		
gripping	( )	tiring	( )		
nipping	( )				
pinching	( )	choking	( )		
pressing	( )	nauseating	( )		
squeezing	( )	sickening	( )		
taught	( )	suffocating	( )		
tight	( )	wretched	( )		

THANK YOU FOR YOUR CO-OPERATION AND ASSISTANCE.

APPENDIX V

DEBRIEFING

Thank you for participating in this study designed to assess the effectiveness of 5 techniques for the alleviation of migraine headaches. Two techniques consisted of attempts to increase hand temperature by relaxation training with or without biofeedback. Two other techniques similarly assessed the effect of attempting to decrease hand temperature. A fifth procedure involved keeping a headache diary without any specific training in relaxation or biofeedback. You have used one technique, while other volunteers have tried the other techniques. The majority of previous research has used only the hand warming procedures, apparantly with moderate benefit to about ½ of the participants. The results of a more recent study would imply, however, that hand cooling should also be effective. Some clinicians think that information from headache record keeping can also help migraine sufferers to gain control of their headaches. Overall, then, the research to date has suggested hand warming training to be beneficial, but the components of the hand warming procedure, and hand cooling and record keeping have not yet been adequately assessed. You are welcome to try any of the techniques in this study, without participating in further research.

Once again, thank you for your valuable participation and assistance.

Barton A. Jessup

APPENDIX VI

AUTOGENIC PHRASES

> Now you will hear a series of phrases designed to help you relax. After you hear each one, please continue to repeat it mentally. Don't worry about getting results. A casual, passive attitude is best. While you are mentally repeating each phrase, put your attention to the part of your body involved. Allow me to repeat the instructions. You will hear a series of phrases designed to help you relax. After you hear each one, please continue to repeat it mentally. Don't worry about getting results. A casual, passive attitude is best. While you are mentally repeating each phrase, put your attention to the part of your body involved. The first phrase is, my right arm is heavy. Please continue to repeat mentally, my right arm is heavy.

At 45 second intervals the following standard autogenic phrases were given:

> My right leg is heavy, My left arm is heavy, My left leg is heavy, Both arms are heavy, My arms and legs are heavy, My right arm is warm, My right leg is warm, My left arm is warm, My left leg is warm, Both arms are warm, Both legs are warm, My arms and legs are warm, My heart beat is calm and regular, It breathes me, My solar plexus is warm, My forehead is cool.
>
> Now bring your attention back to the room that you are in by shaking your hands loosely from the wrists, taking a deep breath, and opening your eyes. Thank you.

The phases for the hand cooling conditions were identical, except that arm and leg temperature references suggested "cool" and the forehead reference suggested "warm". (After Luthe, 1969)

APPENDIX VII

W1	33.18 .95	32.95 .91	33.10 1.05
W2	33.06 .85	32.72 .89	32.92 .87
C3	33.50 1.62	32.90 1.65	33.00 .83
C4	33.22 .94	32.70 1.80	33.17 1.13
X5	33.25 .76	33.10 .69	32.83 .89
WK	1-4	5-8	9-12

Means (upper numbers) and Standard Deviations (lower numbers) of Minute 15 Forehead Temperature Within Each Treatment by Block Cell

Note: Abbreviations: w=hand warming, c=hand cooling, x=control group, wk=weeks.

t-tests at Block 2 (c.f. hypothesis 3, page 125) W1-W2 t (94) = 1.18, NS: C3-C4 t (94) = .75, NS.

APPENDIX VIII

W1	33.51 2.10	33.42 2.37	33.82 3.70
W2	32.98 3.05	32.58 3.66	32.38 4.65
C3	33.55 9.20	33.18 2.33	30.80 4.75
C4	33.00 3.67	32.74 3.41	33.22 2.94
X5	32.81 .76	32.11 .69	32.05 .89
WK	1-4	5-8	9-12

Means (upper numbers) and Standard Deviations (lower numbers) of Minute 15 Finger Temperature Within Each Treatment by Block Cell

Note: Abbreviations: w=hand warming, c=hand cooling, x=control group, wk=weeks.

t-tests at block 2 (c.f. hypothesis 3, page 125) W1-W2 t (94) = 2.4, $p < .05$, in counter-theoretical direction; C3-C4 t (94) = 1.29, NS.

APPENDIX IX

W1	.83 .93	.67 .81	.52 .65
W2	2.06 1.87	1.33 1.42	1.52 1.65
C3	2.23 2.44	1.94 2.24	1.85 2.48
C4	1.79 2.00	1.65 2.03	1.75 2.36
X5	2.35 2.04	1.92 2.17	1.79 2.12
WK	1-4	5-8	9-12

Means (upper numbers) and Standard Deviations (lower numbers) of Number of Headaches per Week Within Each Treatment by Block Cell

Note: Abbreviations: W=hand warming, c=hand cooling, x=control group, wk=weeks.

t-tests (c.f. hypothesis 1, page 125): Block 1 W1-C3 t (94) = 5.38, $p<.05$; Block 2 W1-C3 t (94) = 5.08, $p\leq.05$; Block 2 W2-C4 t (94) = 1.21, NS; Block 1 W2-X5 t (94) = 1.04, NS; Block 2 W2-X5 t (94) = 2.19, $p<.05$.

APPENDIX X

W1	1.11 1.10	.88 1.06	.85 1.09
W2	1.74 .96	1.39 1.16	1.28 1.02
C3	1.48 1.32	1.43 1.04	1.08 1.11
C4	1.36 1.07	1.24 1.07	1.15 1.13
X5	1.53 .95	1.41 1.06	1.25 1.07
WK	1-4	5-8	9-12

Means (upper numbers) and Standard Deviations (lower numbers) of Headache Intensity per Week Within Each Treatment by Block Cell

Note: Abbreviations: W=hand warming, c=hand cooling, x=control group, wk=weeks.

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