A CONTROLLED COMPARISON OF BIOFEEDBACK METHODS AND RELAXATION IN THE TREATMENT OF MIGRAINE HEADACHES.

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TO GAYLE.

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ABSTRACT.

The study was designed to investigate the comparative efficacy and specificity of action of finger temperature biofeedback, temporal artery amplitude biofeedback and relaxation as treatments for migraine headache. A heart rate feedback condition (placebo control) and a waiting list control condition were also included. Patients' symptoms were evaluated using a modified version of Waters Headache Questionnaire. Dependent variables were finger temperature, temporal artery amplitude, heart rate, frequency of headaches, intensity of headaches, duration of headaches and an index of headache activity. An analgesic index, frequency of vasoconstrictor drug use and amount of prophylactic medication taken, were also recorded. Monthly records of headache activity were taken for each patient over the period of a year. The experimental patients were given a three month baseline, a three month treatment period (consisting of ten one-hour treatment sessions) and a six month follow-up period.

Group analysis of the physiological data provided little evidence that biofeedback procedures enabled patients to gain control of the relevant physiological parameters, although the clinical results suggested the superiority of finger temperature and heart rate (placebo) feedback in reducing headache activity. As a result of this anomaly an investigation into the relationship between the clinical changes and changes in physiological measures for each individual patient was carried out. Of the physiological measures, a decrease in mean temporal artery amplitude over all subjects accounted for the highest proportion of the variance in clinical outcome. Implications are drawn for current theories of migraine headache, and biofeedback methodology is critically evaluated.

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CHAPTER 1

MIGRAINE HEADACHES.

MIGRAINE HEADACHES

INTRODUCTION

Migraine is an ill defined, self limiting condition which takes up much of the time of general practitioners. It involves no risk of mortality but can cause extreme distress to the sufferer, occurring with little warning and at times which may be inconvenient and socially embarrassing. Millac (1980) stated that headaches lead 70% of the British population to seek medical advice at some stage in their lives. Phizacklea and Wilkins (1978) reported that in a single handed urban practice of 3000 patients, headaches accounted for 192 consultations in a six month period and that an estimated 25,000 patients are referred annually to hospital outpatient departments with headache. DeLozier and Gagnon (1975) estimated from an American survey of ambulatory patients, that 944.9 million visits concerning headaches were paid to general practitioners between May 1973 and April 1974. Of the 60 most frequent patient problems, headache was 14th and was the 8th most frequest symptom orientated problem. Appenzeller (1979) estimated that at least 40% of all North Americans have experienced severe headaches at some point in their lives.

Migraine is one of a variety of headache types. The following classification is taken from Diamond (1975):

HEADACHE CLASSIFICATION

1. Traction and Inflammatory

Mass lesions (tumors, oedema, haematoma, etc).

Diseases of the eye, ear, nose, throat, teeth.

Cranial neuralgia.

Allergy.

Infection.

Arteritis, phlebitis.

Cervical osteoarthritis.

Chronic myositis.

2. Psychogenic

Tension (anxiety).

Depressive.

3. Vascular

Migraine.

a) Classic.

b) Nonclassic.

Cluster (periodic migrainous neuralgia).

Facial.

Opthalmoplegic.

Hemiplegic.

Migraine is a condition for which there are no signs that can be objectively measured and doctors must rely solely on a subjective description of symptoms by the patient in order to make a diagnosis. The clinical definition of migraine has been a subject of dispute

for many centuries and remains so today. Friedman, Finley, Graham, Kunkle Ostfield, Wolff (1962) recommended the following definition;

Vascular headache of the migraine type.

'Recurrent attacks of headache, widely varied in intensity, frequency and duration. The attacks are commonly unilateral in onset; and usually associated with anorexia and sometimes preceded by, or associated with, conspicuous sensory, motor and mood disturbances; and are often familial'.

Nevertheless, it is often difficult to differentiate between migraine and other types of headache, as there are no clear cut lines whereby a headache can be diagnosed as migrainous or not.

Migraine may be divided into two main subgroups. First, classic migraine where the headache is preceded or accompanied by visual aura, sensory or speech disturbances, and second, nonclassic or common migraine, which is not associated with the occurrence of sharply defined focal neurological disturbances. In both of these types of migraine headache, the pain is unilateral and may be associated with nausea or vomiting. During the painful phase of the attack, patients may also experience neurological symptoms such as numbness, 'pins and needles', or giddiness.

The warning signs reported to precede the onset of a classic migraine headache may be useful in helping the sufferer begin treatment at the earliest possible moment in the migraine attack. The following is an extract from a list of warning signs complied by the Migraine Trust (1971):

Visual disturbances (double vision, difficulty in focusing, temporary partial blindness, spots or lines, and dazzling display of coloured lights)
Dizziness
Hallucinations
Nausea
Vomiting
Tingling Sensations
Sensitivity to noise or light
Depression
Uncommon energy and vigour
Loss of appetite
Excitability
Difficulty in speaking
Increase in weight
Blotchy patches or rashes on the skin
Swelling of fingers, waist or breasts
Numbness
Elation
Pains in neck and shoulders
Irritability and tension
Unusual hunger
Weakness and trembling
Talkativeness
Increase in volume of urination
Unusual pallor

As well as the 'classic' and 'common' migraine variants, other less common types may occur. 'Hemiplegic' migraine and 'Opthalmoplegic' migraine are vascular headaches characterised by sensory and motor phenomena which persist during and after the headache (Friedman et al, 1962). 'Facial' migraine, otherwise known as 'Lower half' or 'facioplegic' migraine is a condition in which the headache is followed by facial paralysis (Bickerstaff, 1977). In early life, some migraineurs may experience the prodromal phase of classic migraine but not the headache. The headache becomes a feature in later life. It is however, uncommon for migraine to start this way in adult life, or for this pattern to occur in patients with well-established attacks (Bickerstaff, 1977).

'Cluster Migraine' (Periodic migrainous neuralgia) is a condition which, although allied to migraine, is sufficiently different to warrant special attention. The symptoms include ciliary neuralgia; histamine cephalalgia, Horton's neuralgia; cluster headache; autonomic cephalalgia; erythromelalgia of the head; greater superficial petrosal neuralalgia and Harris's neuralalgia. It affects men six times more often than women, occurs in bouts lasting 3-12 weeks or even longer at intervals of six months to two years and usually starts in the third or fourth decade. In each bout at least one attack and sometimes several occur each day, with a characteristic predisposition to occur between midnight and 3 am (Bickerstaff, 1977).

This brief introduction illustrates the diversity and magnitude of the problem associated with the migraine headache and its variants. Despite much clinical and research attention, it is still a relatively little understood phenomenon and many myths have grown up about the incidence of the condition and the characteristics of the sufferers. As this project is concerned specifically with the classic migraine headache, problems of definition for diagnostic purposes, establishing prevalence rates and examining beliefs that surround the condition will be covered in chapter 3.

AETIOLOGY AND TREATMENT OF CLASSIC MIGRAINE

Current evidence supports the view that cranial arterial distention and dilatation are implicated in the painful phase of the headache, but cause no permanent changes in the involved vessels (Friedman et al, 1962). Classic migraine headaches have long been treated with medications designed to influence the observed vasodysfunctions. Chapters 4 and 5 are concerned with physiological

and biochemical changes associated with classic migraine headaches. Also, the merits of acute and prophylactic medication in the treatment and management of classic migraine is overviewed in chapter 6.

As mentioned, many of the available drug treatment regimes are directed at the prevention or the re-establishing of biochemical and calibre changes in the intra- and extra-cranial arteries; however an editorial in Hemicrania (1971) stated that:

"It must be remembered that there are at least two causes of a migraine attack. There is the primary cause, the often familiar but still obscure diathesis or constitutional predisposition that loads the gun, and the secondary cause that pulls the trigger".

Research is now being directed at both sets of causes, the constitutional inadequacy and the numerous secondary factors that trigger the headache and associated symptoms. The following trigger factors have been reported as initiating migraine attacks:

Anxiety, worry, emotion, depression, shock and excitement
Over-exertion
Physical and mental fatigue
Bending or stooping
Excess or sleep deprivation
Changes of climate
Artificial light
Bright lights
Penetrating smells
Intense odours
Alcohol
Certain foods (fried food, chocolate and cheese)
Oral contraceptives
Travel
Use of sleeping tablets
Change of routine
Loud noises
Hot baths
Lack of food
Menstruation
High blood pressure
Toothache

(Thompson , 1978).

The variety of proprietary medications available for the treatment of classic migraine have been considered to reflect the lack of efficacy of any one particular treatment approach (Sargent, Green and Walters , 1973b). Different modes of action of the various medications may also indicate the absence of a single factor aetiology. Given that dependence, side effects and rebound headaches are major disadvantages of drug treatments, it is not surprising that alternative treatments have been considered.

Thompson (1978) stated that 'treatment, consists in the first place, of trying to avoid precipitating factors'. As has been shown, there are various triggering factors and the avoidance of one does not mean that other factors, perhaps unavoidable ones, may not also be operative. Thompson (1978) also recommended that 'should an attack threaten, the victim should immediately lie down' and that 'when an attack is fully developed, rest in a bed, in a quiet darkened room is essential'. Neither of these recommendations may be possible for many sufferers, given specific situations at the onset of the headache, even though obvious warning signs may precede the onset of the pain.

As the prevention of a migraine is more desirable than the acute treatment of an attack (given the limitations and variable effectiveness of drugs) a variety of alternative approaches has been considered by clinicians. Although such approaches as insight psychotherapy and problem orientated behaviour therapy have met with some success, an increasingly popular approach to the management and treatment of classic migraine is biofeedback.

CHAPTER 2

BIOFEEDBACK.

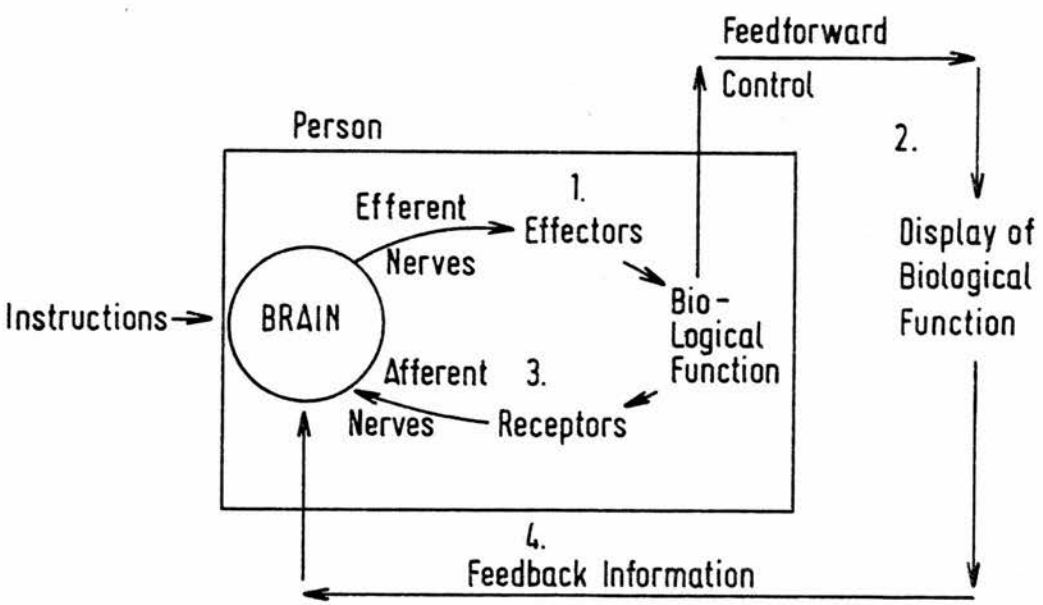
BIOFEEDBACK

Biofeedback has been defined as: "any technique which increases the ability of a person to control voluntary physiological activities by providing information about those activities". (Olton and Noonberg, 1980).

It has long been accepted that biofeedback can effect the skeletal musculature. Prior to 1969, little evidence existed to support the notion that biofeedback could influence the autonomic nervous system. Following experiments which appeared to demonstrate the control of autonomic function by operant conditioning techniques (Miller 1969), an increase in the diversity of biofeedback applications has taken place. The application of biofeedback in clinical practice has not been restricted by a lack of success in replicating Miller's original findings. (Miller and Dworkin 1974)

The following is a theoretical representation of the nervous system components underlying biofeedback (adapted from Olton and Noonberg ,1980).

MECHANISMS UNDERLYING BIOFEEDBACK



The diagram represents the parallel components of the external biofeedback display and the internal self control aspects of biofeedback. Within the subject, (1) efferent neurones stimulate the effector system which alters the biological function under observation. (2) These changes are registered on the biofeedback display via transducers attached to the feedforward control system. (3) The changes in the biological function are registered by internal receptors and the information is sent through afferent nerves to the brain. Simultaneously, (4) the subject receives information concerning the internal changes via the biofeedback display. This is a general representation of the principles underlying the application of biofeedback; the ultimate aim of biofeedback is that the information received about internal responses will enable the subject to gain volitional control over those responses.

The following is a brief overview of some of the clinical applications of biofeedback.

CARDIOVASCULAR TRAINING

Much of the original work in this area was conducted by Engel and colleagues (summarised in Engel and Bleeker , 1974) working with various cardiac disorders including atrial fibrillation, tachycardia and premature ventricular contractions. Although these studies provided promising evidence for the effects of biofeedback; even with patients who had received little benefit from other treatments, there were no controls for placebo effects and no extensive baseline data were obtained. However, this work has been replicated by Pickering and Miller (1977). Furthermore, other cardiac disorders such as sinus tachycardia, supraventricular tachycardia and paroxysmal

atrial tachycardia have been successfully treated, although more evidence regarding transfer of control from the laboratory to every-day life and longer follow-up periods are required. Also the rationale behind treating cardiac arrhythmias via control of heart rate remains obscure. It has been suggested that gaining control over heart rate modifies sympathetic and vagal tone to the heart, leading to reduction in abnormalities, but this is highly speculative. It should also be noted that little attention has been paid within biofeedback to the role of psychological factors in cardiac disorders, although the importance of such factors has long been recognised (Yates , 1980). A final problem is that Benson, Alexander and Feldman (1975) reported that training patients in progressive relaxation may be as effective as biofeedback.

HYPERTENSION

Many attempts have been made to treat hypertension with biofeedback. Benson, Shapiro, Tursky and Schwartz (1971) used biofeedback to lower systolic blood pressure of seven hypertensive patients. A marked improvement in five out of seven hypertensive patients was reported, two of these patients bringing down their blood pressure to within normal range. Over all patients the mean systolic pressure was reduced from 165 to 148 mm Hg. This work has been replicated by other workers, with generally consistent results; although recently Surwit, Shapiro and Good (1978) were able to obtain only very small, clinically non-significant changes. A further problem is that long term follow-up suggests that these effects are very short lived. Biofeedback for high blood pressure may therefore not be particularly useful, especially since it is technically very

complex. Further, Patel (1977) has shown in a number of studies that passive relaxation and meditation seem to be superior to biofeedback in reducing blood pressure. The complex technology of biofeedback may therefore not be required.

ELECTRO-ENCEPHALOGRAPHIC (EEG) FEEDBACK

There is now much evidence to show that subjects can be trained to control EEG frequency, particularly they can be trained to increase the proportion of alpha rhythm (8-12 hz). Alpha rhythm is typically found during states of meditation and relaxation; during such states, pain perception and anxiety are diminished. However, alpha feedback is beset with methodological problems; whether the subjects keep their eyes open or closed, the effects of ambient illumination, and so on. In view of such problems, it would appear that alpha training has little to offer in the way of clinical application. Melzack and Perry (1975) found that alpha training had little effect in the treatment of pain. However, it would appear that one promising area of EEG training is that of sensory motor rhythm (SMR) training. Sterman and Friar (1972) trained three epileptics to generate SMR and found a reduction in abnormal slow-wave activity and polyspike discharges. All three patients demonstrated a reduction in seizures. Later Sterman (summarised in Sterman,1977) reported on five cases of epilepsy, using an ABAB design. He found improvement in four out of the five cases. The potential usefulness of this approach in treating epilepsy has been confirmed by other workers (Lubar and Shouse , 1977). However, the precise nature of SMR remains to be elucidated, and in other studies it has been found that patients improved without a concomitant change in SMR but with an associated

change in other rhythms. It would therefore seem unlikely that therapeutic effects are due to the production of a particular rhythm but a 'normalising' of the EEG and hence the suppression of general epileptic activity.

ELECTRO-MYOGRAPHIC (EMG) FEEDBACK

EMG feedback differs from other methods of biofeedback in that the biological response of interest is the activity of the striate muscle, which is relatively under voluntary control. Typically the electrical activity generated by muscle activity is transformed into a noise, such that a high frequency click or high pitch noise is indicative of tension, and a low frequency click or low pitch noise is indicative of relaxation.

One of the most common disorders to which EMG feedback has been applied is the so-called tension headache. Recent work however has shown that the distinction between tension and migraine headache is not so clear cut as was once believed (Martin and Mathews , 1978); however, many biofeedback researchers have continued to assume that they are entirely different clinical entities. Much of the original work was reported by Budzynski, Stoyva and colleagues (Budzynski, Stoyva, Adler and Mullaney ,1973). In a series of essentially uncontrolled reports, Budzynski et al (1973) observed that EMG from the frontalis muscle was effective in reducing tension headache activity. Unfortunately, as with the early migraine studies, the effects of feedback were confounded with the effects of relaxation, relaxation being given simultaneously with feedback. When the relevant comparisons were made, it appeared that there was little advantage in using EMG feedback (Cox, Freundlich and Meyer , 1975).

Gray, Lyle, McGuire and Peck (1980) found that relaxation was more effective than EMG feedback and that whether the electrodes were placed on the painful site or elsewhere on the head, did not seem to be crucial. There is now much evidence to suggest therefore that simple training in progressive relaxation is just as effective as EMG feedback treatment of tension headaches.

One of the most successful applications of EMG feedback to clinical problems, is in the area of neuro-muscular disorders. Andrews (1964) reported on a series of 20 patients with hemiparesis, who had had no return of function in the year since onset. Seventeen of these patients were able to develop well modulated action in a previously paralysed arm in very few sessions. In a large scale but uncontrolled study, Brundy, Korein, Grynbaum, Friedmann, Weinstein, Sachs-Frankel and Belandres (1976) used EMG feedback with 114 neuromuscular disordered patients; the diagnoses included hemiparesis, torticollis, dystonia and spinal cord and peripheral nerve injury. All patients had had conventional therapy with little or no recovery. The treatment took place over a period of eight weeks. During the follow-up period of three years, 50% of the patients had significant clinical improvement. Wooldridge and Russell (1976) trained twelve spastic children in the control of head position. Movement of the child's head to the right produced a sound in the right ear, and a movement to the left produced a sound in the left ear. The children were thus trained to control head position during a variety of everyday activities. All the children responded to some degree and this control generalised to other areas of functioning.

Other EMG applications have also produced clinically impressive results. Engel, Nickoomanesh and Schuster (1974) trained six patients

who had been incontinent of faeces for five years from a variety of causes including diabetic neuropathy and gastrointestinal surgery. They were trained to produce external sphincter contractions in synchrony with internal sphincter relaxation. During the follow-up period of five years, four of the patients were completely continent and there was improvement in the other two. This application is particularly impressive because it would appear that there is no viable alternative treatment to this very debilitating condition.

EEG feedback has also been used to teach pregnant women appropriate muscle control during labour. Gregg (1978) gave EMG feedback to 100 women during pregnancy and found a significant reduction in the duration of the first stage of labour; in addition, less pain was reported and less sedative medication was requested.

Webb (1974) has also used EMG feedback as an aid for the blind in appropriately portraying emotional state and thus enhancing interpersonal interactions.

Other areas where EMG feedback appears to be useful include the treatment of blepharospasms (Peck ,1977), writers cramp (Reavley, 1975), and severe tremors (LeBoeuf , 1976).

SKIN TEMPERATURE FEEDBACK

Skin temperature training has been one of the most popular methods of biofeedback, possibly because it is comparatively straightforward from a technological point of view. Skin temperature bears a close correspondence to blood flow in the underlying tissue. Accordingly, it is possible that skin temperature training may be useful in the treatment of disorders of peripheral blood flow.

One of the major disorders of peripheral blood flow is

Raynaud's Disease, a predominantly female disorder characterised by decreased blood flow, frequent vaso-spastic attacks and increased susceptibility to frostbite in the hands and feet. Taub and Emurian (1976) treated four patients with Raynaud's Disease, training them to increase hand temperature. All patients kept records of the frequency of vaso-spastic attacks, and reported a significant decrease. Success rates of 91 - 95% have been reported but typically studies reporting such figures were not well controlled and had small numbers. Keefe, Surwit and Pilon (1980) gave 21 female subjects either autogenic training, progressive relaxation training, or a combination of autogenic training and temperature feedback. All of the patients improved irrespective of the method of treatment. It would appear therefore that there is little advantage in using temperature biofeedback; equal therapeutic success may be obtained by using relaxation.

The most common clinical application of finger temperature training has been in the treatment of migraine headaches. The pioneers in this area are Sargent, Green and Walters (1972). They used differential temperature training whereby patients were trained to increase the temperature difference between finger and forehead. Initial success rates were claimed to be around 70% using large numbers of patients. However this work has been severely criticised on the grounds that no control groups were used, little baseline data were reported and there was an inadequate statistical evaluation of the results. Most crucially, biofeedback was given at the same time as autogenic training, thus one cannot determine which was the crucial ingredient producing therapeutic change. Despite these inadequacies, the work of Sargent et al (1972) has stimulated considerable further research. A detailed summary of the application of finger temperature

biofeedback for migraine is included in chapter 7.

CEREBRAL VASO-MOTOR RESPONSE (CVMR) FEEDBACK

Snyder and Noble (1968) demonstrated that subjects could be trained to decrease arterial pulse amplitude in the finger, when given direct feedback of the amplitude. Christie and Kotses (1973) demonstrated that increases and decreases in cephalic vasomotor responses could also be achieved, although the changes were small and there was no difference from baseline levels. However these initial findings have stimulated researchers to apply CVMR feedback clinically. The superficial temporal artery is the vessel most closely associated with the alleged cerebral vascular abnormality in migraine headache; it may therefore be possible to train migraine patients to control temporal artery amplitude and thereby gain control over their headaches. Although initial clinical findings are encouraging, a clear relationship between clinical and vasomotor changes has yet to be reported. Also, the rationale for CVMR feedback does not appear to be consistent with the rationale for finger temperature feedback. The former involving vasoconstriction of the temporal artery, is associated with increased sympathetic activity; but finger temperature feedback is associated with decreased sympathetic activity. However, both claim to be successful procedures in the alleviation of migraine headaches. This question will be addressed in greater detail in chapter 7.

CONCLUSION

In the area of biofeedback, claims of general effectiveness and of wide applicability have gone beyond the available date. Much

more carefully designed research is required, using appropriate control groups as the extravagant speculations pervading this area have done biofeedback a disservice (Brown, 1974). Yates (1980) stated that biofeedback may have only an ancillary or minor role to play in some treatment methods. Nevertheless, that role may be an important one in both practical terms and, in some cases, in suggesting new treatment approaches.

Hiebert (1976) stated that the popularity of biofeedback has increased to the point where serious investigation into the therapeutic claims and treatment methodologies is warranted. Engel-Sittenfeld and Engel (1978) suggested that the treatment of migraine through biofeedback methods is probably one of the best examples of how biofeedback has been used in a clinical context without explicit diagnostic procedures and without deep knowledge of the mechanisms underlying effects. Adams, Feuerstein and Fowler (1980) stated that although biofeedback provides promising leads in the development of effective physiological treatments for migraine headaches, the claims of these techniques must be substantiated by well-designed treatment outcome studies. They concluded that the most reasonable research tactic would be to conduct a clinical trial including a variety of treatment groups, a placebo group and a no-treatment waiting list control group. The clinical trial should also examine the active components and mechanisms proposed to be responsible for the therapeutic effects. The clinical research criteria specified by Adams et al (1980) emerge from a detailed critical appraisal of the use of biofeedback (included in chapter 7) and have been incorporated in the design of the present research.

CHAPTER 3

EPIDEMIOLOGICAL ASPECTS OF MIGRAINE HEADACHES

EPIDEMIOLOGICAL ASPECTS OF MIGRAINE HEADACHE

This review will be concerned with four areas relevant to determining the prevalence of migraine in the community. Firstly, an overview of clinical impressions will be outlined; secondly, the problems of diagnosis and its implications for epidemiological studies will be considered. Thirdly, a brief overview of contemporary epidemiological studies will be given and the concluding part of the chapter will outline current impressions on the epidemiology of migraine.

According to a survey by DeLozier and Gagnon (1975), headaches (undifferentiated) was among the top 14 principle problems reported by out-patients attending office-based general practitioners in the one year period between May 1973 and April 1974. This symptom alone accounted for over 12 million office visits in the U.S.A.; categorising these patients by sex indicated that 29% were male and 71% female.

Prevalence rates for migraine as distinct from other forms of headpain is a matter of debate, the definition of migraine, which is obviously central to the issue, having led to much controversy. Critchley (1962) stated that the only criterion necessary to establish a diagnosis of migraine is "the presence of recurrent headache, irrespective of any preceding, accompanying or subsequent phenomenon". This statement reflects the lack of specificity employed by some clinicians in the definition of migraine.

Although the definition of migraine as used by Critchley (1962) may be considered 'overinclusive', more frequently employed definitions (Gowers , 1888 ; Friedman, Finley, Graham, Kunkle, Ostfield and

Wolff, 1962) are also deficient. The description given by Gowers (1888) includes the features accepted as essential parts of the diagnosis. He wrote "migraine is an affection characterised 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 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".

This definition, as can be seen, is limited by the use of such adverbs as 'usually' and 'frequently', and the Friedman et al (1962) definition, although essentially the same as the above, has the following qualifying phrase; 'all characteristics are not necessarily present in each attack or in each patient'. Hence the determination of prevalence rates is subject to interrater variability.

As stated, sex differences in migraine have been an area of interest, and as indicated by DeLozier and Gagnon (1975), females appear to be most vulnerable. Selby and Lance (1960) reported that 60% of 500 vascular headache cases from an Australian sample were women, whereas Ziegler, Hassanein and Hassanein (1972), in an American study, reported no differences between males and females. More recently, Waters and O'Connor (1975) reported an 8% higher incidence rate among women. Adams, Feuerstein and Fowler (1980), stated that since the majority of epidemiological studies have been conducted in Britain or Australia, cross cultural factors may account for some of the variance in reported incidents.

Social class has been implicated as being a significant feature in the incidence of migraine headaches. A Research Committee of the Council of General Practitioners (1962) found a higher incidence of migraine in professional classes and that a social gradient in recorded prevalence of consultations was evident; a decrease in consultation rates was associated with decreasing social class.

It has been hypothesised that the increased prevalence of migraine in professional persons may be associated with occular disorders caused by continued close work. Williams (1966) stated that prolonged eye strain, especially in the presence of uncorrected errors of refraction is considered to precipitate migraine attacks.

Walker (1959), reported that in 300 consecutive patients over the age of 40 years, the prevalence of migraine rose as blood pressure increased. However, Robinson (1969), found that certain symptoms, and in particular headache, made general practitioners more likely to measure a patient's blood pressure. This would give an underestimate of the incidence of high blood pressure in non-headache patients.

Familial prevalence is also an area of considerable interest. Liveing (1873) was the first to state that migraine was a familial condition, subsequent studies have strengthened this impression; (Lennox , 1941 ; Walker , 1959 ; Childs and Sweetnam , 1961). However, methods used to verify the hypothesis are suspect, as some authors include a family history as part of their diagnosis. Hence if borderline cases are diagnosed as migrainous or not according to family history, it could be possible to provide a tautological

demonstration that migraine is a familial trait.

An alternative to using clinical information for investigating migraine prevalence rates is to conduct epidemiological studies. Epidemiological studies are important because the population from which the cases come is known, a factor uncontrolled in clinical studies. Waters (1970) used an epidemiological approach in an attempt to resolve the basic difficulty of diagnosis. He used a questionnaire to determine the prevalence of the three cardinal migraine symptoms; unilateral headache, warning signs and nausea, without introducing the term migraine. It was found that of subjects diagnosed of migraine based on questionnaire responses, there was a 90% agreement with independent clinical rates. (Waters and O'Connor, 1970).

Waters and O'Connor (1971) employed the questionnaire to investigate 2933 females aged 20-64 years. Subjects with the three migrainous features were identified and the prevalence of migraine was estimated as 19%. In this study, 117 women kept diaries of headache features. Of the 289 headaches reported from the migraine group, all had at least two of the three cardinal features, whereas none of the 243 headaches experienced by the non-migrainous group had any of the three features. Waters and O'Connor (1971) discovered that the incidence of both migraine and non-migrainous features was significantly higher during the menstrual period. Interestingly, 84 migraines occurred within the seven day period after menstruation and 52 occurred in the seven days before menstruation, contradicting the theory that migraine is common

before menstruation. On examining patients who displayed migraine features, patients who did not have migrainous headaches and subjects free of headaches, no evidence of systolic or diastolic blood pressure differences, no social class or intelligence differences or proportion with visual acuity difficulties was found. Waters and O'Connor (1971) interviewed first degree relatives of randomly selected probands with migraine, non-migraine headaches and without headache and found that although first degree relatives of migraineurs did have a higher preponderance of migraine symptomatology, the difference was not statistically significant.

Results from community studies raise the question as to whether the observed migraine symptoms do constitute a distinct clinical entity or whether it is a point on a headache continuum. Current literature has considered migraine as a syndrome of three symptoms and from the respondents of a later study (Waters, 1973), the number of subjects displaying unilateral headache, warning signs and nausea, and the incidence of each feature alone on the basis of chance were calculated. The results suggested that for the combinations of two and three symptoms there was an excess of 'observed' over 'expected' values. However the information was period prevalent data taken over twelve months, and did not indicate the extent to which the features occurred together during one attack. The information did however enable Waters (1973) to compare two theories of migraine in an attempt to account for observed prevalence figures and examine implications for diagnosis. The first of these theories would state that the three

features of migraine may frequently be associated with any types of headaches and hence occur in one subject by chance, migraine may then be observed in the small additional group with three features that cannot be explained by chance occurrence. On this basis, prevalence rates of 1.3% for both males and females would be expected. The alternative theory would state that subjects displaying any of the three features may be considered as having migraine. With this second hypothesis, prevalence rates of 38.3% for men and 57.5% for women would be expected. The prevalence rates obtained in community studies would suggest that neither of these theories adequately account for the observed figures. A third theory was considered by Waters (1973) which stated that the more severe the headache, the greater the probability of occurrence of the three migrainous features. Using this model, it was found that although prevalence rates were not as high as those observed, they were higher than would be expected by chance. In conclusion this study provides evidence to support the concept that migraine might be more appropriately considered as the extreme of a headache continuum, rather than a distinct clinical entity.

This overview of epidemiological methodology, gives an indication of the difficulties involved in attempting to establish prevalence rates for migraine headache. However, given the above difficulties, similar questionnaire studies have been used to examine conventional ideas concerning migraine headache.

Clarke and Waters (1974) investigated prevalence rates in General Practice in Britain. Of 3500 patients sent questionnaires, 88.1% of males and 93.3% females responded. Results indicated that

prevalence of headaches was higher with women than men and declined with age in both. There was also a tendency for the prevalence of headaches to be slightly, but not significantly higher in the 'better' off areas, especially with male respondents. Although it was reported that the average severity of the male responders was not as great as the females, it was determined that within the year prior to questionnaire, an average of a quarter of a working day per man was lost. Thus, for every man aged between 15-64 years, a total of 745 male replies, approximately 186 days were lost due to migraine headaches. Clarke and Waters (1974) calculated that 28.7% of all females and 19.5% of all male patients had migraine.

Clinical studies would indicate that pressure and tension may be significant factors in the precipitation of a migraine attack. On this basis, the prevalence of migraine may be greater in industrialised than rural areas. Mills and Waters (1974) examined this hypothesis by comparing the prevalence of migraine symptoms in the five Isles of Scilly. Of the five, St Mary's is the largest, with the largest population. The results indicated that headaches were more problematical on St Mary's than the smaller islands; however, the authors stress that because of the small populations, the results may be due to chance. A comparison of prevalence rates between the Scilly Isles and London indicated that the Scilly Isles had slightly lower rates although differences were small and not consistent across age groups. Thus the information may be regarded as inconclusive given the differences in population sizes.

An increasing area of concern with migraine, is the effect of oral contraception. Phillips (1971) conducted a study of 57 females

and found a significantly increased incidence of headaches amongst women taking oral contraceptive preparations. The headaches were frequently of a type indistinguishable from classical migraine in that prodromal features, lateralised pain, vomiting and photophobia were present. In others, headaches were accompanied by depressive symptoms and had no features of migraine. Patients with true migraine were generally worse whilst taking oral contraceptives and the headaches often assumed a cyclical pattern in that the commonest time of occurrence was between courses of pills. Kudrow (1975) found that in 70% of cases where oral contraception was discontinued, a reduction in headache frequency occurred.

There have been few epidemiological studies with Schoolchildren, although the area of childhood migraine is of considerable interest, not least from the treatment aspect of the disorder, where potent and potentially addictive drugs are to be avoided.

In a study conducted in a Girls Grammar School, Moss and Waters (1974) found that 93.1% of the students recorded headaches within the previous year, fewest headaches being reported in the 11-12 years age group. Migraine features were also found to be very common, 92.2% of respondents experienced one or two or all three migrainous features within the previous year. No clinical validation of the questionnaire was attempted although a prevalence rate of 28.4% was estimated. Interestingly, the results contradict the notion that the menarch precipitates migraine as a majority of the girls aged 11-12 years who had not reached the menarche were already experiencing headaches.

Small and Waters (1974) conducted a similar study in a mixed sex Comprehensive School. They found that 96.8% of the girls and 91%

of the boys had experienced at least one migraine feature in the previous year.

Although the studies concerning schoolchildren have not been clinically validated, the fact remains that there is a high morbidity from a migrainous type of headache in schoolchildren, prevalence rates being estimated at 31.5% for girls and 26.1% for boys.

Heredity as a feature of migraine has been a major feature of clinical and epidemiological research. Waters (1971) and Adams, Feuerstein and Fowler (1980) have questioned the role of genetic factors in the aetiology of migraine headaches. Although concordance rates of 26% and 13% have been found for monozygotic and dizygotic twins respectively, no shared patterns of precipitants and characteristics of attacks could be found between pairs of twins (Lucas , 1977). Adams et al (1980) concluded that because minimal data are available and contradictory findings as well as methodological inadequacies characterise most of the studies, genetic assumptions are at best tentative.

In summary, this chapter has emphasised;

a. The unsatisfactory nature of present definitions of migraine.

b. That individuals with headache but without migrainous features at any time, are relatively uncommon in the general population, indicating that migraine may not be a completely separate entity from other forms of headache.

c. That using a clinical validation technique, prevalence rates have been found to be fairly uniform across studies, ranging from 15 - 19% in males and 24 - 29% in females.

d. The prevalence of migraine depends mainly on sex and age and

appears independent of social class.

e. Comparable prevalence rates found in Grammar and Comprehensive schoolchildren indicate a lack of association between migraine and intelligence.

f. Migraine does not appear to be associated with occular disorders.

g. Although there appears to be an association between migraine and hormone levels, the exact nature of the relationship has yet to be determined.

h. It appears that the heredity factor in migraine is still an unresolved area.

CHAPTER 4

CEREBRAL AND PERIPHERAL BLOOD FLOW STUDIES IN RELATION TO MIGRAINE HEADACHES.

CEREBRAL AND PERIPHERAL BLOOD FLOW STUDIES IN RELATION TO MIGRAINE HEADACHES

INTRODUCTION

This chapter will assess the rationales underlying the use of temporal artery amplitude and finger temperature biofeedback as treatments for classic migraine.

CEREBRAL BLOOD FLOW IN RELATION TO MIGRAINE HEADACHES

The use of temporal artery amplitude biofeedback as a treatment for migraine is based upon observations that the painful phase is associated with increased temporal artery amplitude pulsations. Reducing the amplitude of the temporal artery is considered to be a method of affecting direct control on the pain producing mechanisms involved in the migraine headache. This section will firstly review physiological experiments that have examined cerebral blood flow changes that occur during the prodromal and painful phases of migraine headaches. The concluding part of the section will briefly examine the relationship between temporal artery vasoreactivity and temporal artery plethysmography.

PERIPHERAL CIRCULATION IN RELATION TO MIGRAINE

The use of finger temperature biofeedback as a treatment for migraine is based on observations that changes in finger blood flow are associated with changes in sympathetic nervous system arousal. A decrease in finger blood flow (and thus finger temperature) is associated with an increase in autonomic arousal and conversely an increase in finger blood flow (and an increase in finger temperature)

with a decrease in autonomic arousal. Learning to increase finger temperature is considered to be an indirect method of decreasing sympathetic arousal and controlling migraine pathology. The first part of the section will examine the relationship between finger temperature and sympathetic arousal. The final part of the section will assess claims that migraine is part of a general dysfunction linked to nervous system difficulties.

CEREBRAL BLOOD FLOW CHANGES IN RELATION TO MIGRAINE

While a primary cause of migraine is still elusive, there is evidence to suggest that the pre-headache phase and the headache itself is associated with changes in intra and extra-cranial arteries. This 'vascular' theory of migraine proposes that the aura phase of the migraine headache is due to constriction of one of the major cerebral arteries and the headache is caused by excessive pulsations of the extra-cranial arteries, specifically the suprorbital, superficial temporal or occipital vessels. (Wolff, 1963).

Apart from the extensive observations by Wolff (summarised by Dalessio , 1972), the vascular theory has received support from Goltman (1936) who observed that a skull defect in a migrainous patient was depressed during the prodromal phase of an attack and bulged during the headache phase. Graham and Wolff (1938) found that an injection of a powerful vasodilator (histamine) immediately after a severe migraine had been eliminated by ergotamine tartrate, caused a transient recurrence of the pain. The headache was observed to be most acute on the side that had been previously involved in the migraine attack and was associated with an increase in amplitude of the temporal artery. Tunis and Wolff (1953) proposed that the pain

during a migraine headache resulted from increased tension within pain sensitive cranial artery walls. They recorded cranial artery pulse waves at intervals prior to, during and after migraine attacks. The artery pulsations were found to be larger during the headache than the non-headache phase and much larger than in subjects with no history of migraine.

Dalessio (1972) observed that although mechanical distension of cranial vessels caused pain, vigorous constriction of arterial walls after local application of adrenaline was unaccompanied by pain. These results would concur with the theory that vasoconstriction is unaccompanied by pain and that vasodilatation is associated with pain.

To further strengthen this position, Skinhøj and Paulson (1969) reported details of a case in which cerebral blood flow was measured unilaterally during the prodromal phase of a migraine attack. A mean reduction in blood flow of 51% was recorded, the greatest reduction was found in the area corresponding to the aura symptoms. Patients examined during the prodromal and headache phases displayed reduced cortical perfusion; in some areas to levels known to be critical for adequate oxygenation.

In conclusion, the above studies support the vascular theory of migraine which proposes that intracerebral vasoconstriction causes hypoxia and ischæmia thus precipitating prodromal symptoms, whilst the rebound vasodilatory phase causes the headache. Recent experimental evidence however, would question the simplicity of this picture.

O'Brien (1971) examined patients who experienced pre-headache symptoms during the painful phase of their migraine attacks.

Bilateral measurements indicated that the aura phase of the migraine was associated with reduced cortical perfusion, but as a general and not a local finding. The headache phase was found to be associated with a small increase in cortical perfusion characteristic of reactive hyperaemia. O'Brien (1973) determined that although the aura phase is associated with generalised cerebral vasoconstriction, the degree may be closely associated with, but is probably not the cause of the phase and that an unidentified third factor may be influential. O'Brien (1973) also stated that vasoconstriction occurs in the preheadache phase of both classic and common migraine and may thus be asymptomatic as regards classification, but an accidental clinical expression of a more widespread process. Extra-cranial dilatation was also observed to be a feature of headaches in patients who did not display prodromal symptoms prior to the onset of the pain. O'Brien (1973) therefore concluded that attacks of migraine are probably biphasic in relation to blood flow changes but that in only a proportion is vasoconstriction associated with symptomatology. The classification of migraine into 'classical' and 'common' varieties, depending on the presence of aura symptoms may thus have no patho-physiological correlate.

Norris, Hachinski and Cooper (1975) observed global decreases of cerebral blood flow in the carotid artery territory during the aura phase of a migraine and considerable perfusion in the headache phase of the same attack. Unexpectedly, ergotamine tartrate administered intra-muscularly, brought definite relief of symptoms with no accompanying change in cerebral blood flow.

Although, as the above studies would indicate, there appears

to be considerable evidence for vasomotor disturbance being associated with migraine headaches, the exact mechanisms and the temporal and spacial aspects of blood flow disturbances remain unresolved.

Attention has been centred upon the increased pulsations and dilatation of proximal parts of the temporal artery because of the association between pain and observed changes in cerebral vasculature. Heyck (1970), however, noted that the 'vascular' theory would not account for the paleness often displayed by patients suffering from migraine headaches. Further, he stated that gross oedema is rarely associated with the painful phase but that it is common to have laxity of tone characterised by sagging soft flesh around the forehead and temples and also a dulled appearance of the cornea. These features would indicate a filling defect in the peripheral vessels and a diminished capillary blood flow, notwithstanding the increased pulsation and prominence of the temporal artery which seems paradoxical.

Whilst observing one patient during a migraine attack, it was noticed that blood extracted from the swollen frontal vein was bright red and indistinguishable from arterial blood. Secondly, the vein could hardly be collapsed despite continual withdrawal of blood, suggesting continual replenishment. On the basis of the above observations, Heyck (1970) suspected the existence of a transient arteriovenous 'shunt' network to be responsible for the pain.

To test the 'shunt' hypothesis, seven migraine and seven non-migraine controls were investigated; arterio-venous oxygen saturation differences between arterial and venous blood flow in cerebral vessels were compared with arterial blood from the arm as a control. Comparisons were made both during the after headaches. The results

suggested that most of the circulating blood being withdrawn from capillary beds had been excluded from metabolic exchange. The scalp blood flow was therefore in excess of its metabolic requirements, being increased in the scalp during a migraine attack even though skin capillaries were constricted. Heyck (1970) proposed that the 'shunt' hypothesis would be the most plausible explanation for this phenomenon since it accounts for the rise in total blood flow associated with the increased pulsation of the temporal artery and the distension of larger veins.

The increase in total blood flow would lead to increased pulsation and distension of the feeding artery as well as to distension of the veins because of abnormally high pressures transmitted through the 'shunt'. At the same time it would produce ischaemia over a wide area of the neighbouring capillary network.

No evidence of 'shunts' were found during attack free periods and Heyck (1970) proposed that a large number of bridging vessels, normally closed, become patent when an attack of migraine is initiated. The opening of arterio-venous 'shunts' in the scalp and possibly in the meninges causes higher arterial pressure and an increase in pulse amplitude, not only in larger arteries (where Heyck (1970) stated it does little harm) but also in thin walled arteries lying in front of the 'shunts'. Thin walled arterioles are thus subject to severe distension, which together with capillary ischemia , pain threshold lowering hypoxia and acidosis, could be sufficient cause of pain.

In all of the above studies, migraine headaches have been the exclusive focus of attention. Mathew, Hrastnik and Meyer (1976)

investigated patients with different types of headaches and found that regional blood flow varied with clinical phase in migraine headache patients alone, and that such variations did not characterise non-migrainous headaches. During the prodrome, blood flow was reduced and during the headache phase, increased cortical perfusion was noted. Those patients who continued to show neurologic signs during the headache phase did not display standard cerebrovascular changes as mixed patterns of reduced and increased blood flow were observed.

Friedman (1976) stated that vasodilatation alone cannot explain the painful phase of migraine as the headache is aggravated by coughing, and the valsalva test, which would infer that a venous component is involved. Edmeads (1977) measured cerebral blood flow during attacks of migraine and observed decreases in flow during the aura phase and increased flow during the headache phase. However, the distribution in time and space of blood flow changes did not always correlate with the clinical features of the attack.

This complex clinical picture was reviewed by Blau (1978) who concluded that the extra-cranial hypothesis was an oversimplification of the mechanisms involved in migraine headache. Blau's main criticism centred around the observation that when the patient is symptom free, arterial pulsations are of the same magnitude as when the headache is graded 7 on a 1-10 scale of headache severity. Although neglecting the possibility that the abruptness of the dilatation may be the precipitant of the pain, Blau (1978) concluded that the symptoms emerge from calibre variation in the lepromeningeal circulation. Vasoreactivity of the pial vessels excite or inhibit

the cortex locally to produce sensory symptoms, this is then succeeded by a phase of more diffuse alteration in the meningeal blood flow arteries, capillaries and dural sinuses.

Although the pattern of events as conceptualised by Blau (1978) differs from Wolff's original hypothesis as to the site of the pain mechanism; meningeal compared to extracranial vessels, experimental support for Blau's position is lacking.

In the light of the above evidence it would appear that focusing exclusively on extracranial vessels as the site of pain production during a migraine attack may be unwarranted. Certainly, before conclusions regarding the therapeutic mechanisms of temporal artery biofeedback can be reached, more should be known about vaso-reactivity characteristics of extracranial vessels. At present there is a dearth of information. Sokolov (1963) reported that when a volume plethysmograph is placed over the temporal artery, increases in blood volume were observed when novel or alerting stimuli were presented and decreases when the stimuli were painful or threatening.

Although plethysmographic recordings are a popular method of investigating vascular activity, difficulties in interpretation arise from the fact that both venous and arterial sides of the vascular bed are both under sympathetic control. The plethysmograph therefore provides a measure of sympathetic nervous system activity without discriminating between the relative influences of different component vessels in the vascular bed. Ideally, a measure of arteriolar tone, venous tone, and the distribution of blood between the true capillaries and other vessels would be required in order to relate these variables to the behaviour and psychological state of the subject (Cook, 1974).

In conclusion, the vascular theory of migraine would appear unable to account for the spatial and temporal aspects of cerebral blood flow in relation to the aura and pain phases of migraine. The site and pain mechanisms involved in migraine are also questioned, an important point when considering the rationale underlying temporal artery biofeedback. Given the lack of knowledge concerning cerebral vasoreactivity and the indication that plethysmographic recording provides information regarding gross sympathetic change, evidence about the mechanisms underlying temporal artery biofeedback is lacking.

PERIPHERAL CIRCULATION AND MIGRAINE HEADACHES

A considerable amount of psychophysiological research has been conducted upon peripheral circulation as an index of sympathetic nervous system activity. Although later chapters will deal in detail with parametric studies and the clinical application of finger temperature biofeedback, an evaluation of the rationale underlying this therapeutic approach will be undertaken.

Much research has been carried out on peripheral blood flow because it is a system responsive to many stimuli; because of its activity varying over a wide normal range without danger to the organism, and because it is a convenient model for the study of autonomic learning (Lynch and Schuri , 1978). Specific interest with respect to migraine was suggested by Sargent, Green and Walters (1972) who observed that in a migraine headache patient, the spontaneous recovery from a migraine attack coincided with a considerable flushing in her hands, resulting in a 10°F rise in finger temperature in two

minutes. Further studies using finger temperature control for the alleviation of migraine headaches have met with varying success (details in Chapter 7).

The rationale employed by Sargent, Green and Walters (1973b) for the use of finger temperature biofeedback in the treatment of migraine is based upon the limbic system as being the major responder to psychologic stress (Papez ,1937). Psychosomatic problems are considered to become chronic somatic processes through the numerous interconnections between the limbic system and autonomic control centres of the mid-brain. The chain of events may be conceptualised as follows: psychologic response - limbic response - hypothalamic response - autonomic response, leading to a somatic response. In the case of migraine (which would appear to be part of a stress related syndrome) the somatic response is a dysfunction of vascular behaviour in the head related to intense sympathetic overactivation. Vaso-constriction in the hands is a function of sympathetic activity and vasodilatation is a one variable indication of decreased sympathetic outflow. Sargent, Green and Walters (1973b)thus concluded that fingerwarming is effective in the amelioration of migraine because patients are learning to 'turn off' excessive sympathetic outflow.

Much of the research on peripheral circulation with respect to migraine has taken two lines of enquiry; firstly, whether peripheral blood flow does provide an adequate index of sympathetic nervous system activity and secondly, whether migraine is part of a general vascular dysfunction.

Lynch and Schuri (1978) stated that peripheral circulation includes two distinct regions, the skin and skeletal musculature.

The primary function of the muscle circulation is to supply essential nutrients to the working muscle and remove waste products and heat resulting from the work. The skin circulation has the primary role of dissipating and conserving heat in order to maintain a constant internal temperature.

The vascular structures concerned with heating the skin consist of an extensive subcutaneous venous plexus, which holds large quantities of blood that can heat the surface of the skin, and in some skin areas, arteriovenous anastomoses, which are large vascular communications directly between the arteries and venous plexuses. When constricted, the anastomoses reduce blood flow into the venous plexuses and when dilated they allow extremely rapid blood flow into the plexuses (Guyton , 1971). The arteriovenous anastomoses do not appear to be under metabolic control but seem to be governed chiefly by the central nervous system and reflex influences from temperature receptors (Detweiler , 1973). Abrahamson (1967) stated that vascularity in the fingers is developed beyond the degree necessary for nutrition of the comparatively thin epidermis, indicating its role in the control of thermal states. Plutchik (1956) stated that using finger temperature as an index of sympathetic arousal is justifiable as although the exchange of heat between the body and the environment occurs over the entire surface, measures of skin temperature of the fingers and toes serve as the most sensitive indices of blood flow changes in superficial vessels. Cook (1974) concluded that many studies confirm that the vasomotor control of the fingers is achieved by the release of vasoconstrictor tone and in none has any evidence for vasodilator control been found. Reduced sympathetic tone can in fact

induce vasodilatation equivalent to that produced by a complete nerve block.

In conclusion it would appear that the circulation of the fingers does provide a sensitive indication of the level of activation of the sympathetic nervous system.

Investigations into whether migraine is part of a generalised disturbance of the peripheral circulation, however, are less conclusive.

As previously stated, the common conception of migraine headache is that it is due to dilation of the extra-cerebral arteries of the dura mater and scalp. Schumacher and Wolff (1941) stated that the therapeutic effect of ergotamine tartrate is thought to be through its vasoconstrictive effects on cerebral vessels. It has also been considered that the characteristic vascular changes associated with migraine might not be limited to the cranial circulation but perhaps be a generalised dysfunction linked to sympathetic nervous system difficulties.

Initially, Kerslake and Cooper (1950) observed that blood flow to the extremities increases when the body trunk is heated; a response to control internal temperature. The hypothesis under consideration is that if migraineurs have deficient vasomotor control then they will exhibit abnormal responses when compared with a control group under experimental conditions. Appenzeller, Davison and Marshall (1963) placed a heat cradle containing six 100 watt bulbs over the chests of ten migraineurs and ten non-migraine control subjects. A venous occlusion plethysmograph placed around the right arm of the subjects monitored vascular responses. Although eight of the ten migraine subjects did not exhibit responses comparable with

those observed in the non-migraine controls, an unequal sex distribution between the groups is considered to have influenced the results (Morley , 1977). Downey and Frewin (1972) measured the responses of migraine patients and normal controls to cooling one hand. Apart from the mean resting blood flow in the hands of migraineurs being higher than in the non-migraine controls, the cold stimulus test indicated a diminished level of reactivity in the vessels of migraine patients. Elliot, Frewin and Downey (1974) recorded the response of oral temperature and hand heat elimination in a group of normal females, normal females on oral contraceptives and a group of female migraine sufferers, to placing one arm in a bath of warm water. It was noted that older migraine patients had a diminished vascular response, although the remaining migraine patients displayed normal vasodilator responses to heating. Morley (1977) concluded that investigations of generalised vasodysfunction in migraine patients were characterised by several methodological shortcomings. The lack of controlled subject selectivity with respect to age, sex, duration of migraine, medication and menstrual cycle; unclear specification of diagnostic criteria, inadequate statistical treatment of results, and a failure to define the experimental phenomenon under investigation, render the results equivocal.

In conclusion, although there is substantial agreement that finger temperature is a sensitive indicator of sympathetic nervous arousal, it remains unclear if migraine is part of a general dysfunction linked to sympathetic nervous system activities. Until more is known about vasomotor function in migraine, it is not possible to conclude whether increased sympathetic nervous system activity is the cause of a migraine or a result of the discomfort experienced.

CHAPTER 5

BIOCHEMICAL BASIS OF MIGRAINE HEADACHES.

BIOCHEMICAL BASIS OF MIGRAINE HEADACHE

INTRODUCTION

From the previous chapter reviewing the role of cerebral blood flow changes in migraine headache, there is strong evidence to suggest a biphasic phenomenon associated with the Classical Migraine crisis. Prodromal and painful phases of the headache correlate with cerebrovascular vasoconstriction and vasodilatation respectively. It would also appear that the vasoconstriction associated with the aura phase manifests not only in intra and extra-cranial vessels, but also in sympathetically innervated peripheral vessels, producing cold extremities such as fingers and toes. Hence, observed physiological changes associated with a migraine crisis have implied the existence of a link between the sympathetic nervous system and migraine headaches.

The types of aura and the site of the pain experienced by migraineurs would, however, appear to have little patho-physiological correlation with the temporal and spatial aspects of observed blood flow changes. It has thus been concluded that generalised cerebrovasodilatation may be associated with, but is probably not the cause of, the painful phase and that a third and as yet unidentified factor may be influential (O'Brien , 1973).

When the variety of triggering mechanisms thought able to precipitate a migrainous headache is considered, including hormonal and dietary factors, it is not surprising that biochemical theories for migraine are prevalent. Support for a biochemical component in the pathogenesis of migraine is offered by Sicuteri (1979) who stated that spontaneous vasodilatation and scalp muscle contraction, even if intense and long lasting, are in themselves incapable of producing

pain in normal subjects. Vasodilating drugs such as papaverine and amyl nitrite may provoke intense relaxation and pulsation of arteries but not induce pain. Only those drugs capable of acting on both receptors of pain and the smooth muscle of vessels, such as histamine, kinin and prostaglandin, can provoke vasodilatation and induce pain. These drugs lower pain thresholds which locally render otherwise silent pulsations painful.

Evidence for biochemical factors in migraine headache is available from both clinical and experimental sources. This chapter will deal with some studies representative of the prevalent areas of research in an attempt to outline current trends in biochemical investigation of migraine headaches.

DIETARY FACTORS

In approximately 25% of 339 migraineurs, dietary factors such as fats, fried foods, chocolates and oranges have been implicated as precipitants of migraine headaches, (Selby and Lance , 1960). Hanington (1967) stated that 30% of migraine headaches were associated with the ingestion of chocolate, cheese and alcohol, all of which contain tyramine. However, Adams, Feuerstein and Fowler (1980) reported that the percentage said to be precipitated by these factors seems excessive and has not been reported by other researchers.

ORAL CONTRACEPTION

Oral contraceptives, pregnancy and the menstrual cycle have received considerable attention as precipitants of migraine (Selby and Lance , 1960 ; Kudrow , 1975). The use of oral contraceptives apparently increases the frequency of migraine headaches (Carroll

1971) and relief from migrainous headaches has been associated with the cessation of oral contraception and the decrease of maintenance oestrogen (Kudrow , 1975).

Lance and Anthony (1966) reported that during pregnancy, 64% of those women whose headaches had previously occurred during menses and 48% of those women whose headache occurred throughout the menstrual cycle experienced relief from their headaches. In general, various studies indicate a 60-80% remission or improvement in migraine headaches during pregnancy (Lance , 1969 ; Epstein, Hockaday and Hockaday , 1975). Among the changes that accompany pregnancy, intrinsic oestrogen levels increase. As the above studies report that decreases in œstrogen levels are associated with decreases in migraine headaches, the results render the role of oestrogen levels unclear. Further research is thus required to determine the specific role that changes in intrinsic and extrinsic oestrogen levels play in migraine.

SEROTONIN

It has been hypothesised that a variety of vasoactive substances including plasmakinin, serotonin and histamine, play critical roles in the aetiology of migraine (Fanchamps , 1974), fluctuations in the levels of these agents perhaps initiating vasomotor changes characteristic of the migraine attack. Of the substances mentioned above, serotonin has received the most attention and, because of serotonin's vasoconstrictive action, it has been suggested that a release of platelet serotonin results in the vasoconstriction associated with the prodromal phase of migraine. The subsequent depletion of platelet serotonin, decreasing the normal

tone of the arteries, would precipitate a passive distention of the arterial walls. In conjunction with the activity of serotonin, plasmakinin is thought to be synthesised, and this would reduce the pain threshold of the receptors in the affected vessels (Fanchamps, 1974).

The importance of the role of serotonin has been inferred from a number of studies. Dalessio (1972) reported on a series of studies conducted in a New York Hospital where a programme monitoring variations in fluid and electrolyte excretion in association with vascular headaches of the migrainous type was undertaken. The results indicated that there was a decreased rate of water, sodium, potassium and creatinine excretion prior to and during the early phases of the headache. Increased excretion of water, sodium and potassium were usual with the subsidence of the headache. These observations were linked to the action and influence of serotonin, as it not only causes vasoconstriction but also has antidiuretic and emetic effects. Slight rises in plasma serotonin levels have been found at the start of migraine attacks, but more convincing marked falls have been noted at the onset of the headache phase (Curran, Hinterberger and Lance , 1965). These changes appear specifically with migraine headaches and have not been observed in other equally severe but non-migrainous headaches.

Further support for the role of serotonin in migraine has been provided by the observed action of reserpine, known to lower plasma serotonin concentration and found to precipitate attacks in those liable to migraine. Phenelzine on the other hand is a monoamine oxidase inhibitor which increases endogenous serotonin production and

is claimed to reduce frequency and severity of attacks. Serotonin given intravenously has also been observed to alleviate migraine attacks (Kimball, Friedman and Vallejo , 1960). Dexter and Riley (1975) observed decreased plasma serotonin levels at the onset of migraine pain in three patients reporting nocturnal migraine. Although elevated serotonin levels result in vasoconstriction and possible initiation of the prodromal phase, consistent reductions (60-80%) in platelet serotonin have been reported during the actual headache phase (Curran, Hinterberger and Lance , 1967). Observations have also shown that patients with vascular headache have abnormalities in platelet serotonin content (Deshmukh and Meyer , 1977), and metabolism (Hilton and Cuming , 1972), and it has been demonstrated that platelet monoamine oxidase activity during migraine is temporarily defective (Sandler, Youdim and Hanington , 1974). These data, with clinical findings that methysergide (a serotonin antagonist) tends to reduce the frequency of migraine attacks, provide support for the role of serotonin in migraine activity.

In an attempt to explain why only some patients should experience migraine headaches, Sicuteri (1979) described three categories of person; hypnoceptors, eunoceptors and hypernoceptors. Hypnoceptors are characterised by high pain thresholds whilst hypernoceptors experience pain on slight stimulation and to a degree in excess of that experienced by the eunoceptor category of patient. Sicuteri (1979), however, does not attempt to test the validity of these conceptual categories, but apparently relies upon the precipitation of migraine as a defining feature.

Awakening in the early morning with a headache is one of the commonest modes of migraine onset (Williams ,1966). Hsu, Crisp,

Kalucy, Koval, Chen, Carruthers and Zilkha (1978) studied plasma levels of noradrenaline and adrenaline, the serotonin precursor tryptophan, glucose, insulin and free fatty acid levels in 19 patients who frequently experienced migraine on wakening. Results showed that plasma total and free tryptophan, glucose, insulin and free fatty acid levels were not significantly different in the migraine group compared with a non migraine control group. However, raised catecholamine levels in the three hours preceding awakening with migraine were considered to indicate the presence of an as yet unidentified stress factor pervading sleep. Sicuteri's (1967) finding that urinary excretion of 3 Methoxy - 4 Hydroxy Mandelic Acid (VMA) was increased during a migraine attack would be consistent with these findings. Failure to find any correlation between plasma tryptophan levels, whether free or bound, and migraine suggests that the role of tryptophan, if any, in the genesis of migraine is unlikely to be a direct one; a conclusion contradictory to studies implicating the role of serotonin in migraine.

The studies considered so far have concentrated mainly upon assays of excreted serotonin metabolites for information about biochemical activity associated with migraine headaches. Poloni, Nappi, Arrigo and Savoldi (1974) measured cerebro-spinal fluid 5-hydroxyindolacetic acid (5-HIAA) levels in patients during spontaneous migraine attacks, headache free periods and following treatment with L-tryptophan. The results showed no differences in 5-HIAA levels between migraine patients in headache free periods and the controls. Although the values obtained during a spontaneous migraine attack appeared to be lower than directly preceding the attack, the decreases did not attain statistical significance. Moreover, all values

recorded were found to be within normal limits. Clinical results however indicated that prolonged L-tryptophan treatment was found to prevent migraine attacks in three out of five migraineurs. It was concluded that the therapeutic results could not be explained merely on the basis of tryptophan administration and its ability to increase the synthesis of brain 5-HT. These findings may thus illustrate the existence of a third factor, combining locally with serotonin at the site of the pain or even acting independently directly upon dilated vessels in an inflammatory fashion.

PLASMA KININ

Wolff (1963) and colleagues attempted to identify such a third factor. They collected fluid from subsurface tissue at the site of local pain of a patient experiencing a migraine attack. A kinin was identified which induced vasodilatation, lowered pain threshold, increased capillary permeability and increased vulnerability of the tissue to injury. Later experiments showed that this kinin was also liberated during neuronal excitation, although such observations could not be accomplished with ease in the average patient. When kinin specimens were obtained, Dalessio (1976) was unable to identify other mediators which he considered were undoubtedly operative at the inflammatory sites.

In conclusion, it appears that although the painful phase of migraine is characterised by vasodilatation, this in itself does not produce the painful experience. Experiments such as the above suggest that a variety of vasoactive substances precipitate vasodilatation and combine locally around distended blood vessels, this combination of vasodilatation and sterile inflammation producing a migraine headache.

In all of the above studies, psychological disposition has received scant attention as a mediator or precipitant of biochemical change. Given the importance of stress, anxiety and various other emotional states purporting to be potent triggering mechanisms for the precipitation of migraine headaches (Pearce ,1971), the biochemical picture may be considered incomplete.

CHAPTER 6

PHARMACOLOGICAL ASPECTS OF THE TREATMENT AND MANAGEMENT OF MIGRAINE HEADACHES.

PHARMACOLOGICAL ASPECTS OF THE MANAGEMENT AND TREATMENT OF MIGRAINE HEADACHES

INTRODUCTION

According to conventional theories (Wolff 1963), migraine headache is considered to be a peripheral disorder, provoked by noxious agents in the vessels of the scalp, meninges and extra and intra-cranial tissues. Thus, painful stimuli are considered to be mechanical: sphygmic waves on an otherwise distended extra-cranial artery. Later studies provided evidence for the involvement of other pain producing substances, such as neurokinin and serotonin, of which the latter has received considerable attention. (Sicuteri, 1979).

Previous chapters have included reviews of these theories, and although a considerable amount of empirical data is available, a unitary theory explaining the occurrence of migraine is still elusive. However, the major types of drugs used in the control of migraine reflect the importance attached to the mechanical and serotonin theories.

This chapter will not attempt to provide an exhaustive review of the relative merits of different drug therapies, but is designed to draw attention to the disadvantages of managing migraine with drugs.

Migraine is characterised by severe headache so one might assume that analgesics would play an important role in its treatment. Fanchamps (1975) however, states that the effect of analgesics upon conventional migraine attacks is in fact very poor. As a consequence of this, the following most frequently administered drugs will be described in terms of their proposed mode of action, their effective-

ness in clinical trials, and their side effects. These drugs are: ergotamine tartrate, methysergide, propranolol, clonidine and pizotifen.

ERGOTAMINE TARTRATE

Ergotamine tartrate is the compound which, so far, has given the best results in the treatment of migraine attacks. It is an open question whether its therapeutic effects are due solely to its vasoconstrictor action or whether other pharmacological actions are involved (Sicuteri, 1959). On testing the compound on 18 patients, Sicuteri (1959) considered it to have considerable therapeutic and prophylactic potential. Ryan (1970) stated that ergotamine tartrate is not habit forming, and when it is administered with caffeine (itself a vasoconstrictor), vasoconstrictor effects appear to be increased with lowered doses of ergotamine. Hence, Ryan (1970) considered its mode of action to be vasoconstrictive, restoring cerebral vascular tone and thus interrupting the pain producing mechanism. The side effects noted by Ryan (1970) consisted almost entirely of gastrointestinal disturbances, including nausea, vomiting and stomach cramps.

Although Waters (1970) found that ergotamine tartrate was not as effective as a placebo, Wilkinson and Wall (1973) stated that in acute attacks, intra-muscular injections of ergotamine tartrate are effective in over 70% of patients. This study indicated that an excess of ergotamine could produce ill effects, which went against the original recommendation by Sicuteri (1959) that the use of ergotamine administered prophylactically would be beneficial. Side effects observed through over-administration of ergotamine included nausea,

vomiting and listlessness. Another feature of excess ergotamine tartrate is, paradoxically, the precipitation of migrainous type headaches which can only be relieved by further doses of ergotamine tartrate. Termination of ergotamine tartrate therapy also causes withdrawal headaches which are again indistinguishable from migraines and can last up to 72 hours (Lucas and Falkowski , 1973).

Prolonged arteriospasm after overadministration of ergotamine tartrate has also been observed (Byrne-Quinn , 1964). In this case, 12 mg Migril tablets consumed daily for six weeks caused a patient's feet to become numb and discoloured with no pulse evident below the femorals. Cyanosed fingers and toes have also been observed.

Bross, Cisek, Czereda and Kozminski (1963) described the case of a patient who developed gangrenous ulcers on the feet after taking ergometrine for abnormal bleeding after the birth of a child. Arterial insufficiency cleared after ergometrine had been withdrawn and the patient had been bed rested for three months. Patients displaying ergotism have also been found to have a higher incidence of other disorders, particularly diabetes and collagen diseases (Hokkanen, Kallansanta and Waltimo , 1974).

METHYSERGIDE

Methysergide is prescribed as a prophylactic medication in the management of migraine. Although methysergide is classed as a drug which inhibits the action of serotonin on receptor sites and not as a vasoconstrictor (Bakal , 1975), there are reports of vascular complications (Rackley, Mengel, Pomerantz and McIntosh , 1966). Acute ischaemia of the lower extremities due to severe arterial constriction has been reported (Johnson , 1966), and retroperitoneal fibrosis and

tolerance leading to a steadily increasing consumption of the drug have also been observed (Lucas and Falkowski , 1973).

Rackley, Mengel, Pomerantz and McIntosh (1966) reported unusual side effects with two patients using methysergide. The patients developed chest pains and vasospasm of the lower limbs, the chest pains being relieved by administration of nitroglycerine and carotid massage. Discontinuing methysergide caused the disappearance of the chest pains and a return of the pulses in the lower extremities. Johnson (1966) observed that the side effects of methysergide were usually mild and transient: they included nausea, giddiness, dizziness, epigastric distress, insomnia, difficulty in concentrating, limb pains and intermittent claudication accompanied by cramping extremity pains. On cessation of the drug, acute peripheral ischaemic reactions required five or more days to subside, and rebound headaches complicated the patient's recovery. Lucas and Falkowski (1973) reported on five patients who displayed cyanosed fingers and weak peripheral pulses, pruritis of the hands, burning and watering of the eyes and a continual urge to defecate. One patient gave clear signs of developing withdrawal headaches on termination of methysergide therapy, after tolerance to the drug had necessitated increased dosage.

PROPRANOLOL

Propranolol is classified as a drug whose action is to block beta-adrenergic receptor sites in blood vessels and therefore prevent vasodilatation. It is considered to be most effective when used prophylactically. Wideroe and Vigander (1974) compared propranolol with a placebo in a double blind clinical trial and found that

propranolol reduced headache frequency to a significantly greater extent than the placebo. Borgensen, Lanng and Eckart-Moller (1974) employed a double-blind, single cross-over trial to compare propranolol with a placebo. The results indicated that propranolol was slightly better in reducing mean number of attacks than the placebo: a reduction to 75% of the pretreatment level with the placebo compared with a reduction to 66% of the pretreatment level with propranolol. However nine of the thirty subjects using propranolol were made worse.

CLONIDINE

Clonidine was originally used as a hypotensive drug as it was thought to act on the central sympathetic outflow (Wilkinson, Neylan and Rowsell , 1972). Later it was found to have a direct effect on reducing the responsiveness of peripheral blood vessels (Heathfield and Raiman , 1972). It was first used as a prophylactic treatment for migraine in 1969 (Wilkinson et al, 1972). Studies have shown that 70% of those patients whose migraine is precipitated by the ingestion of tyramine-containing foodstuffs are helped by this drug (Wilkinson et al, 1972). Although encouraging results regarding the therapeutic efficacy of the drug have been reported (Sjaastad ,1972), subsequent findings have cast doubt on it. Shaw and Saunders (1972) used a double blind, cross-over trial to evaluate the efficacy of clonidine compared with a placebo, the results of which failed to attribute any therapeutic benefits to clonidine. Neither frequency nor duration of attacks diminished and the authors were at pains to point out that any potential benefits were not obscured by inclusion in the trial of patients whose excessively frequent and prolonged attacks might be attributed to emotional factors. It was

however concluded that "many patients improved with the passage of time regardless of treatment, perhaps due to the special interest that had been taken in them, the opportunity to discuss problems and the hope that the cure was just around the corner".

PIZOTIFEN

Pizotifen is used as a prophylactic medication. Initial pharmacological reports suggest that it has a marked anti-serotonin activity and a powerful antihistamine effect. Unlike methysergide it has no effect on pain produced by the synergistic action of serotonin and bradykinin on vessels. Side effects include drowsiness, weight gain and mental depression, which constitute the commonest causes of subjects defaulting in clinical trials. All controlled studies have shown pizotifen to be a significantly more effective therapeutic agent than placebo, but less effective than methysergide. In other forms of headache - tension, post traumatic, inflammatory and trigeminal neuralgia - pizotifen has generally had little effect (Speight and Avery , 1972).

CONCLUSION

This brief review is an outline of some of the most popularly recommended and administered drugs in the acute treatment and prophylactic management of migraine headaches. As yet, no effective treatment has been developed that does not have significant side effects and risks that seriously affect the patients' acceptance. The plethora of drugs available, of which the above are a sample, is evidence of the lack of a truly successful treatment of migraine (Sargent, Green and Walters , 1973b). Although some are effective in

some respects; such as the vasoconstrictor and serotonin inhibitory effects of ergotamine and methysergide, dependence, side effects, withdrawal headaches and peripheral arterial complications made continued use inadvisable. Bakal (1975) would contend that the pharmacological prevention of headache is only successful for as long as the patient continues to use it. Given the disadvantages and limitations of drug therapies as outlined above, and the fact that pain relief may be a 'green light' for some patients to undergo further those stresses that originally precipitated the headache, an alternative approach to the control of migraine headache would appear warranted.

CHAPTER 7.

LITERATURE REVIEW.

SECTION 1 : EMPIRICAL STUDIES.

SECTION 2 : CLINICAL STUDIES.

SECTION 1

CONTENTS

BIOFEEDBACK LITERATURE REVIEW

INTRODUCTION

Wide public interest has been aroused over the past ten years by the development of biofeedback techniques. It not only offers a method of investigation into the control of internal physiological processes, but also suggests the potential benefit in applying these techniques for the amelioration of certain disorders of physiological functioning.

This review will be confined to two major aspects of the biofeedback literature:

Section 1. An appraisal of the empirical studies concerned with demonstration of volitional peripheral temperature control (finger and differential earlobe temperature), temporal artery amplitude and digital pulse amplitude control.

Section 2. An appraisal of the literature relating the clinical application of volitional finger temperature control, temporal artery amplitude control and relaxation techniques in the amelioration of migraine headaches.

A. Biofeedback : Methodological Issues

Although this review will consider both peripheral temperature and temporal artery amplitude biofeedback studies, the relatively extensive empirical literature available on finger temperature control lends itself to highlighting methodological and procedural aspects of biofeedback research. This is not to say that temporal artery amplitude studies are not subject to the same issues, merely that they are still to be reported extensively in the literature.

Finger temperature is a variable of considerable interest to biofeedback researchers since peripheral temperature cannot only be measured with near perfect validity and reliability, but the changes which do occur are slow and hence 'noise' relative to 'signal' is low. Although it would seem to be a straight forward matter to determine whether finger temperature can be brought under voluntary control given the complexity and sensitivity of the biofeedback equipment available, there are difficulties inherent in such investigative procedures. The following section will outline some of the procedural and methodological factors.

One of the most important independent variables in biofeedback research is the display of the function under investigation. Display modalities are visual, auditory, digital and occasionally tactual; within each modality there is a wide variety of possible feedback displays. An appreciation of the complexity of possible feedback combinations can be acquired by considering a simple taxonomy of feedback displays which could be applied across the four modalites mentioned (visual, auditory, digital and tactual):

Within versus end of trial feedback : information presented 'within' the trial period to monitor ongoing progress, or

presented on conclusion of the trial period as an assessment of overall performance.

Proportional versus binary change : presentation of a signal whose value is analogous to the activity level of the function under investigation (often referred to as 'analogue' feedback). Proportional feedback is a quantitative measure of change whereas binary feedback is a qualitative measure giving information that change is apparent, but not the amount of change.

Continuous versus non continuous feedback : information presented instantaneously may be considered as being continuous; information presented at discrete intervals representing the state of activity since the last reading was presented, may be considered to be non continuous information.

The aim of biofeedback is to present clear and potentially useable information to the subject. 'Continuous' feedback of a rapidly fluctuating physiological function may not provide meaningful information to the subject, even though the information may be detailed and accurate. Similarly, 'Binary' feedback may provide sufficient information if a set criterion of change is desired, (e.g. to increase or decrease finger temperature) but not if quantitative aspects of the subjects' performance are under observation. Hence it is important to consider the purpose of biofeedback in determining the type of biofeedback display to be used.

There is a dearth of information regarding the empirical merits of the above displays and parameters. Information that is available covers a diversity of physiological functions; E.E.G., (Lubar and Bahler ,1976) ; Frontalis muscle activity (Budzynski and Stoyva

1969) ; Heart rate increases and decreases (Blanchard, Scott, Young and Haynes , 1974 and Colgan , 1977). Yates (1980), in a review of the literature regarding the efficacy of different types of feedback, states that unsystematic approaches to the problem have not enabled any clear results to emerge. Certainly, an unsatisfactory situation arises when biofeedback from a specific system is presented to a subject, and little is known about the reactive effects of measurement devices on that system, and of other possible counfounding factors.

Before further considering methodological issues related to biofeedback and especially finger temperature control studies, it should be made clear that measures of finger temperature are not linearly related to finger blood volume. At lower finger temperatures, relatively small increases in blood volume can cause large increases in temperature. When skin temperature has reached around 34°C, further considerable increases in blood flow are accompanied by small rises in temperature (Felder, Russ, Montgomery, and Horowitz , 1954). Given that finger temperature cannot be used to measure absolute blood flow changes in the finger and that 'thermal lag' effects delay indications of change, skin temperature recordings may still give valuable information regarding peripheral circulation when changes are constant over several minutes (Lynch and Schuri , 1978).

Although skin temperature changes constant over several minutes may give information about peripheral circulation,methodological issues regarding the acquisition and interpretation of this information should be considered. One important and influential variable is the phenomenon of naturally occurring variation or the 'drift' effect. In peripheral blood flow studies, drift is often observed as a gradual decrease in temperature, especially obvious in the fingers.

It has been noted that ambient temperature has a differential effect upon the rate of drift in finger temperature studies; the lower the ambient temperature, the faster the rate of drift. The phenomenon of drift is investigated in a pilot study included in this thesis. The importance of drift effects when interpreting physiological information is obvious. Differential influences of drift on the voluntary control of finger temperature could lead to situations where decrease values are accentuated and increase values are minimised; hence the importance of control and baseline information. Yates (1980) recommends two methods for countering the effects of drift. The first controls for the effects of room temperature, outside variations in temperature and the effects of activity prior to the subject entering the baseline phase by allowing the subject to sit in the experimental room, held at a constant temperature, for half an hour before starting the experiment. The second method is to use no-feedback intertrial intervals as discrete baselines within the feedback session. No-feedback intertrial intervals have however been shown to contaminate succeeding trial periods. Blankstein, Zimmerman and Egner (1976) proposed that this is because subjects are continuing to plan strategies for the next trial period rather than resting. Peper (1976) concluded that poor control is obtained during the trial periods because rapid switching from control to rest periods does not give subjects an opportunity to adequately test control strategies. Yates (1980) also suggested that a major disadvantage of employing intertrial intervals as a means of controlling for drift is that a reverse effect or upward drift can be demonstrated.

Sex differences in finger temperature values have also been observed; not only do females tend to have lower starting temperatures but they also drift downwards faster (Yates ,1980).

As well as the above methodological and physiological factors, Taub, Emurian and Howell (1974) claimed that 'person factors' are also influential in biofeedback. Taub et al (1974) stated that peripheral response control is facilitated by an informal, warm and friendly approach by the experimenter to the subject. However, no information was given about the type or nature of the feedback task, nor of the instructions administered. Given the absence of such information and the inability to replicate these findings, Lynch and Schuri (1978), the results should be treated as equivocal.

In conclusion, this selective review has considered some aspects of the methodology of biofeedback research. A pilot study specifically concerned with 'drift', its implications for baseline administration and physiological response interpretation is presented later in the study. It is however clear that a multitude of complex methodological issues make biofeedback a fruitful area of research.

The following is a review of empirical studies concerning the biofeedback control of peripheral blood flow. It is not a definitive analysis of all methodological issues in biofeedback, but is rather an attempt to evaluate the empirical findings within the context of some important methodological issues.

AUTHOR	INDEPENDENT VARIABLE	DESIGN	DEPENDENT VARIABLE	SUBJECT/SESSION	OUTCOME
Attfield and Peck (1979)	Migraine vs Normals and Relaxation vs Biofeedback	Control	Finger temp. Temporal artery amplitude. Forehead blood flow.	10 migraine 10 normals 6 treatment sessions	Finger temp. group significantly higher tempsthan relaxation . No relationship between temp change Forehead blood flow and superficial temp art amp.
Boudewyns (1976)	Experiment 1 Relaxation vs Electric Shock and Threat of Electric Shock	Single Group	Finger temp and self report of stress	21 normals, 10 minute 'relaxation' 7 min 'stress' phase with 4 electric shocks 15 minute 'relaxation'	Finger temp decreases with stress phase and increases with relaxation. Finger temp not related to other variables but related to self report measures.
	Experiment 2 Relaxation vs Electric Shock and Threat of Electric Shock	Single Group	Finger temp, self report for stress. Skin conductance, Pulse rate	21 normals 10 minute 'relaxation' 15 minute 'stress' phase	
Christie and Kotses (1973)	Instructions to increase vs decrease cephalic vaso-motor response	Group	Vasodilation and Vaso-constriction. Number of pulse beats above or below inter-trial criteria	8 normals 6 sessions Last only analysed. 'Avoidance of white noise' as reinforcement	Increase Group Compliance in all experimental periods. Decrease Group Complied in all but one period. No differences from baseline.
Crawford, Friesen and Tomlinson-Keasey (1977)	Anxiety provoking topics of conversation vs Pleasure conversation	Group	Finger temp.	40 subjects 4 sessions	Anxiety conversation - significant finger temp decrease in last 2 of 4 sessions. Pleasure conversation no temp change.
Fumoto (1977)	1. Pursuit tracking task 2. Adding digits 3. Uchida-Kraepelin psychodiag-nostic track-ing test	Group	Finger skin blood flow and pulse rate	8 subjects	Concentration on a task shows decrease in finger skin blood flow

AUTHOR	INDEPENDENT VARIABLE	DESIGN	DEPENDENT VARIABLE	SUBJECT/SESSION	OUTCOME
GARDNER and Keefe (1976)	Instructions to increase vs decrease temperature and information vs no information regarding temperature	Groups	Temperature of right index finger	40 subjects. 12 x 30 minute training sessions on 12 consecutive days. Tone & visual centre needle biofeedback	Changes in temp in specified direction. After 4 sessions - subjects who knew that information was related to finger temp showed superior performance. After 12 sessions, no difference.
Gillespie (1981)	Group A Finger temp feedback Group B Task relevant imagery and explicit verbal suggestion	Controlled	Finger temp	12 6 females. 6 males. 5 training sessions. Session 10 x 80 second trials 9 x 10 second intertrial intervals	Biofeedback Superior finger temp elevation than imagery and suggestion. Reliable differences in performance within four sessions
Herzfeld and Taub (1976)	Neutral vs thermal suggestions and instructions to increase and decrease finger temp	Group	Whole hand temp change. From five locations on the hand	5 normal subs. 3 to decrease 2 to increase 2 or 3 baseline days. 10 training days	Small but reliable differences in whole hand temp in 4 of 5 subjects. Greater temp changes on suggestion than non-suggestion days
Hunter, Russell, Russell and Zimmerman (1976)	Consistent vs mixed reinforcement and presence vs absence of learning Disorder	Group	Finger temp	60, 6-9 yr old children. 30 learning disabled 30 matched normals. Five 15 min sessions	Average increase of 0.38°F. Consistent reinforcement better than mixed. Learning disordered children better than normals.

AUTHOR	INDEPENDENT VARIABLE	DESIGN	DEPENDENT VARIABLE	SUBJECT/ SESSION	OUTCOME
Johnston (1977)	Verbal instructions vs visual analogue feedback. Left vs right hand. Instructions to increase vs decrease digital pulse amplitude	Group	Digital pulse amplitude	16 subjects 8 sessions	Control of pulse amplitude demonstrated with feedback, but did not add to control obtained with verbal instructions
Kappes and Michaud (1978)	Contingent vs non-contingent EMG feedback	Group	EMG. Hand temp. Anxiety. Locus of control.	12 subjects. Selected on high anxiety test scores. 10 sessions <u>Group 1</u> 5 contingent - 5 non-contingent EMG feedback <u>Group 2</u> Reverse of Group 1.	Non significant temp changes between contingent and non-contingent sessions.
Keefe (1975)	Instructions to increase vs decrease finger temp	Group	Forehead and finger differential temperature loop. Absolute finger temp.	8 subjects. Visual and auditory analogue feedback	Finger temperature changed as specified.
Keefe (1978)	<u>Group 1</u> Response specific instruction plus biofeedback <u>Group 2</u> Thermal suggestion plus feedback <u>Group 3</u> Instructions to rest plus feedback	Group	Finger temp	60 females. Five training sessions on five consecutive days. 2 follow up sessions, 1 and 2 weeks after	Biofeedback plus response specific instruction; (Group 1) or thermal suggestion; (Group 2) or thermal suggestion alone (Group 5) produced significant increases in finger temperature. Maintained at 1 and 2 weeks follow-up

AUTHOR	INDEPENDENT VARIABLE	DESIGN	DEPENDENT VARIABLE	SUBJECT/SESSION	OUTCOME
Keefe (1978) (cont)	Group 4 Response specific instructions without biofeedback Group 5 Thermal suggestions without feedback Group 6 Instructions to rest without feedback				
Keefe and Gardner (1979)	Instructions to increase vs decrease finger temperature	Group	Finger temp	Experiment 1 10 subjects 5 sessions Experiment 2 6 subjects 20 sessions	Experiment 1 Highly significant differences. 2.5°F increases 2.9°F decreases Experiment 2 Significant effect noted after three sessions. Further practice did not enhance effect
Keen and Montgomery (1978)	Interoceptive reinforcement (I.R.) vs elicited operant paradigm (E.O.P.) Dominant hand vs E.O.P., Non-dominant hand vs meter feedback vs no feedback control	Control group	Finger temp and pulse amplitude	29 college students 1 session	Greatest increase in temperature occurs in baseline period. EOP dominant hand > EOP non-dominant hand > I.R. meter feedback and no feedback control
King and Montgomery (1980)	Experiment 1 Instructions to increase finger temp (Group 1) vs Contingent feedback (Group II) vs non-contingent feedback (Group III) vs Contingent FB plus monetary incentive	Group	Finger temp	24 subjects 5 sessions	No difference in finger temp across all groups

AUTHOR	INDEPENDENT VARIABLE	DESIGN	DEPENDENT VARIABLE	SUBJECT/SESSION	OUTCOME
King and Montgomery (Cont)	Experiment II Instructions (I) vs Contingent (II) vs Non-contingent (III) vs Contingent feedback + somatic activity (IV) --- Autogenic phrases and thermal imagery suggests accompany every experimental condition, in both experiments	Group	Finger temp	32 subjects 5 sessions	Group IV, greater increase in finger temp.
Koppman, McDonald and Kunzel (1974)	Instructions to increase vs decrease blood volume pulse (BVP) of temporal artery	Single group	Cephalic vasomotor response (CVMR). EMG Zygomatic process. EMG Trapezius. Finger pulse volume. Chest respirometer.	9 migraineurs 6 ♂ 3 ♀ 2-4 weeks 2 to 3 sessions per week	Acquired control for dilation & constriction of superficial temporal artery amplitude. Only 'blood volume pulse' (BVP) modulated. Independent of other responses thus specific cerebral control
Lynch, Hama, Kohn and Miller (1976)	Experiment 1 Differential hand - hand temperature Experiment 2 Differential finger temp (same hand)	Group	Experiment 1 hand temp differences Experiment 2 Finger temp differences	4 children (9-11 years) 12 sessions	Experiment 1 Three out of four successful in complying Experiment 2 One out of four successful in complying --- Confound feedback and reward
Ohno, Tanaka, Takeya and Ikemi (1977)	Temp 'Increase' biofeedback group vs temp 'decrease' biofeedback group vs no feedback vs false feedback	Group	Digital temperature	40 subjects 3 sessions	Temp 'increase' significantly differs from temp 'decrease' group but not from baseline. --- Confound reward and reinforcement

AUTHOR	INDEPENDENT VARIABLE	DESIGN	DEPENDENT VARIABLE	SUBJECT/SESSION	OUTCOME
Price and Tursky (1976)	Binary analogue feedback vs Yolked (false) feedback vs Relaxation (autogenic phrases) vs neutral tape control and migraine vs normals	Control	Digital and extra-cranial blood volume	80 subjects 40 migraineurs 40 controls 1 session	No statistical differences in temperature change. High positive correlations between digital and extra-cranial blood flow. No incremental utility of biofeedback over simple relaxation in producing vasodilation.
Roberts, Schuler, Bacon, Zimmerman and Patterson (1975)	High vs Low Scorers on Harvard Group Scale of Hypnotic Susceptibility and Tellegen Absorption Scale	Group	Differential hand temp training	14 subjects, 7 in each group. 16 sessions Auditory feedback	Large and reliable performance and learning effects were unrelated to hypnotic susceptibility or capacity for absorbed imaginative attention
Sheridan, Zimmer, Finch and Eifler (1978)	Experiment 1 Autogenic phrases	Group	Experiment 1 finger temp Differential personality inventory (DPI) Scale of altered consciousness (SAC)	Experiment 1 40 subjects ½ given DPI, before ½ after autogenic tape	DPI fails to predict hand temperature
	Experiment 2 Autogenic phrases given 1 week after peripheral temp taken and DPI filled out	Group	Experiment 2 Finger temp D.P.I.	Experiment 2 47 subjects who scored high SAC	DPI fails to predict hand temperature
	Experiment 3	Group	Experiment 3 Finger temp. State trait anxiety inventory Alpert-Haber scale for facilitating, debilitating anxiety	Experiment 3 22 students who complained of 'high test anxiety'	No correlation between measured debilitating anxiety and temperature - but facilitative anxiety correlated with temp $r = +0.48$ ($p<0.05$)

AUTHOR	INDEPENDENT VARIABLE	DESIGN	DEPENDENT VARIABLE	SUBJECT/SESSION	OUTCOME
Simpson (1973)	Finger pulse volume feedback vs false feedback	Groups	Finger pulse volume. G.S.R. Heart rate. Respiration. Subjective rating of relaxation.	17 ♂ subjects 2 sessions Blue light (Visual binary feedback) Reinforces low arousal	FPV increased for subjects who knew that 'low arousal' was associated with feedback. Other measures failed to reveal differences
Simpson and Nelson (1976)	Finger pulse volume biofeedback and relaxation vs relaxation	Group	Finger pulse volume. Respiration rate. Skin conductance. Heart rate. Subjective rating of relaxation.	20 subjects 2 sessions	Finger pulse volume is significantly higher in the feedback group in the second session. Other measures failed to distinguish between the groups
Snyder and Noble (1968)	Digital vasoconstriction vs vasomotor stability vs no reinforcement	Control	Digital vaso-constriction	30 subjects 1 session	Experimental subjects reinforced for vaso-constriction - significant increase in number of constrictions
Steptoe, Mathews and Johnston (1974)	Differential ear lobe temperatures	Single group	Ear lobe temperature	8 subjects. 6 sessions in 3 weeks	0.3°C difference achieved
Stern and Pavloski (1974)	Experiment 1 Group I Reinforced for finger pulse vasoconstriction vs Group II Reinforcement yolked to above vs Group III Same number of reinforcement, but not for vasoconstriction	Group	Finger blood volume (pulse)	90 subjects. 30 per group, 1 session. Binary feedback (light) as reinforcement. Presented for 3 seconds only	Vasoconstrictions Group I > Group II > Group III
	Experiment 2 As above			45 normals	Group differences, highly significant

AUTHOR	INDEPENDENT VARIABLE	DESIGN	DEPENDENT VARIABLE	SUBJECT/SESSION	OUTCOME
Surwit (1977)	Simple vs complex feedback	Group	Digital temperature	15 subjects 2 sessions	(Simple: Analogue meter feedback Complex: Above, with binary feedback with lights and tones) Both groups - significant increase but complexity of feedback did not enhance learned temperature increases
Surwit, Shapiro and Feld (1976)	Increase vs decrease instructions	Groups	Digital temperature	16 subjects 7 sessions in 9-19 days	Temperature changes found primarily in decrease direction
Taub & Emurian (1976)	Increase vs decrease instructions	No specific design/s	Finger temperature	21 subjects. Not all groups had same number of subjects. White light intensity used as biofeedback. Session number and length varied	Downward temperatures more successfully achieved
Thompson and Russell (1976)	Feedback - instructions to increase vs yolked for financial reward vs relaxation group yolked for monetary reward	Group	Finger temperature. Financial incentive (based on performance)	35 subjects. 4 sessions counterbalanced audio,visual. No feedback	Biofeedback is necessary for learning to increase finger temperature
Wand, Slattery, Haskell and Taub (1978)	Temperature from web dorsum and four other places on hand	Group	Hand temperature	3 subjects. 21 training sessions	2 out of 3 subjects showed anatomical specificity but not until 16th session

AUTHOR	INDEPENDENT VARIABLE	DESIGN	DEPENDENT VARIABLE	SUBJECT/SESSION	OUTCOME
Willerman, Skeen and Simpson (1976)	Increase vs decrease and No task vs intellectual task	Group	Finger temperature	19 sessions Study 1 N = 10 Study 2 N = 9	Increases and decreases can be maintained without detectable decreases in problem solving efficiency

The role of relaxation in volitional finger temperature control studies

As Engstrom (1976) suggested that some degree of mental and physical passivity is important during the performance of biofeedback tasks, it is important to determine whether changes in finger temperature are a function of the feedback or a consequence of the passive state of the subject.

Thompson and Russell(1976) and Attfield and Peck (1979) have found that feedback of finger temperature with instructions to increase finger temperature, facilitated vasodilatation to a significantly greater extent than progressive relaxation alone. Price and Tursky (1976) found no incremental utility in the use of feedback over relaxation exercises in producing finger temperature increases. However, these findings may have been a function of the single session design employed by Price et al (1976) rather than the subjects responsiveness.

Ohno, Tanaka, Tayeka and Ikemi (1977) trained independent groups of subjects to raise and lower finger temperature. Although the increase and decrease groups differed significantly after training, neither succeeded in obtaining a significant change from its own baseline level. Two control groups (no feedback and false feedback) produced results which indicated that very little success had been achieved in this study. The subjects of this experiment were however uninformed about the nature of the task and thus no conclusive evidence about the effects of feedback upon finger temperature can be drawn.

Although these findings may question the sensitivity of finger temperature as an index of relaxation, a comparison of high and low arousal states provides a different impression of peripheral

blood flow sensitivity. Boudewyns (1976) monitored finger temperature during relaxation periods and periods during which electric shocks were threatened and administered. The electric shock or 'stress' periods, corresponded with a decrease in temperature whereas relaxation periods were characterised by an increase in finger temperature. Crawford, Friesen and Tomlinson-Keasey (1977) observed decreases in finger temperature in subjects allowed to discuss anxiety provoking topics, but no increases in peripheral temperature when pleasureable topics were discussed; rather there appeared to be a return to baseline levels. The three minute trial periods used in this study may not have provided sufficient time for the 'passive' vasodilatory response to develop fully, carry over effects and thermal lag masking potential vasodilatory responses. Butschek and Miller (1980) repeated this experiment incorporating a nine minute trial period (3 x 3 minute periods) with Anxiety and Pleasure topic groups. Both groups showed a decrease in finger temperature; however the anxiety topic groups displayed a significantly greater decrease, a result supporting that of Crawford et al (1977).

In conclusion it appears that 'stress' responses are associated with peripheral vasoconstriction and that relaxation may cause a slow return to baseline measures. Evidence also suggests that biofeedback induced finger temperature changes may be significantly greater than those associated with relaxation. These conclusions are equivocal as confounded designs and the lack of adequate controls confuse the issue.

<u>The role of information display in volitional finger temperature control studies</u>

Fundamental to biofeedback experiments is the presentation of

a physiological response or an analogue of that response, so that control can be acquired. Methods of presenting the information should therefore be investigated to determine their relative efficacy in enabling subjects to acquire feedback skills.

Thompson and Russell (1976) administered counter-balanced presentations of auditory, visual and no feedback to subjects instructed to raise their finger temperature. Financial reward and reinforcement are confounded in this design, financial reward being based on performance although rate of reward was yolked to no-feedback and relaxation training groups as controls. Although the type of feedback is not adequately described and the effects of financial reward upon feedback performance is yet to be determined (Hume 1977), the authors concluded that feedback is necessary and a training effect is evident within 4 x 15 minute periods.

Surwit (1977) examined 'complexity' of presentation and found that analogue meter feedback representing 'simple' feedback produced as effective results as 'complex' feedback using binary feedback with lights and tones. The single effect of any component was however not investigated.

The use of 'consistent' (contingent) and 'mixed' reinforcement for finger temperature elevation was studied by Hunter, Russell, Russell, and Zimmerman (1976). In this experiment 30 learning disabled 6-9 year old children were age matched with 30 normal children. Feedback consisted of a complex arrangement of moving toy trains and variable intensity lights. Hunter et al (1976) found that learning was demonstrated only for the contingently reinforced group. An incidental finding that the learning disabled children were superior in performance to the normal children

indicates grounds for further research.

Given that biofeedback equipment is relatively expensive, it would appear logical to investigate the simplest and most economical displays. This does not seem to be a consideration in the above experiments. Keen and Montgomery (1978) further complicate the issue in comparing Interoceptive reinforcement (IR) with Elicited operant paradigm (EOP) for dominant and non-dominant hands using meter feedback and a no-feedback control. IR is the presentation of an overall subtle sensation of warmth and a soft red light contingent on finger temperature elevation, the monitored hand shielded to prevent effects on the transducer. EOP is the emersion of the non-monitored hand in alternating cool and warm baths to capitalise upon vasomotor reflexes. Although EOP appeared to be most effective in eliciting temperature increases the greatest increases were found during the baseline period across all groups. The results from this experiment may have been influenced by a number of factors including drift effects. Laterality of vasomotor responses in the EOP condition may have caused a 'consensual reaction' (Lynch and Schuri , 1978) which would indicate that temperature changes were due to the external thermal conditions rather than internal operant control. Also the effects of changing ambient temperature; albeit minimal as in the IR condition, may also have led to a consensual reaction.

The role of instruction in volitional finger temperature control studies

The presentation of biofeedback is usually accompanied by instructions regarding demands of the task. Ohno, Tanaka, Tayeka and Ikemi (1977) provided no formal instruction with a temperature increase and a temperature decrease group; the presence or absence

of a light was the only form of information the groups received. A no-feedback and a false-feedback group were included as controls. The results indicated that temperature changes during the three sessions significantly differed between the increase and decrease groups but neither group differed from their own baseline. The results could indicate the redundancy of 'instructions', although very different results may have been observed had instructions been used. Also, considering that only three sessions were used and that binary visual feedback provides limited specific information about the amount of control achieved, the results are supportive of a biofeedback effect.

In the typical biofeedback experiment, increase and decrease instructions have been used as control conditions without using a control group instructed to maintain finger temperature stability. It is therefore often difficult to determine whether 'increase' and 'decrease' control has been established independently, or whether significant differences are due to one function changing more than the other. This question will be approached in more detail in the pilot study section.

Gardner and Keefe (1976) reported significant differences between groups instructed to 'increase' and then 'decrease' finger temperature. An 'information' and a 'no-information' condition regarding the meaning of the feedback information, was employed across both experimental conditions. After four sessions, subjects in the 'information' condition showed superior performances in both of the instructed directions. After twelve sessions, however, no difference in the ability to increase and decrease finger temperature was evident between the two information groups. It would therefore appear that

instructional set is not essential to the subjects overall performance and skill acquisition.

Surwit, Shapiro and Feld (1976) observed that temperature differences with opposing instructional 'sets' was primarily due to a change in the decrease direction. In contrast, Keefe (1975) compared increase and decrease instructions with subjects given differential finger-forehead temperature feedback; absolute finger temperature changes were also monitored. Visual and auditory analogue feedback were presented over 12 15-minute sessions. It was found that finger temperature changed in the specified direction and that differential finger temperature correlated highly with absolute finger temperature, validating earlier observations of Sargent, Green and Walters (1973b). Keefe and Gardner (1979) extended the above study which had used 12 sessions to investigate the effect of a larger number of experimental sessions. Results of the first part of the study indicated highly significant finger temperature differences after five sessions, the second part of the study employed 20 sessions and although a significant effect was noted after three sessions, the opportunity for further practice did not enhance the effect.

Instructions given to subjects in biofeedback experiments may include suggestions regarding mental strategies which might be employed to gain volitional control. King and Montgomery (1980) suggested that increases in finger temperature can only be obtained by skeletal mediation, independent of instructions. Comparing an autogenic instruction, a contingent feedback, a non-contingent feedback and a financial incentives group (without feedback), no differences in finger temperatures were noted. When, in a second experiment, the financial incentive group was replaced by another

contingent feedback group allowed to use a range of specific physical methods for elevating finger temperature, an increase was noted for this group.

Although acceptable somatic exercises included isometric tension and changes in respiration, no controls for the influence of limb movement were applied. King and Montgomery (1980) admit the absence of constraints to prevent subjects positioning their fingers towards their body, palms of the hands or other heat sources. As Lynch and Schuri (1978) have observed that isometric exercises of the shoulders and trunk and a variety of respiratory manoeuvres succeed in decreasing finger temperature, it may be that the finger temperature increases reported were achieved by uncontrolled strategies.

In conclusion, it appears that instructions are not critically important in the acquisition of finger temperature control providing feedback is available. It has also been suggested that the minimum number of feedback sessions required to achieve finger temperature control is three and that additional sessions do not increase the subject's ability to further increase finger temperature (Keefe and Gardner, 1979). King and Montgomery (1980) concluded that the role of somatic mediation was a more important factor in achieving finger temperature control than instructions, contingent, non-contingent feedback and financial incentives. However, lack of adequate controls render the evidence equivocal.

The role of suggestion in volitional finger temperature control studies

Instructions are a set of directives given by the experimenter to their subjects in an effort to orientate them towards the task characteristics in the feedback situation. Instructions may include

proscriptive and prescriptive statements regarding the acceptability of certain types of behaviour during the feedback session. Extending or supplementing instructions with proposals regarding cognitive strategies that may influence a subject's ability to effect desired changes may be construed as introducing the element of 'suggestion'. Suggestion need not always be verbal and explicit; financial incentives based upon performance, thermally related autogenic phrases and hypnosis may all have an effect upon performance of feedback tasks. Due to the often abstract and individual nature of suggestion, investigations into its effects are fraught with methodological difficulties. Largely as a consequence of these factors, the role of suggestion in biofeedback is seldom controlled for in experimental studies.

Herzfeld and Taub (1976) compared neutral and thermal suggestions for subjects instructed to increase and decrease whole hand temperature. Temperature changes were taken from five locations on the hand and it was found that four out of five subjects were able to produce small but reliable changes in whole hand temperature; the greatest changes occurring on days thermal suggestions consistent with instructed temperature changes were administered. More recently, Herzfeld and Taub (1977) found that a combination of externally imposed (as compared with internally generated) thermal suggestions and biofeedback is more successful in eliciting temperature changes than biofeedback alone. Again temperature elevation and depression instructions were used as comparisons. Keefe (1978) compared the effects of thermal suggestion, response-specific instruction and instructions to rest; with and without finger temperature biofeedback. Although response-specific instructions plus biofeedback and thermal suggestions plus

biofeedback produced significant increases in finger temperature, thermal suggestions without biofeedback also produced significant finger temperature increases. These effects were maintained over a two week follow-up period, indicating that the subjects who received thermal suggestions were able to increase finger temperature without the aid of feedback. This was not the case with subjects who received response-specific instructions but were not provided with feedback. These findings have not however been replicated; Gillespie (1981) found that thermal suggestions presented visually and verbally, relevant to increasing finger temperature, were not as effective as feedback alone.

In conclusion it would seem that the role of suggestion in finger temperature control is of considerable interest. Quantitative evidence implies that the combined effects of thermal suggestion and feedback are superior to the effects of feedback alone. However, the lack of conclusive evidence may be a result of the poorly designed experiments that characterise the area, hence it would seem a vital area for further research.

The role of imagery in volitional finger temperature control studies

Richardson (1969) stated that two types of imagery have been used in biofeedback, memory imagery (self induced images of warm and cold conditions) and imagination imagery which is a term covering a wide variety of cognitive strategies. Imagination imagery is distinguished from memory imagery mainly by its lack of personal experiential reference, by its being more vivid than memory imagery and by its tendency to occur in unusual states of consciousness. The importance of imagination imagery lies in its potential link with

the subjective states alleged to be associated with some kinds of biofeedback training and the possibility that these states may act, via imagination imagery, as links with the somatic changes which biofeedback produced.

Maslach, Marshall and Zimbardo (1972) reported that induced hypnotic imagery was capable of creating controlled peripheral skin temperature changes. Although the results show substantial bi-directional changes between two hands in experimental compared to control subjects, there was no information regarding ambient temperature or method of temperature recording. Most importantly, it is not clear whether the temperature changes were absolute or differential changes due to decreases in one hand relative to the other; a phenomenon reported by Surwit, Shapiro and Feld (1976). Clark and Forgione (1974) found that gingival and digital responses to thermal images did not correspond in any systematic way, and that 'cold' and 'warm' memory image instructions produced the same digital vascular responses; initial constriction followed by progressive dilatation. Blizard, Cowings and Miller (1975) taught subjects to imagine heaviness and warmth or coolness and lightness in their hands. Significant increases in heart rate and respiration rate accompanied the induction of coolness and lightness but the reverse was not the case for the induction of heaviness and warmth and no differences in finger temperature resulted from differential induction of the two states. Dugan and Sheridan (1976) found that subjects instructed to imagine their hands in very warm water or in ice cold water (but with no feedback) produced significant increases and decreases in peripheral finger temperature respectively. All ten subjects using cooling imagery showed a significant change in one hand while six showed

corresponding changes in the other hand as well. These results were however well within the range of temperature changes that would be expected for drift effects (Yates , 1980).

It has been suggested that peripheral blood flow changes and the ability to influence and control physiological responses may not only vary with levels of arousal (Boudewyns , 1976 ; Crawford, Friesen and Tomlinson Keasey , 1977 and Butschek and Miller , 1980), but also as a function of personality variables. Roberts, Schuler, Bacon, Zimmerman and Patterson (1975) investigated the ability to produce differential hand temperature changes with high and low scoring subjects on 'hypnotic susceptibility' and 'absorbed imaginative attention' scales. Large and reliable performance and learning effects were found to be unrelated to hypnotic susceptibility or capacity for absorbed imaginative attention. As no instruction as to the use of imagery was given, the results should be treated as equivocal.

In conclusion, the relative significance of feedback and imagery instructions has been poorly researched. The results render any comparative evaluation regarding the importance of imagery and bio-feedback as undetermined.

D. Pulse amplitude response studies

Compared with finger temperature feedback, there is a dearth of information about the conditionability of vasomotor pulse activity. Vasomotor activity in the skin is under sympathetic control, the vessels are therefore capable of active vasoconstriction but passive vasodilatation (Cook , 1974). The mechanism underlying vascular pulse conditioning is similar to that underlying finger temperature feedback; in that vasolability of peripheral pulsations is related to the arousal level of the sympathetic nervous system.

Snyder and Noble (1968) reinforced subjects for decreasing finger pulse volume, using binary-visual feedback. A group of subjects reinforced for maintaining vasomotor stability, also given binary-visual feedback, and a no-reinforcement group, were used as controls. A significant increase in the number of vasoconstrictions during the acquisition phase of the experiment was observed for the contingently reinforced group. Stern and Pavloski (1974) replicated the Snyder and Noble (1968) study using 30 experimental subjects and 60 yolked controls, half of whom received reinforcement identical in time and number to the experimental group (true yolking) and half who received the same number of reinforcements per minute but never for vaso-constricting (partial yolking). Highly significant group differences between the contingently reinforced, true and partially yolked groups were found. Hence it would appear that there is evidence to suggest that biofeedback conditioning of digital vasoconstriction is possible. Simpson (1973) and Simpson and Nelson (1976) found that vasodilatation of the digital pulse could be elicited to an extent greater than that observed for false feedback or for progressive relaxation groups.

These findings appear consistent with the observations recorded during finger temperature feedback experiments although Yates (1980) stated that the results of Simpson (1973) and Simpson and Nelson (1976) were influenced by drift effects.

Increasing interest has developed in the biofeedback control of cephalic vasomotor responses, the reason being that direct modification is obtained at the alleged site of the primary 'pain mechanism' responsible for migraine headaches (Wolff , 1963). Two studies of particular interest were conducted by Christie and Kotses (1973) and Koppman, McDonald and Kunzel (1974). Christie and Kotses (1973) gave instructions to dilate or constrict cephalic vasomotor responses using red and green lights to signal the onset of the dilatation and constriction periods respectively. Success was reinforced by the avoidance of aversive white noise. A similar 'white noise avoidance' paradigm was used by Koppman, McDonald and Kunzel (1974) both experiments using number of pulse beats above or below inter-trial criteria and not an initial baseline criterion as a measure of success. Christie and Kotses (1973) found that although there were significant differences in pulse amplitude between the trial and inter-trial periods, differences were not significantly different from baseline recordings. Koppman, McDonald and Kunzel (1974) analysed time on target as the index of activity without including measures of amplitude change. Given these methodological and statistical shortcomings, Koppman, McDonald and Kunzel (1974) stated that it is premature to conclude 'that acquired control of temporal artery constriction and dilatation is demonstrated independent of other responses and that the control is a specifically cerebral phenomenon'.

E. Summary : Volitional peripheral temperature control, temporal artery amplitude control and digital pulse amplitude control.

Experimental evidence regarding volitional control of finger temperature, temporal artery amplitude and digital pulse control has been found deficient on a number of methodological, procedural and physiological grounds.

Major criticisms include:

a. The interpretation of physiological responses in terms of volitional control whilst neglecting other possible influences (i.e. 'Drift').

b. The inclusion of multiple independent variable designs such as 'consistent versus mixed reinforcement (contingent versus non-contingent) for finger temperature elevation with learning disordered versus non-learning disordered child subjects' (Hunter, Russell, Russell and Zimmerman , 1976) . 'Verbal instructions versus analogue feedback for increases and decreases in digital pulse amplitude in dominant versus non-dominant hands' (Johnston ,1977). 'Binary analogue feedback for increases in finger temperature versus yolked false feedback versus relaxation with autogenic phrases versus a neutral information condition with migraine and non-migraine patients (Price and Tursky , 1976).

c. Few studies have controlled for the effects of feedback by using a no-feedback group or a feedback group instructed to maintain baseline levels of responding.

d. There is a tendency to draw conclusions about the subject's ability to control physiological responses based on the use of widely differing displays, combinations of displays and the presentation of feedback from multiple sites.

e. Prior to the administration of feedback, a stabilisation period of at least half an hour has been suggested (Yates, 1980). In general, researchers have neglected to include details of stabilisation periods.

f. Changes in temporal artery amplitude measures were obtained using 'time on target' scores, where feedback was contingent upon a proportion of the temporal artery pulsations occurring below a specific amplitude criterion. The criterion was adjusted at inter-trial intervals and although inter-trial intervals overcome the exaggerated effects of downward drift, a reverse (upward) drift effect appears to occur (Yates, 1980). Given that amplitude measures were not obtained, it is difficult to assess the exact nature of the changes.

g. A variety of experimental feedback periods have been used in different studies. Herzfeld and Taub (1976) used 50 second trials and 10 second inter-trial no-feedback intervals. Surwit (1977) and Surwit, Shapiro and Feld (1976) used 75 second trials and 10 second inter-trial no-feedback intervals. Given that thermal changes may be slow (due to thermal lag and to sympathetic nervous system dynamics) it is difficult to determine how far conclusions from these results can be generalised.

As most of the studies display the above deficits as single or multiple features in their methodology, the question of whether volitional control can be achieved remains equivocal although evidence strongly suggests that it is possible. Steptoe, Mathews and Johnston (1974) investigated voluntary control of differential temperature changes in the ear lobes. They chose the ear lobes, primarily because of the absence of active skeletal musculature and the specificity of

the response which could be obtained. Although the task proved difficult for the subjects, results showed temperature change in the instructed direction, which would be consistent with an ability to control peripheral vascular responses. The striking feature was the specificity of control given the unusual feedback location. Whether this result is relevant to finger temperature or temporal artery amplitude changes where different types of vessels are involved, still remains to be elucidated.

SECTION 2

CONTENTS

CLINICAL STUDIES

Clinical studies of the treatment of migraine headaches have been assessed in the following terms:

1. Independent variables : treatment procedures.
2. Design ; to be discussed below.
3. Dependent variables : measures of treatment effects.
4. Subjects : number and diagnosis.
5. Diagnostic criteria : for patient selection.
6. Treatment : frequency, duration and type of feedback given.
7. Concurrent Treatments : other than dependent variables.
8. Bioelectric responses : reported physiological findings.
9. Follow-up : duration and procedure used to obtain information.
10. Clinical response.

DESIGN

The demonstration of treatment specific effects depends on sound research design. The research designs in the studies reviewed are placed in four general classes. The classes are based on the degree to which each design can isolate treatment specific effects, and on the practice of other reviewers (Jessup, Neufeld and Merskey, 1979).

Anecdotal case study

This type of report usually describes a patient, the treatment procedure and the apparent clinical benefit. It lacks control over the many non-treatment variables that can account for patient improvement.

Systematic case study

In this design a target response is repeatedly and systematically

measured both during a pretreatment baseline (A) and during treatment (B) (A-B design). Support for treatment effectiveness increases if the baseline is long and improvement occurs when the treatment is introduced. A stronger version of the systematic case study is where the treatment is followed by a second no treatment baseline (A-B-A design). The treatment effectiveness gains support if the target symptom changes during the treatment phase and reverts during the second baseline. The major problem with the A-B-A design in applied clinical work is the difficulty in concluding an experiment with a baseline phase if some improvement had occurred during the treatment phase. This concern has led to the greater use of the A-B-A-B design where the treatment is reintroduced after having once been withdrawn (Barlow, Blanchard, Hayes, and Epstein ,1977). However, the generalisability of systematic case study results to other subjects and to other settings sometimes appears questionable. Carry over effects and appropriate statistical analyses are also issues.

Group outcome study

The target symptom is measured pre and post treatment in a group of similar patients. Often two or more groups receiving different treatments are included in the same group outcome study. However any report classed as a group outcome study will always lack a true control group who receive the non specific aspects of treatment such as attention, suggestion and assessment. Multiple group outcome studies are comparisons of different treatments; consequently group outcome studies cannot isolate treatment specific effects. Additional rigour may be gained by taking extended baseline measures, thus making the group outcome design similar to a set of systematic case studies.

Control group study

This design permits the clearest isolation of treatment specific effects. A control group study requires at least two groups of comparable patients, a treatment group and a no-treatment group who are assessed at the same time. This procedure controls for the passage of time and for the variety of non-treatment effects that have come to be labelled 'spontaneous remission'. The inclusion of an attention placebo group separates the effects of attention and expectancy from the treatment of interest, and is hence the most powerful design.

AUTHOR	INDEPENDENT VARIABLE	DESIGN	DEPENDENT VARIABLE	SUBJECTS	DIAGNOSTIC CRITERIA	TREATMENT	CONCURRENT TREATMENT	BIOELECTRIC RESPONSES	FOLLOW-UP METHOD, DURATION	CLINICAL RESPONSE
Adler and Adler (1976)	Interpretive psychotherapy + EMG + temp feedback with passive concen'n vs all minus temp feedback	Group, (BF + others vs others)	Frequency Drugs	58. 22 migraine 19 tension 12 mixed 5 cluster	-	5 x 1 hr sessions. 2 per week	Medication (unspecified)	-	1-5 years (Method unspecified)	Tension = 88% success Migraine = 81% success Mixed = 60% success Cluster = 60% success
Alvin (1974)	Hand skin temp feedback (Preferred vs non-preferred hand)	Group	'Impression' of improvement	10 migraine 10 non migraine	-	6 sessions	-	-	-	$\frac{2}{3}$ migraine subjects improve. Suggesting that increase in hand temp induces headache improvement
Anderson, Basker and Dalton (1975)	Hypnotherapy vs Stemetil (Prochlorperazine)	Group	1. No of attacks 2. Severity: Grade 4 or 0	47, 23 hypnotherapy 24 chemotherapy	Whitty and Hockaday (1968)	Hypnotherapy 6 sessions Stemetil 20 mgm daily for 1 month Thereafter 10 mgm daily for 11 mths	Ergotamine when necessary for Stemetil group	-	1 year continuous care	Hypnotherapy better than chemotherapy

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Andreychuk and Skriver (1975)	Handwarming FB vs Alpha EEG FB vs self hypnosis. High vs low hypnotisability (HIP)	Group	Self monitored headache index. (Durn' x INT = (HI))	33 migraine	-	10 x 45 min sessions over 10 weeks	-	-	No follow-up except for final ½ of 10 wk treatment period	Handwarming = 79% improvement EEG = 43% improvement Hypnosis = 36% improvement
Ansell (1977)	Hypnotherapy (Handwarming and head-cooling by suggestion)	Anecdotal	-	1 ♀	Description	-	-	-	-	Improvement noted - Handwarming initially induced by handwhirling, then hypnotherapy used to induce same
Beasley (1976)	Group I Autogenic phrases + relaxation + EMG & skin temp F.B. vs Group II Skin temp FB vs Group III Relaxation + autogenic phrases vs Group IV No treatment	Control	1.Intensity 2.Frequency 3.EMG 4.Digital temp	37 ♀	-	10 x ½ hr sessions	-	-	-	Improvement GpI > GpII (no sig. diffs) Both > GpIII and GpIV. No improvement for Gps III and IV.

AUTHOR	INDEPENDENT VARIABLE	DESIGN	DEPENDENT VARIABLE	SUBJECTS	DIAGNOSTIC CRITERIA	TREATMENT	CONCURRENT TREATMENT	BIOELECTRIC RESPONSES	FOLLOW-UP METHOD, DURATION	CLINICAL RESPONSE
Benson, Klemchuk and Graham (1974)	Transcendental meditation	Group	1.Severity on 3 point scale 2.Medication	17 migraine 4 cluster	-	4 consecutive daily sessions	Medication: 1.Ergotamine and methy-sergide 2.Analgesics 3.Narcotics	-	Baseline 3 months. Follow-up 4-14 mths. Method un-specified	6 out of 17 migraines improved. No pattern for medication, but decrease over time. Cluster patients response variable.
Bild (1976)	CVMR feedback vs EMG FB vs waiting list control	Control	1.Intensity 2.Medication 3.Frequency 4.Duration	19	-	12 x 30 min sessions	Under dep. var.	Reduction in CVMR. Reduction in EMG	Baseline 45 days Follow-up 45 days	Improvement CVMR > EMG > Waiting list. (No difference in intensity changes)
Blanchard, Theobald, Williamson, Silver and Brown (1978)	Finger temp FB + autogenic phrases vs progressive relaxation vs waiting list	Control	Intensity frequency duration medication	30 migraine	Checklist	12 sessions over 6 weeks	-	-	-	Finger temp + auto phrases and prog rel'n improve over baseline. Relaxation superior at end of treat-ment but not after 1 month
Crosson, Andreychuk, Tiemann and Phillips (1978)	Finger temp FB + autogenic phrases vs hypnosis bio-feedback (Hypnosis, temp FB & autogenic phrases)	Group	1.Intensity 2.Duration	10	-	9 sessions within a 3-4 week period	-	Temperatures included	Baseline & follow up not included	Relationship between temperature changes and headache activity not clear

AUTHOR	INDEPENDENT VARIABLE	DESIGN	DEPENDENT VARIABLE	SUBJECTS	DIAGNOSTIC CRITERIA	TREATMENT	CONCURRENT TREATMENT	BIOELECTRIC RESPONSES	FOLLOW-UP METHOD, DURATION	CLINICAL RESPONSE
Daniels (1976)	Hypnotic induction to warm hands and cool head	Anecdotall case studies	1.Frequency 2.Intensity 3.Duration	3	Descriptions	6 x 20 min sessions	-	-	Baseline: Retrospect-ive. Follow up: 9 mths after treatment. Retrospect-ive.	Subjects expressed satisfaction. Frequency, intensity and duration decreased during experiment, but recurred afterwards.
Daniels (1977)	Deep muscle rel'n. Cue controlled rel'n. Hypnotic hand warm. Cognitive behaviour therapy and 'thought stopping'	Anecdotal unsystem-atic case study	Anxiety symptoms and headaches	1	-	3 sessions	Diazepam	-	1 year	Less symptoms. Reduced valium.
Diamond and Franklin (1976)	Finger temp FB + EMG FB + relaxation + autogenic phrases	Group	Response rated as good, fair, or non	32 children 10-18 years old	$\frac{2}{5}$ criteria	-	-	-	-	$^{26}/_{32}$ good $^{3}/_{32}$ fair $^{2}/_{32}$ non $^{1}/_{32}$ lost in follow up

AUTHOR	INDEPENDENT VARIABLE	DESIGN	DEPENDENT VARIABLE	SUBJECTS	DIAGNOSTIC CRITERIA	TREATMENT	CONCURRENT TREATMENT	BIOELECTRIC RESPONSES	FOLLOW-UP METHOD, DURATION	CLINICAL RESPONSE
Diamond, Diamond-Falk and De Veno (1978)	Skin temp FB + Autogenic phrases + muscle relaxation + EMG FB	Single group	Intensity, general improvement	407 questionnaires returned from 556 sent out	-	Local Patients 8 sessions in 4 wks and 2 daily sessions at home for 4 weeks Out of Town Patients 2 sessions per day for 2 wks and 2 daily sessions at home for 4 weeks	-	-	5 year retrospective study. Questionnaires posted.	Biofeedback (undifferentiated) best for young non drug habituated females.
Feuerstein and Adams (1977)	CVMR + EMG FB	4 systematic case studies	Frequency. Duration. Intensity. Blood pressure. Resp'n Rate. Blood pulse volume (BVP). Blood volume (BV)	4. 2 migraines 2 tension	Screening procedure - no specific definition	6 EMG + CVMR Counter-balanced. Auditory FB	Aspirin Analgesic Valium	No objective data. Time on target analysed	Baseline - 4 weeks Follow-up- 9 weeks	'Feedback activity effective when directed at pain mechanism'. Frequency decrease by 72% Duration decrease by 73% Intensity increase by 34%

AUTHOR	INDEPENDENT VARIABLE	DESIGN	DEPENDENT VARIABLE	SUBJECTS	DIAGNOSTIC CRITERIA	TREATMENT	CONCURRENT TREATMENT	BIOELECTRIC RESPONSES	FOLLOW-UP METHOD, DURATION	CLINICAL RESPONSE
Feuerstein Adams and Beiman (1976)	CVMR + EMG	Systematic case study (ABACA)	Frequency, duration, EMG, CVMR. (vasospasms) BVP,	1 ♀ mixed headache	-	Tone off for BVP/ vasospasm decrease. 6 session EMG in 6 weeks (B) 6 session CVMR in 7 weeks (C)	Librium, Compazine, Tranxene, Demoral as needed, Ergomar,	No change in CVMR during EMG FB. No objective data.	Baseline A = 5 wks Follow-up A = 8 wks	No change in CVMR associated with EMG - but decrease in headache activity. CVMR reduction related to decrease in headaches. Reduction on Tranxene only.
Friar and Beatty (1976)	CVMR vs finger pulse volume	Group	Frequency Duration Location Intensity Medication	19	questionnaire rated for certainty of diagnosis. Response to Ergotamine used as positive diagnostic indicator	8 sessions in 3 wks, Auditory FB	Mild analgesics, Vasocon-strictors, Sedatives,	CVMR and finger pulse volume decrease, CVMR from forehead,	Baseline 30 day Follow-up 30 day	CVMR decrease = decrease in incidence in major attacks. No differences between groups in intensity

AUTHOR	INDEPENDENT VARIABL E	DESIGN	DEPENDENT VARIABLE	SUBJECTS	DIAGNOSTIC CRITERIA	TREATMENT	CONCURRENT TREATMENT	BIOELECTRIC RESPONSES	FOLLOW-UP METHOD, DURATION	CLINICAL RESPONSE
Gainer (1978)	Assertion training and systematic desensitisation and finger temp FB and finger temp descrimination training	Anecdotal case study	Frequency, Duration, Medication, Intensity, Disability,	1	No neurological signs - descriptive	Assertive training & systematic des completed 1976. (Wright 1976) Fing temp FB + fing temp descrim'n training follows 6 wks later	-	-	Baseline - 4 weeks Follow-up 8 weeks	Assertive Training and Systematic Desensitisation - no improvement. Finger Temp FB - no prodromata .: training ineffectual. Temp Desc Training 79% improvement. Headaches ceased after 4 weeks (2 descrimination sessions)
Graham (1974)	Finger temp FB vs hypnosis vs finger temp and hypnosis	Group	Frequency, Duration, Intensity, Premedication,	30 (screened for hypnotic susceptibility)	-	To increase finger temp by 2°F in 1 minute	-	Feedback alone better at reaching criteria	1 month follow-up	All groups - decrease in frequency, intensity, medication. All groups mediation decrease at follow-up.
Hay and Madders (1971)	Muscle relaxation	Group	Frequency, Intensity, Duration, Subjective estimation of progress,	98 initially and _ 20 new candidates annually for 6 years	GP referral No diagnostic criteria	6 sessions in 6 wks 1.Rationale 2.Exercises 3.Group discussion	-	-	Baseline - unspec'd. Follow-up 1 year. Questionnaires at beginning and end of treatment	69 patients - decrease in frequency, severity or duration. 29 patients - no change.

AUTHOR	INDEPENDENT VARIABLE	DESIGN	DEPENDENT VARIABLE	SUBJECTS	DIAGNOSTIC CRITERIA	TREATMENT	CONCURRENT TREATMENT	BIOELECTRIC RESPONSES	FOLLOW-UP METHOD, DURATION	CLINICAL RESPONSE
Johnson and Turin (1975)	Finger temp (cooling and warming)	Systematic case study	Number of headaches. Pills. Duration of headaches	1	-	24 sessions	Unspecified	Graphs in °F.	Baseline 5 weeks	All 3 dependent variables worsened over baseline and cooling. Improved with warming Finger temperature.
Koppman, McDonald and Kunzel (1974)	Temporal art amp. Blood volume pulse (BVP)	Group	-	9	Carnick patient headache checklist	12 sessions Auditory feedback	-	Amplitude not measured	-	Specific vasomotor control. No correlation between finger and cerebral blood flow variations.
Kentsmith Strider, Copenhaver and Jacques (1976)	Concentrative meditation and finger temp FB and relaxation training	Anecdotal case study	Intensity Duration Medication DBH levels	1	Descriptive. 2/3 criteria. Responseto Ergotamine	-	Ergotamine discontinued after 2 weeks	Blood analysis for plasma DBH. Increase in finger temp	Baseline = 30 days Follow-up = 9 months (Total time 10 months)	Cessation of headaches.
Lambley (1978)	Anti-reductionist approach	Anecdotal case study	Frequency. Duration. Subjective emotions.	3	Anecdotal	-	-	-	2 discontinued 1 follow-up 18 months	Felt happier with relationships

AUTHOR	INDEPENDENT VARIABLE	DESIGN	DEPENDENT VARIABLE	SUBJECTS	DIAGNOSTIC CRITERIA	TREATMENT	CONCURRENT TREATMENT	BIOELECTRIC RESPONSES	FOLLOW-UP METHOD, DURATION	CLINICAL RESPONSE
Lutker (1971)	'Muscle relaxation' & 'relaxing at feelings of pressure build up' & 'think relaxed'	Anecdotal case study	General headache improvement	1	Build up of pressure. (No migraine symptoms.)	-	Medication stopped after 2nd day of 'relaxing at feelings of pressure build up'	-	Follow-up 2 months	Complete alleviation
Medina, Diamond and Franklin (1976)	Self relaxation and EMG FB and autogenic phrases & finger temp FB	Group	Intensity Frequency Medication	27 13 migraine 14 tension	-	EMG (auditory FB) then finger temp training	-	Increase of $2^{o}F$ in first minute	Baseline 11 months Follow-up 15 months	9 migraine patients improved. 4 tension patients improved.
Mitch, McGrady and Iannone (1976)	Autogenic feedback training & finger temp FB	Group	Frequency Intensity Duration Medication	20 'uncomplicated' migraines. Mixed headache, tension headache	-	Treatment over 12 weeks	-	-	Baseline 6 months Follow-up for 10 patients 6 months	Uncomplicated migraines were successfully treated, 65% pts improved on 2 or more measures. 35% pts improved on 1. In presence of tension - EMG and counselling necessary

AUTHOR	INDEPENDENT VARIABLE	DESIGN	DEPENDENT VARIABLE	SUBJECTS	DIAGNOSTIC CRITERIA	TREATMENT	CONCURRENT TREATMENT	BIOELECTRIC RESPONSES	FOLLOW-UP METHOD, DURATION	CLINICAL RESPONSE
Mitchell and Mitchell (1971)	Study 1 Relaxation vs Combined desensitisation vs no treatment	Control	Anxiety levels and minor targets. Intensity of headaches,	17	-	Lecture	-	-	Baseline 8 weeks Follow-up 32 weeks	No change for relaxation patients. No change in anxiety levels. Significant improvements in attitude to minor targets.
	Study 2 No treatment vs desensitisation vs combined des't (after pharmacotherapy) vs combined des't (no previous pharmacoth'y)	Control	Frequency of headaches, Duration of headaches, Severity of headaches, Minor targets	20	-	15 sessions	-	-	-	Both 'combined' desensitisation groups better than single model or no treatment control. Minor target improvements.
Montgomery and Ehrisman (1976)	EEG and alpha feedback and relaxation	Group	Frequency Severity Duration	22	-	-	-	-	Follow-up 6 months to 3 years. Questionnaire. Assessed at end of baseline. End of training. End of follow-up	13 of 22 questionnaires returned (50%). Significant Reduction in symptoms. Some recurred between end of treatment and follow-up.

AUTHOR	INDEPENDENT VARIABLE	DESIGN	DEPENDENT VARIABLE	SUBJECTS	DIAGNOSTIC CRITERIA	TREATMENT	CONCURRENT TREATMENT	BIOELECTRIC RESPONSES	FOLLOW-UP METHOD, DURATION	CLINICAL RESPONSE
Mitchell and White (1976)	Self recording and self monitoring and Stage 1 Problem analysis Goal setting Behavioural management Stage 2 13 self change skills	Systematic case study	Discomfort	1	Not specified (descriptive)	Self recording 8 weeks Self monitoring 8 weeks Stage 1 - 8 weeks Stage 2 - 6 weeks	-	-	Baseline - Retrospective. Follow-up Monthly 8 months	Self recording & monitoring = no improvement. Stage 1 52% improvement Stage 2 72% " Over 8 months 100% improvement
Mullinix, Norton, Hack and Fishman (1978)	Finger temp feedback True vs false feedback	Control	Intensity Medication	11	Specified - minimum of 2 out of 5 symptoms	Auditory feedback 6 sessions in 3 weeks	-	True feedback - Greater temp increase than false	Baseline 8 weeks Follow-up 3 weeks	All patients used similar or smaller amounts of medication than pre-treatment.
Paulley and Haskell (1975)	Group technique and individual therapy	Group	Improvement 1. Non 2. Slight 3. Major 4. Syndrome shift	800+	Description of personality traits	6-8 weekly ½ hr session with physiotherapist for relaxation and understanding the 'migraine personality'	-	-	Follow-up 66 patients for 1-8 yrs	2 out of 3 patients achieve major reduction

AUTHOR	INDEPENDENT VARIABLE	DESIGN	DEPENDENT VARIABLE	SUBJECTS	DIAGNOSTIC CRITERIA	TREATMENT	CONCURRENT TREATMENT	BIOELECTRIC RESPONSES	FOLLOW-UP METHOD, DURATION	CLINICAL RESPONSE
Peck (1980)	Finger temp vs EMG feedback	Group	Frequency Intensity Duration	8	Waters Question-naire $^2/_3$ criteria	6 weekly sessions Fing temp FB / EMG. (Fing temp-visual FB EMG - aud FB.) 3 or 4 wks betw'n cross over	-	Temperature - no increase. EMG - decrease less than 1v	Follow-up- 1st 24 wks 2nd 73 wks	1st follow-up (day to day records). No change in intensity and duration. Frequency decreases for both groups. 2nd Follow-up. Revertion to baseline for both groups.
Reading and Mohr (1976)	Finger temp feedback	Group	Duration. Frequency. Intensity on 3 point scale	6	-	Visual analogue feedback	-	Rise of 0.1°C is criteria of success	Baseline- 4-6 weeks Follow-up 2 months	Decrease frequency Decrease duration Decrease intensity (3 point scale)
Sargent, Green, Walters (1972)	Differential (hand-head) temp train-ing and auto-genic phrases	Group	Intensity. Type and potency of medication	75. 63 migraine 10 tension 2 cluster (records for 33)	-	Weekly until skill mastered	Medication. Psycho-therapy	-	-	Of 33 patients, 32 were migraine. Rater 1 = 29 improve Rater 2 = 26 improve Rater 3 = 22 improve

AUTHOR	INDEPENDENT VARIABLE	DESIGN	DEPENDENT VARIABLE	SUBJECTS	DIAGNOSTIC CRITERIA	TREATMENT	CONCURRENT TREATMENT	BIOELECTRIC RESPONSES	FOLLOW-UP METHOD, DURATION	CLINICAL RESPONSE
Sargent, Green, Walters (1973)	Differential (hand-head) temp training and autogenic phrases	Group	Frequency, Severity, Associated symptoms. Duration, Degree of disability,	75. 57 migraine 11 tension 5 combined 2 cluster	-	Weekly until skill mastered	-	Not sure whether hand or head changes in temp	Baseline 1 month Follow-up every 1-3 months. Referred to clinic over 1-3 yrs	After 180 days 81% helped.
Sargent, Green, Walters (1973)	Differential (hand-head) temp training and autogenic phrases	Group	Severity, Potency & number of analgesics	28. 20 migraine 6 tension 2 'undecided'	Friedman, Finley, Graham, Kunkle, Ostfield & Wolff (1962)	Weekly or bi-weekly sessions until skill mastered	Medication	-	Follow-up 1-22 months	Of 15 migraines evaluated, 12 improved 3 unimproved. Of 4 tension evaluated 2 improved 3 unimproved.
Solbach and Sargent (1977)	Handwarming and autogenic phrases	Group	Headache records, Medication, Frequency, Intensity, Duration, Demographic & personal variables	110.	-	3 weeks to 3 months Weekly/ Bi-weekly sessions	Medication	-	270 days of training & follow-up. Questionnaires sent after 270 days. 38 returned. 18 follow-up by telephone	36(33%) drop out. 74 complete (graduate) 270 days. Graduates show greater decrease in intensity/duration/ medication.

AUTHOR	INDEPENDENT VARIABLE	DESIGN	DEPENDENT VARIABLE	SUBJECTS	DIAGNOSTIC CRITERIA	TREATMENT	CONCURRENT TREATMENT	BIOELECTRIC RESPONSES	FOLLOW-UP METHOD, DURATION	CLINICAL RESPONSE
Stambaugh and House (1977)	EMG (BF) and relaxation and autogenic phrases and hypnotic approaches	Systematic case study (ABACADAE etc) 23 phases	Frequency Intensity Duration Medication	1	Description of headache precipitated by head trauma	Relaxation + hetero-hypnosis + autogenic heat transfer + auto-hypnosis and others (23 phases)	Analgesics	-	Baseline 22 weeks Follow-up 1 month & 8 months	Verbal report of minimal headaches. Decrease in analgesics. Stresses importance of auto-hypnosis
Sturgis, Tollison and Adams (1978)	CVMR with EMG	Group (counter-balanced)	Type. Frequency. Intensity. Degree of disability. Duration. Medication.	Migraine and muscle control 2	-	15 CVMR + 15 EMG sessions Counter-balanced across subjects. CVMR - 8 wks. EMG - 12 wks.	-	% - relative changes	Baseline 7 weeks. BVP 8 weeks. EMG 12 weeks. Follow-up 16 weeks. Headache forms collected weekly	CVMR improves migraine headaches. EMG improves tension headaches.
Turin and Johnson (1976)	Finger temp	3 systematic 4 unsystematic case studies	Frequency. Duration. No pills.	7	-	6-14 weeks 2 sessions weekly	Ergotamine and Cafergot	2 out of 3 decrease temp.	Baseline 4-6 wks	Improvement on warming (not cooling) Frequency = 59% decrease Duration = 46% decrease Medication = 40% decrease

AUTHOR	INDEPENDENT VARIABLE	DESIGN	DEPENDENT VARIABLE	SUBJECTS	DIAGNOSTIC CRITERIA	TREATMENT	CONCURRENT TREATMENT	BIOELECTRIC RESPONSES	FOLLOW-UP METHOD, DURATION	CLINICAL RESPONSE
Warner and Lance (1975)	Relaxation	Group	Migraine & chronic tension. Improvement. Medication. Frequency. Duration. Severity.	25 13 tension 8 migraine 4 mixed	-	4 sessions at weekly intervals	-	-	Questionnaire sent after 6 months Those not returning questionnaires were phoned	Migraines n = 12 2 = free of headaches 6 = decrease of 50% 4 = no change 0 = worse 7 decrease in medication
Werder (1978)	Finger temp FB and relaxation and autogenic phrases	Group	Activity. Duration. Disability. Degree of discomfort. Drug usage.	4 children (10-17 yrs)	-	3 patients seen weekly for 6 weeks. 1 patient daily for 5 days	Aspirin Cafergot Florinal Darvocet-N	-	Follow-up 3 monthly 1 telephoned after 2 yrs	1 still in treatment 3 improved
Wickramaskera (1973)	Finger temp (differential hand-head training)	2 systematic case studies	Frequency Intensity Medication	2	-	16 wks EMG 10 wks finger temp (1 session per week)	Analgesics	Finger temp increase	Baseline 3 weeks Follow-up 3 months Not specified how	EMG - no response. Duration and intensity decreases as finger warming increases
Zamani (1974)	CVMR vs relaxation	Group	Frequency, Duration. Peak pain rating. Medication.	14	-	CVMR - 8 sessions, Relaxation 6 sessions,	-	-	Baseline 6 weeks Follow-up 6 weeks	CVMR most effective

For evaluation purposes, each of the above studies is placed into one of the following six categories.

1. Finger temperature feedback as a single independent variable.
2. Finger temperature feedback and other strategies as a composite independent variable.
3. Temporal artery amplitude feedback.
4. Temporal artery amplitude feedback and other strategies as a composite independent variable.
5. Progressive relaxation as a treatment procedure.
6. Other treatment approaches.

Category 1. : Finger temperature feedback as a single independent variable.

Of the eight experiments using finger temperature feedback as a single treatment strategy, two utilised group controlled designs (Beasley , 1976 and Mullinix, Norton, Hack and Fishman , 1978).

Beasley (1976) found that finger temperature biofeedback alone was not as effective in reducing headache frequency and intensity as a combination of autogenic phrases, relaxation, Electromyograph feedback (EMG) and finger temperature biofeedback. A combination of autogenic phrases and relaxation was however found to be ineffective in reducing headache frequency and intensity. As the single effect of EMG biofeedback was not controlled for, conclusions regarding the comparative effects of finger temperature and EMG biofeedback cannot be reached.

Mullinix, Norton, Hack and Fishman (1978) is the only experiment to employ a placebo control group. They reported that an improvement in migraine symptoms was not correlated with finger temperature increases as improvement was similar between a 'true' finger temperature

feedback group who showed an increase in finger temperature and a 'false' finger temperature feedback group who did not. The authors concluded that the study supports the growing notion that raised finger temperature is not correlated with positive therapeutic responses in migraine patients.

Of the remaining studies in category 1, four employed group designs: (Alvin , 1974 ; Reading and Mohr , 1976 ; Graham ,1974 and Peck , 1980). Alvin (1974) reported 'impressionistic' findings of the effects of finger temperature control with ten patients. Claims that two thirds of the patients improved should be treated with caution since dependent variables, diagnostic criterion, duration of follow-up and details of temperature changes were not reported. Reading and Mohr (1976) reported an improvement in frequency, intensity and duration of headaches in six patients over an eight week follow-up period. Successful elevation of finger temperature was defined as a rise of $0.1^{\circ}C$ or more, but given that a short stabilisation period of two minutes preceded the treatment phase, drift may have contaminated the results. The uncontrolled nature of this study makes it unlikely that the elimination of placebo and expectancy effects was achieved. Graham (1974) reported that a $2^{\circ}F$ rise in finger temperature could be achieved through hypnotic induction without the aid of feedback. A finger temperature biofeedback group and a hypnosis plus feedback group also achieved finger temperature increases of similar magnitude. As improvement was uniform across the groups, no conclusions can be drawn regarding the superiority of finger temperature control. Andreychuk and Skriver (1975) compared finger temperature feedback, alpha EEG feedback and self hypnosis. Although all groups showed a significant improvement, the 79% reduction in headache 'density'

achieved by the finger temperature feedback group compared with the 43% and 36% reductions as shown by the EEG and hypnosis groups respectively, would indicate the comparative superiority of finger temperature feedback. Peck (1980) was unable to demonstrate a differential effect with EMG feedback and finger temperature biofeedback. Even though no temperature increases were observed and EMG changes were minimal (less than 1 microvolt) a decrease in frequency of headaches was reported at a 24 week follow-up. A second follow-up at 73 weeks found that all dependent variables reverted to baseline.

Category 2 : Finger temperature feedback and other strategies as a composite independent variable.

In this category there is one control group study, twelve group design studies, one systematic case study and four anecdotal case studies.

The main feature of these studies is the inclusion of strategies designed to enhance the ability of the patient to elevate finger temperature. As Graham (1974) has shown with hypnotically induced finger temperature elevation without feedback, such strategies themselves may be potent modifiers; thus the addition of such strategies should be accompanied by control procedures to investigate their individual roles in the achievement of any change. Most commonly, autogenic phrases (Schultz and Luthe ,1969) have been used as an aid to finger temperature elevation (Blanchard, Theobald, Williamson, Silver and Brown , 1978 ; Sargent, Green and Walters , 1972 ; Sargent, Green and Walters , 1973a; Sargent, Green and Walters , 1973b ; Solbach and Sargent , 1977 and Mitch, McGrady and Iannone , 1976). The studies by Sargent, Green and Walters 1972, 1973aand 1973b, Solbach and

Sargent (1977) and Mitch, McGrady and Iannone (1976) all report the combined effect of autogenic phrases and finger temperature control on migraine activity, although experimental design deficiencies, poor diagnostic assessment and follow-up procedures prevent clear conclusions from being drawn. In the controlled study by Blanchard et al (1978), it was found that progressive relaxation training was as effective, or slightly more effective than autogenic finger temperature control training, but the effectiveness of finger temperature feedback alone as a treatment remains unclear. Werder (1978) found that three out of four child patients improved with a combination of finger temperature control, autogenic phrases and relaxation exercises and Medina, Diamond and Franklin (1976) reported positive findings when Tension and Migraine patients were treated with EMG feedback as an aid to relaxation in a complicated study including finger temperature control with autogenic phrases.

EMG feedback has been used in combination with finger temperature feedback when symptoms of both migraine and tension headaches have been reported by patients (Diamond and Franklin , 1976 and Diamond, Diamond-Falk and DeVeno , 1978). However, due to lack of control groups, conclusions about the comparative effectiveness of the treatments cannot be made. Crosson, Andreychuk, Tiemann and Phillips (1978) compared finger temperature feedback plus autogenic phrases with finger temperature feedback plus hypnosis plus autogenic phrases. The absence of follow-up information regarding headache activity pre-empts any conclusions from being reached. Adler and Adler (1976) compared interpretive psychotherapy plus EMG feedback plus passive concentration plus finger temperature control with a group receiving the same treatments minus finger temperature biofeedback. Although an overall 81% improvement was reported, the importance of finger

temperature alone was not discussed. A lack of information regarding finger temperature changes, the inclusion of a heterogeneous group of headache sufferers (tension, migraine, mixed and cluster headaches) without diagnostic information further confuse the issue.

Gainer (1978) combined assertive training, systematic desensitisation, finger temperature biofeedback and temperature discrimination training. Although the patient was able to raise finger temperature during the feedback sessions, it was beneficial only after she was taught to become aware of the peripheral coolness that preceded the migraine attack. Such mastery enabled the subject to raise finger temperature at a critical time in the headache cycle and avert the onset of the painful phase. This procedure however, does not necessarily indicate that finger temperature elevation is the reason for headache aleviation, but rather raises the question as to whether attentional divertion is the major feature of the strategy. Kentsmith, Strider, Copenhaver and Jacques (1976) monitored dopamine-b-mono oxygenase blood levels whilst using meditation and relaxation to supplement finger temperature biofeedback. The authors speculate that the relief of migraine by biofeedback is related to the suppression of autonomic activity rather than to the direct modification of the pathogenesis of migraine. Again confounding treatment strategies render the results equivocal.

In conclusion these studies are characterised by the inclusion of different headache types without clear diagnostic differentiation, short follow-up periods with unspecified methods of information collection and a lack of control for placebo and expectancy effects. Although in general, positive therapeutic results are reported, given the deficiencies concerning physiological and methodological issues,

these studies fail to provide unequivocal support for the effectiveness of finger temperature biofeedback in the treatment of migraine.

Category 3: Temporal artery amplitude control as a single independent variable.

Of the four studies using temporal artery amplitude feedback, only one employed a control condition (Bild , 1976). Friar and Beatty (1976), Koppman, McDonald and Kunzel (1974) and Zamani (1974) all employed group studies. Bild (1976) found that temporal artery amplitude biofeedback 'seemed' more effective than EMG feedback which in turn was more effective than no-treatment in reducing migraine activity. However, there were no differences in the intensity of headaches between the two treatment groups.

Friar and Beatty (1976) found that temporal artery amplitude feedback reduced the incidence of major attacks over a 30 day follow-up period compared with patients given vasoconstrictive finger pulse feedback. This study failed to indicate the precise placement of the temporal artery plethysmograph stating that a ramification of the superficial temporal artery was used as the monitored site. This is an important point as the rationale for this procedure rests on the assumption that a modification of the pain mechanism is taking place. Friar and Beatty (1976) also used positive response to ergotamine tartrate as a diagnostic criterion for migraine headache, reasoning that it provided pharmacological evidence that induced decreases in pulse amplitude relieved migraine. However, Alvin (1974) and Morley (1977) statedthat as vasolability may be impaired by the chronic use of vasoactive drugs, care should be taken if using this as a major diagnostic feature.

Koppman, McDonald and Kunzel (1974) and Zamani (1974) concluded that temporal artery biofeedback is effective in reducing migraine headache activity, but as control conditions were lacking and the follow-up periods were short (Zamani employs a six week follow-up period, Koppman, McDonald and Kunzels (1974) is unspecified), these results are equivocal.

Category 4 : Temporal artery amplitude feedback and other strategies as a composite independent variable.

This category contains one group study (Sturgis, Tollison and Adams , 1978), and two systematic case studies (Feuerstein, Adams and Beiman , 1976 and Feuerstein and Adams , 1977).

Sturgis, Tollison and Adams (1978) used Blood Volume Pulse (BVP) feedback and EMG feedback in a counterbalanced design with two subjects suffering from combined migraine and muscle tension headaches. It was concluded that BVP and EMG relieved the respective migraine and tension components of the headaches. However it was found that the subjects were unable to demonstrate a decrease in BVP during the feedback phase but could during the post feedback voluntary control phase. If a decrease in BVP had been demonstrated during the feedback phase and continued during the no-feedback voluntary control phase, it would have suggested that a biofeedback skill had been acquired. These results however suggest that a decrease in blood pulse volume was associated with unspecified factors related to the cessation of feedback.

The two systematic case studies by Feuerstein, Adams and Beiman (1976) and Feuerstein and Adams (1977) also suffer from methodological inadequacies. Both studies included patients with mixed headaches

without adequate diagnostic specification, and include three types of vasomotor activity as indices of change (blood pulse volume (BVP), blood volume (BV) and number of vasospasms). The authors conclude that 'idiosyncratic patterns of activity emerge during biofeedback training' but are unable to relate the significance of any pattern to apparent changes in headache activity.

In conclusion, lack of diagnostic criteria, inconsistent positioning of plethysmographs and inclusion of idiosyncratic patterns of activity as evidence of vasomotor change render the results equivocal.

Category 5 : Relaxation as a treatment procedure.

Of the seven studies in this category, two incorporated relaxation as a single treatment variable (Mitchell and Mitchell, 1971 and Warner and Lance, 1975). In the first part of a two part study, Mitchell and Mitchell (1971) compared the effects of relaxation with combined desensitisation (including relaxation) and a no-treatment control. Although the no-treatment group improved significantly more than the relaxation group in this first part of the study, results from the second part of the study indicated that combined desensitisation was more effective at reducing headache activity than a single model (relaxation) approach or providing no treatment. Warner and Lance (1975) used relaxation as a treatment with mixed, migraine and tension headache patients. Relaxation was found to reduce headache activity in eight of the twelve migraine patients although the design did not control for non specific treatment effects.

The remaining studies supplemented relaxation exercises with a variety of other treatment strategies including group discussions (Hay

and Madders ,1971), mental passivity concentrated upon decreasing pressure build up (Lutker, 1971). EEG and alpha feedback (Montgomery and Ehrisman, 1976) and EMG with autogenic phrases and hypnosis (Stambaugh and House ,1977). The complexity of the designs and poor control of dependent variables make any conclusion regarding the comparative efficacy of relaxation as a treatment for migraine tentative.

Category 6 : Other treatment approaches.

Benson, Klemchuk and Graham (1974) found that transcendental meditation appeared to be of limited usefulness in the treatment of migraine headaches. Less than one third of the 17 migraine and four cluster headache patients were rated as showing improvement in headache activity. Mitchell and White (1976) conducted a study designed to determine whether self help management was superior to symptom orientated interventions. The patient was involved in an intensive two stage study for over a year and although a complete cessation of headaches was reported, no conclusions regarding the comparative efficacy of any part of the treatment package can be made. Ansell (1977) used auto-suggestion of handwarming with one patient and reports a positive therapeutic effect without including temperature change information. Lambley (1978) used an 'anti-reductionist' approach to the treatment of migraine in three patients. The treatment package included drug and behaviour therapies combined with psycho-dynamic bioenergetics and affect modifying behaviour therapy. Clinical responses included statements such as 'patient feels happier about their relationship' but reports on changes in migraine pathology are limited.

In conclusion, the use of uncontrolled multi-strategy interventions renders any judgement regarding the comparative efficacy of the treatments or components of the treatment as tentative.

I. SUMMARY

The experiments concerning finger temperature biofeedback have failed to demonstrate a relationship between increases in finger temperature and decreases in migraine pathology. Those experiments which infer such a relationship may be considered inconclusive due to design deficiencies and a lack of adequate controls. On this basis, expectancy and placebo effects may have contaminated the results. Blanchard, Andrasik, Ahles, Teders and O'Keefe (1980) used a meta-analysis procedure to determine the relative efficacy of finger temperature feedback, finger temperature feedback with autogenic phrases and relaxation exercices compared with medication placebo treatments. Blanchard et al (1980) concluded that the superiority of the behavioural treatments over the medication placebos was an indication of their therapeutic potential. However until controlled studies attempting to relate treatment interventions with hypothetical pain mechanisms are carried out, finger temperature biofeedback remains of unproven value.

Although temporal artery biofeedback is considered to be a promising treatment approach for migraine headache (Jessup, Neufeld and Mersky , 1979), there are a number of criticisms which are evident as features of the above studies. Firstly, E.M.G. feedback has been used as a control for temporal artery amplitude feedback with heterogeneous groups of headache sufferers (Bild , 1976 ; Feuerstein, Adams and Beiman , 1976 ; Feuerstein and Adams , 1977 ; Sturgis, Tollison and Adams , 1978). It would seem preferable to use a homogenous group of migraine patients and compare temporal artery amplitude feedback with the other most frequently used biofeed-

back approach, that of finger temperature control, especially as positive therapeutic outcomes have been reported for both. The second major criticism is the lack of detailed information regarding changes in temporal artery pulse activity that take place during biofeedback. When detailed information is given regarding changes in temporal artery pulse activity, the number of subjects is small and 'idiosyncratic responses' have been observed.

In conclusion, any study attempting to overcome the above methodological deficits and assess comparative effectiveness of biofeedback approaches in the treatment of migraine headaches, should incorporate the following features:

1. A homogeneous group of classic migraine headache sufferers should be selected on the basis of carefully defined diagnostic criteria.
2. The independent variables (treatments) should be chosen on the basis of experimental evidence showing that they are of potential benefit in the treatment of migraine.
3. The independent variables (treatments) should be unitary and unconfounded by other treatment strategies.
4. The independent variables (treatments) should be related to hypothetical pain mechanisms.
5. Dependent variables should include physiological responses, indices of headache activity, and concurrent treatments (medication).
6. Attention placebo and waiting list control groups should be used.
7. Changes in headache activity and medication consumption should be recorded over a baseline and a follow-up phase of adequate duration.
8. The treatment sessions should have a standard format, duration and frequency.

CHAPTER 8

PILOT STUDY.

PILOT STUDY

INTRODUCTION

The objective of biofeedback is to enable an individual to gain voluntary control over otherwise relatively involuntary processes. Before biofeedback is presented, a stable level of responding is established during a pre-experimental or pre-trial baseline phase. Experimental results are then based on the assumption that this stable level of responding would otherwise be maintained during the experimental period if biofeedback was absent. The sophistry of this assumption was illustrated by Yates (1980) who monitored finger temperature changes in two groups of male and female subjects seated for a period of 90 minutes in rooms with ambient temperatures of 20^{o} and 25^{o}C. The results that were obtained from the group seated in the lower ambient temperature room could have been interpreted as showing successful control in a downward direction, as although no feedback or instructions were provided, a significant decrease in temperature was observed. Although the decrease displayed by the higher temperature group was less evident, it was still obvious. This change in responding, Yates (1980) labelled as 'drift'. Before the concept of drift had been described, consistently small magnitude changes which characterised biofeedback peripheral vasodilatation experiments (summarised by King and Montgomery , 1980) had not been adequately explained. Surwit, Shapiro and Feld (1976) proposed that the 'arousal-like' reflexes produced during biofeedback experiments explained the ease of conditioning vasoconstriction compared to vasodilatation. Dugan and Sheridan (1976) proposed that vasodilatation may be due to the biofeedback stimulus creating orientational

responses (Sokolov , 1963) competitive with target behaviour.

Given that biofeedback experiments may be contaminated by uncontrolled influences, it was considered a pre-requisite of the clinical application of biofeedback treatments to investigate the effects of experimental procedures and ambient temperature upon the two target responses under investigation in this study: finger temperature and temporal artery amplitude.

This study was conducted to determine whether finger temperature or temporal artery amplitude changes could be observed during rest, relaxation and concentration tasks, in the absence of biofeedback. As respiratory manoevres are known to be associated with decreases in finger temperature (Lynch and Schuri , 1978), respiration patterns were monitored to investigate covariance between respiration rate and depth, temporal artery amplitude and finger temperature.

EXPERIMENT 1

THE EFFECTS OF AMBIENT TEMPERATURE, REST, RELAXATION AND A CONCENTRATION TASK ON FINGER TEMPERATURE, TEMPORAL ARTERY AMPLITUDE/RESPIRATION RATE AND DEPTH.

The first experiment was designed to investigate the effects of an extended baseline period on finger temperature and temporal artery amplitude. Respiration rate and depth were also monitored to determine the extent of covariation between the physiological responses.

METHOD

Subjects

Eight volunteer subjects, four male and four female, took part in the experiment; there were two males and two females in each of

two groups. Ages ranged from 20-32 years, (mean 24.7 years). All subjects were technical or secretarial staff from a university department and were free of peripheral vascular abnormalities.

Physiological Measures

The subjects were seated in a dimly lit, sound attenuated room kept at a constant temperature of 22$\pm$ 1°C. An adjacent room contained a Devices MX6 Recorder, on which was recorded finger temperature, temporal artery pulse amplitude and respiration depth and rate. Finger temperature was obtained using an NiCr/NiAl thermocouple probe sensitive to $\frac{1}{100}$°C, attached to the volar aspect on the third phalange of the third finger on the right hand, and connected to a Comark Electronic Thermometer (Type 1608) and thence to the MX6 Recorder. A Devices Light Reflectance plethysmograph was used to detect temporal artery pulse amplitude, which was also recorded on the MX6 Recorder. All recordings of temporal artery amplitude were made on intensity 'D' to enable objective evaluation of change. All subjects wore Beyer Dynamic Headphones during the session; in addition to providing a means of communicating, they helped to protect the photo-plethysmograph from extraneous light. The body of the headphones was placed over the ear; only the light sponge outer rim overlapped the plethysmograph so as not to disturb its placement. A second thermocouple was placed immediately under the nostril to record temperature fluctuations of inhaled and expired air. The thermocouple lead was securely fixed to spectacle frames, which were taped in position. From the air temperature changes, rate and relative depth of respiration were recorded.

Procedure

Each subject attended for one session which consisted of five

phases: (a) rest periods (3 phases); (b) a concentration task (1 phase); (c) a relaxation procedure (1 phase).

In sessions for group 1, the phase order was (a) (b) (a) (c) (a); in sessions for group 2, the phase order was (a) (c) (a) (b) (a). Groups were treated similarly in all other respects. Each phase in each session was of 15 minutes duration with an additional interval between each phase to permit re-establishment of temperature stability point. Prior to the start of the session, all subjects were given an adaptation period of 20 minutes.

(1) General Instructions: Subjects were given the following general instructions:-

"I want you to sit quietly for a few minutes before the experiment begins. When you are relaxed the experiment will start. During the experiment there will be two tasks I will ask you to complete, each task taking 15 minutes, and between each task there will be a rest period of a similar length. The experiment will take a little over an hour and a half. Do you feel comfortable? If you feel the need to move, do so quickly, but do not move excessively or fall asleep. Please do not breath through your mouth at all. Any questions?"

At the end of each phase, the experimenter outlined the conditions of the next phase.

(2) Concentration Task: Instructions for the concentration task were as follows:-

"I am going to play you a news item; I want you to listen to it carefully. This will be played five times during this fifteen minute period. I want you to remember as much of what is played as possible. At the end of the 15 minutes I will ask you one or two questions about it. Sit quietly and relax, and concentrate on the tape."

The following tape of one minute duration was then presented at minutes 1, 4, 7, 10 and 13 of the phase:-

"How often are young male deaths due to road accidents? According to a briefing paper published by the Office of Health Economics, in the 15-19 age range, accidents accounted for 62% of deaths among males. Women, on the other hand, are far more likely to fall down stairs when they are 80 than to kill themselves on a motorbike at 17; 16% of all accidental deaths to females happen at this age. Accidents cause 40 deaths a day in England and Wales, a total of 15,069 in 1976; a number which has not increased this century but the death rate expressed in the context of population growth has fallen by about 36% since the beginning of the century to a level of 306 per million of the population in 1976; that is, 2.5% of all deaths, although this proportion is considerably higher in children than adults."

(3) Progressive Relaxation: Subjects were given instruction in progressive relaxation exercises (abbreviated from Jacobson, 1938). The instructions were presented over the headphones by the experimenter and subjects were asked to remain as still as possible for the duration of the phase.

(4) Rest Phase: Subjects were simply asked to make themselves comfortable and sit quietly.

Data Reduction and Analysis

Finger temperature stability was considered to have been achieved when temperature remained within 0.2°C for four consecutive minutes with a minimum of 10 minutes and a maximum of 20 minutes. All subjects were allowed to acclimatise to the experimental environment for 15

minutes prior to the administration of the baseline period.

Baseline temperature was calculated as the mean of the last four minutes of the stabilisation phase. During training sessions, finger temperature was examined every 30 seconds and the mean deviation from the established baseline was calculated; this procedure provided a conservative assessment of the degree of temperature change, since it incorporated data from all stages of the training session. Temperature was allowed to restabilise to the above criterion between each phase. Temporal artery pulse amplitude was examined at the same intervals and was averaged over a five second period. Respiration depth was also sampled every 30 seconds, time linked to the finger temperature and temporal artery amplitude measures. Respiration rate was assessed every minute. (See Appendix 2.1).

Results

Two Way (order x trials) split plot Analyses of Variance were used to examine temporal artery pulse amplitude and finger temperature variations. No significant differences were found across the 'order' or 'trial' factors for temporal artery pulse amplitude recordings. For finger temperature, no significant difference between the 'order' factors was found, but a significant difference between 'trial' factors was obtained ($F = 8.65$, $p > 0.01$).

The significance of the linear component of the 'trial' sum of squares ($F = 33.57$, $p > 0.01$) indicates that the trend in finger temperature was due to the temporal aspects of the experiment irrespective of experimental conditions (see Figure 1).

MEAN CHANGES IN TEMPORAL ARTERY AMPLITUDE RESPIRATION DEPTH RESPIRATION RATE AND FINGER TEMPERATURE IN EXPERIMENT 1.

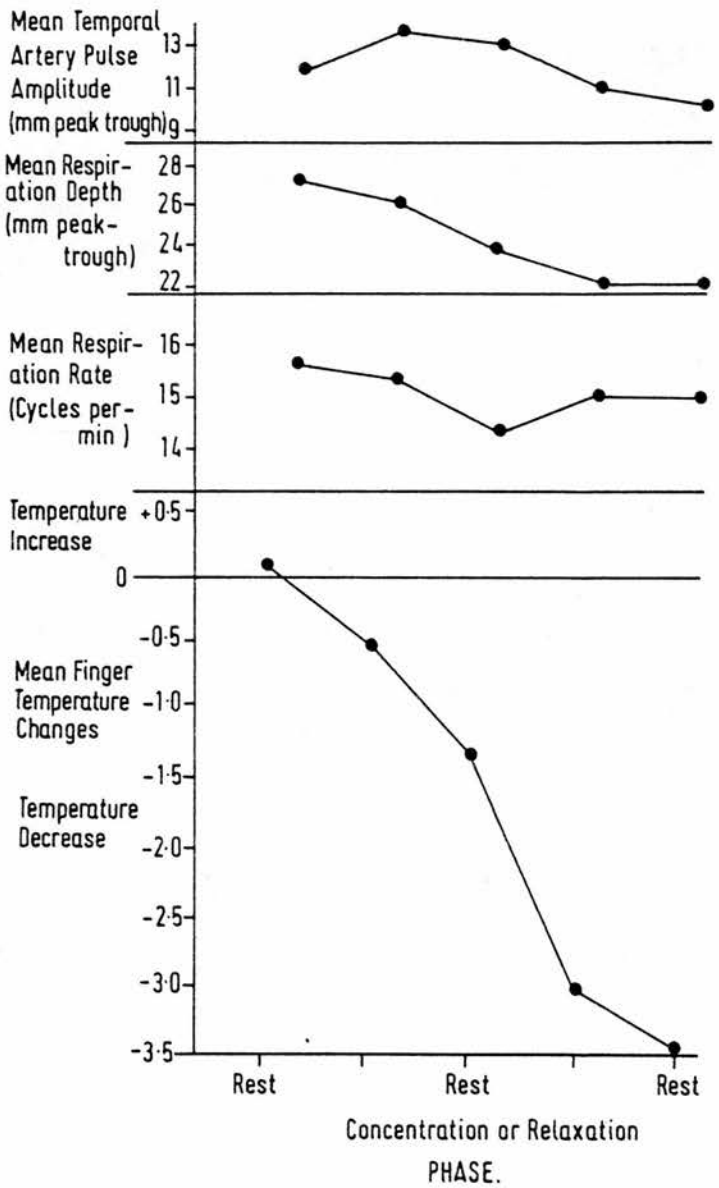


FIGURE 1

Pearson Product moment correlation coefficients between finger temperature, temporal artery pulse amplitude and respiration measures showed wide variation across subjects and phases with no consistent significant relationships emerging.

DISCUSSION

Finger temperature decreased consistently throughout the session; however this was not related to the specific tasks and instructions presented. Temporal artery pulse amplitude, respiration depth and respiration rate remained constant throughout experimental conditions. No consistent relationships were found between finger temperature, temporal artery pulse amplitude and the respiration measures. It would appear therefore that over the extended no-feedback baseline period, finger temperature showed drift in a downward direction (negative drift) at an ambient temperature of $22\pm1^{\circ}C$, when a pre-experimental reference point is used. These findings validate Yates (1980) suggestion as the drift was independent of the conditions provided.

The consistent 'negative drift' observed in finger temperature is clearly antagonistic to the usual clinical goal of temperature increase as incorporated in the clinical application of biofeedback for migraine headaches.

EXPERIMENT 2

THE EFFECTS OF RELAXATION, A CONCENTRATION TASK AND FINGER TEMPERATURE BIOFEEDBACK UPON FINGER TEMPERATURE.

The first experiment showed that, over a number of conditions presented in a single session, finger temperature gradually decreased. The decrease was independent of the presented conditions but was consistent over time, indicating a temporal, rather than task related phenomenon.

The second experiment was designed to investigate this further, over a larger number of sessions, by examining the effects of adding

biofeedback and instructions to raise finger temperature. This condition was compared with finger temperature changes obtained under near identical conditions but without instructions to raise finger temperature, and with task demands which would deny the opportunity of using thermally related imagery. The effects of progressive relaxation on temperature were also examined.

METHOD

Subjects

There were six subjects, three male and three female; age ranged from 27-32 years (mean 29.6 years). All were from university technical and secretarial staff and all were free of peripheral vascular abnormalities. None of the subjects had participated in Experiment 1.

Procedure

This experiment comprised six weekly sessions; each session consisted of a relaxation phase, a concentration task phase (resembling finger temperature biofeedback) and a true biofeedback phase. The phases were fully counter-balanced within sessions to control for the effects of order and of carry-over from one phase to another. Thus all subjects received all possible combinations of experimental conditions over the sessions. Each phase within the session was of 10 minutes duration and between each phase was an interval of varying duration during which finger temperature restabilised to a criterion of no more than $0.2^{\circ}C$ change over four consecutive minutes. Prior to the start of the sessions all subjects were given a baseline period to permit temperature stabilisation as in Experiment 1.

(1) Progressive Relaxation: Instructions were given as in Experiment 1.

(2) Temperature Feedback: Finger temperature was recorded as in Experiment 1, except that the output from the Comark thermometer was fed into a Solatron 7055 Micro-processor Digital Voltmeter (DVM). The LED display of the DVM was transmitted by a Pye FET Lynx automatic camera to a TV monitor in the subject's room; this continuous numerical display functioned as the feedback stimulus, presenting updated finger temperature every second, accurate to $\frac{1}{100}$ °C.

(3) Concentration Task: The stimuli presented in this phase were identical to those used in the biofeedback phase but differed in that the display was not related to subject changes; and in that subjects were instructed to memorise aspects of the LED display (see below for details). The task was arranged as follows:-
When the DVM is in operation, small internal currents are shown on the LED display. Changes in the display were obtained by manually switching between DC and AC measurement. The maximum figures (+15.00 and -15.00) were displayed after the DVM had been left on for several minutes. The DVM was programmed to display the change at 1 second intervals, the same intervals as used with the biofeedback display.

(4) Instructions: The following basic instructions were presented to all subjects with appropriate modifications for the counterbalancing of the phase orders:-
"This experiment consists of three phases:- Phase 1: You will be played a set of relaxation instructions which will last for six minutes. On completion of the exercises there will be a short time interval for you to maintain a state of mental and physical tranquility. The phase will last for ten minutes. Phase 2: This display has

nothing to do with your finger temperature; in fact it has nothing to do with any measurement of you at all. You will see that the display will alter at 1 second intervals in either an upward or downward direction, as indicated by the sign in front of the figures, one sign indicating a positive number and another sign indicating a negative number. The numbers over the next ten minutes will change continuously. Please watch them carefully and try to remember the highest positive number and the lowest negative number registered throughout the ten minute period. It is not important to remember the whole number, but I would prefer it if you could remember at least the first two digits. I will then ask you to recall them at the end of the ten minute period. Phase 3: You will have presented to you your finger temperature changes at intervals of 1 second. I want you to raise your finger temperature; use any means but physical ones. Do not touch the thermometer probe or use any bodily movement in any way. Sit quietly and concentrate upon making your finger temperature warmer. Please do not fall asleep. Do you have any questions?"

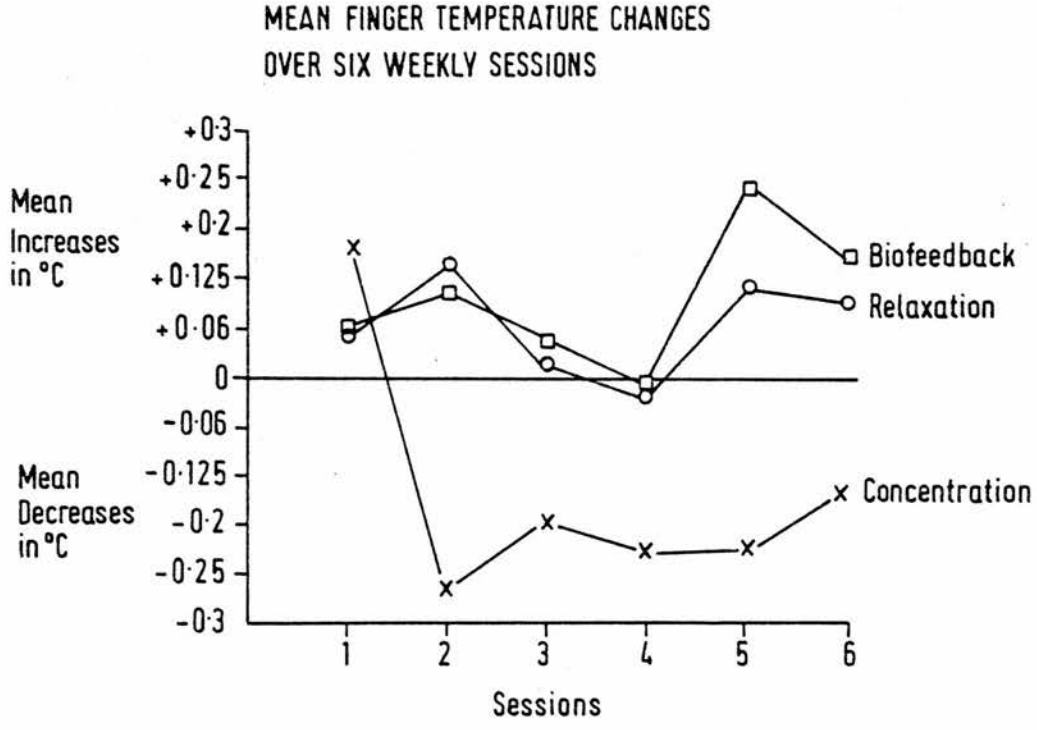
At every change of phase the experimenter entered the subject's room and informed them of the next experimental phase. When the subjects were confident of the nature of the next task, they were left to prepare themselves.

Results

As in Experiment 1, finger temperature was examined every 30 seconds and the mean deviation from the pre-experiment phase baseline was calculated. (Appendix 2.1). A repeated measure, Analysis of Variance of the three conditions over the six sessions revealed

significant differences. ($F=6.94$, $p > 0.01$). A Post Hoc analysis of significance (Scheffe Test) revealed a significant difference between the relaxation and concentration task conditions ($t=3.12$, $p > 0.01$) between the concentration task and biofeedback conditions ($t=3.55$, $p > 0.01$), but no difference was found between the biofeedback and relaxation conditions ($t=0.41$ n.s.). Figure 2 shows the mean temperature changes across subjects for each session separately. The apparent close correspondence between temperature variations in the relaxation and biofeedback phases ($r = +0.797$) is just short of statistical significance ($t=2.64$, $p < 0.058$).

FIGURE 2.



DISCUSSION

Experiment 1 showed that during an extended period of maintained passive attention, a significant decrease in finger temperature was observed. The decrease was not associated with a change in temporal artery pulse activity, respiration rate or respiration depth and was independent of experimental conditions. As temperature did not restabilise to the baseline level after each experimental condition, as would be expected if orientation had occurred (Sokolov , 1963), the results would appear to be consistent with the drift hypothesis. Experiment 2 further investigated the decrease in finger temperature and found that it was counteracted when biofeedback was provided.

In Experiment 2 a close correspondence between the temperature variations in relaxation was noted, but failed to reach significance. This correspondence may reflect a common mechanism between finger temperature and relaxation, associated with similar autonomic changes. This would be consistent with previous reports indicating a positive relationship between relaxation and skin temperature (Boudewyns , 1977). However, conclusions regarding the correspondence in temperature between the relaxation and feedback groups must be tentative especially as a decrease in temperature during relaxation was evident in Experiment 1.

One implication of these data is that when biofeedback is provided, small changes (or even no change) in finger temperature may still be a demonstration of control, since under comparable experimental conditions but without feedback, temperature will probably decrease.

CONCLUSIONS

1. Experiment 1 showed that finger temperature decreases in the passive attentive subjects are independent of temporal artery amplitude changes, respiration changes and experimental conditions. It is proposed that the decrease in temperature is most appropriately explained in terms of drift.
2. The presentation of finger temperature biofeedback militates against the negative drift in finger temperature.
3. Small finger temperature increases obtained during biofeedback may still be a demonstration of control, given that a temperature decrease may occur in the absence of feedback.

CHAPTER 9

MAIN STUDY:

A CONTROLLED COMPARISON OF BIOFEEDBACK METHODS AND RELAXATION IN THE TREATMENT OF MIGRAINE HEADACHES.

MAIN STUDY

INTRODUCTION

The rationale for a controlled clinical group outcome study emerges from the previous chapters which reviewed the theoretical, empirical and clinical status of finger temperature biofeedback, temporal artery pulse amplitude biofeedback and progressive relaxation. It is indicated that there are possible therapeutic benefits to these treatment approaches, however future studies should seek unequivocally to demonstrate such benefits. The aim of this study was to evaluate the relative contributions of finger temperature biofeedback, temporal artery pulse biofeedback and progressive relaxation training in the treatment of classic migraine headaches. An attention placebo condition (biofeedback mediated heart rate decrease) and a waiting list control group were incorporated to fulfil Paul's (1966) stringent criteria for control groups.

HYPOTHESES

1. Finger temperature for the finger temperature biofeedback group will be significantly higher than in the other treatment conditions.

2. Temporal artery amplitude pulsations will be significantly reduced in the temporal artery feedback group as compared with the other treatment conditions.

3. Heart rate decreases for the heart rate biofeedback group will be significantly greater than in the other treatment conditions.

4. In the progressive relaxation group, heart rate would decrease, temporal artery amplitude pulsations will remain stable

(evidence from pilot study) and finger temperature will decrease. A decrease in finger temperature is anticipated due to the effects of 'drift' (evidence from the pilot study), which is contrary to other findings (Boudewyns ,1966).

5. That increases in finger temperature and decreases in temporal artery pulse amplitude will be associated with a decrease in headache activity and medication.

6. The therapeutic value of finger temperature and temporal artery amplitude biofeedback will be greater than for progressive relaxation exercises. Patients in the attention placebo and waiting list control conditions will show no clinical benefit over the experimental period.

PATIENTS

The patients were collected from two sources, the out-patient department of a general hospital, and a community general practice. The collection and assessment of the patients was carried out over the period of one year (January 1979 to January 1980). All patients had histories of headaches and had received neurological investigation. 67 patients were assessed over the one year period of which 13 were rejected on the basis of their diagnosis. The remaining 54 patients were included in the project. Six patients withdrew from the project prior to the treatment period. The remaining 48 patients were randomly assigned across the five experimental conditions. The randomisation procedure was based upon random number tables, thus the allocation of a patient to a particular condition was independent of patient characteristics. There were 38 female and 10 male patients; the mean age was 38.02 years (standard deviation 13.4 years) and the

length of illness ranged from 3 years to 35 years. Of the 48 patients, 8 were in Social Class 4, 16 in Social Class 3, 13 in Social Class 2 and 9 in Social Class 1. (2 were unclassified.)

Allocation to the experimental conditions was as follows:

Finger temperature control condition

10 patients, 7 female and 3 male. Mean age 37.7 years (SD 15.5). Length of illness ranged from 6 to 28 years. 1 patient dropped out during the treatment period.

Temporal artery pulse amplitude control condition

10 patients, 8 female and 2 male. Mean age 31.6 years (SD 14.4). Length of illness ranged from 8 to 31 years. 2 patients dropped out during the treatment period.

Progressive relaxation condition

9 patients, 7 female and 2 male. Mean age 34.9 years (SD 11.4). Length of illness ranged from 4 to 20 years. 2 patients dropped out during the treatment period.

Heart rate control condition

10 patients, 9 female and 1 male. Mean age 43.9 years (SD 13.6). Length of illness ranged from 3 to 45 years. 1 patient died and 1 patient dropped out during the treatment period.

Waiting list control condition

9 patients, 7 female and 2 male. Mean age 41.9 years (SD 9.6). Length of illness ranged from 5 to 19 years. 2 patients received treatment at the end of the waiting list period. 6 patients declined to reply to the offer of treatment and 1 patient deferred the offer of treatment indefinitely.

Of the 39 patients in the four treatment conditions, 1 died and 6 withdrew during the treatment period of the project. There was a treatment attrition rate of 15%.

DIAGNOSIS OF CLASSIC MIGRAINE HEADACHE

The diagnosis of classic migraine has been a topic of considerable debate, as reviewed in chapter 3. This project incorporated a modified version of the Waters (1970) headache questionnaire for diagnostic purposes. The major difficulties underlying the diagnosis of classic migraine for the purpose of clinical evaluation studies are as follows:

1. Factor analysis of questionnaires from clinically diagnosed migraine headache sufferers has failed to reveal a single factor which encompasses the three migraine features. (Zeigler, Hassanein and Hassanein , 1972 and Peck and Attfield , 1981). However Peck and Attfield (1981) did observe a factor which comprised of two of the three classic features, a criterion which would be considered to indicate the presence of migraine using the diagnostic guidelines of Friedman, Finley, Graham, Kunkle, Ostfield and Wolff (1962).

2. There is considerable overlap in the pathophysiology of migraine and muscle tension headache (Bakal and Kaganov , 1977) to the extent that 'mixed' headaches have been considered a variant of migraine in clinical evaluation studies (Adams, Feuerstein and Fowler , 1980).

It was intended that the above difficulties would be minimised given that the Waters (1970) questionnaire has shown a 90% agreement with independent clinical raters for the diagnosis of migraine. Further, that the questionnaire was supplemented with questions designed to identify patients displaying predominantly migraine features in the relative absence of tension headache symptoms (Bickerstaff , 1977) and cluster headache symptoms (Ekbom 1970) In this way, it was intended that a homogenous group of predominantly

classic migraine headache patients would emerge.

The questions were divided into two categories:

Category 1, identifying the symptoms of classic migraine (7 questions)

Category 2, identifying symptoms of tension and cluster headache (4 questions).

Category 1 (see questionnaire. Appendix 1.1)

The patient scored 1 point per question if:

Question 14. Headache is usually or always unilateral (symptom 1)

Question 16a. Sensory/Motor disturbance is present in aura phase (symptom 2).

Question 16b. Sensory/Motor disturbance is present in aura phase (symptom 2).

Question 15. Other pre-headache warning symptoms during the aura phase (symptom 2).

Question 19. Visual disturbance is present in aura phase (symptom 2).

Question 20. Loss of appetite accompanies headache (symptom 3).

Question 21. The patient ever/usually or always vomits during the headache (symptom 3).

Category 2

The patient scored 1 point per question if the following were absent:

Question 11. The headache usually or always has a tight band quality to the pain.

Question 9. The headache can be relieved by movement or walking around.

Question 10. The patient experiences unilateral nasal congestion, watering eyes or rhinorrhea as a regular feature of their headaches.

Question 18. The headaches occur in 'clusters' over a short period of time with periods of six months or more separating the 'clusters'.

To constitute a diagnosis of classic migraine, each patient scored at least 4 points from category 1, of which two of the three major symptoms were included, and 4 points in category 2. If a score of 5 points was obtained in category 1, a minimum of 3 points were obtained in category 2 for the patient to be diagnosed as suffering from classic migraine. However, those patients who scored more than 2 points in category 2, irrespective of the category 1 score were excluded from the project on the basis that significant pathology of another headache type was apparent. The minimum 'point' score to constitute a diagnosis of migraine was therefore 8. Of the 67 patients assessed, 13 scored less than 8 points, 20 scored 8 points, 24 scored 9 points, 6 scored 10 points and 4 patients scored 11 points.

Information from the 67 questionnaires obtained during this study was incorporated in a statistical analysis of the Waters Headache Questionnaire. The results of this study, 'Migraine symptoms on the Waters Headache Questionnaire : A Statistical Analysis' (Peck and Attfield ,1981) are included in Appendix 1.2.

APPARATUS

The patients were seated in a dimly lit sound attenuated room kept at a constant temperature of $22 \pm 1^{\circ}C$. An adjacent room contained the following:

A heated stylus, Devices MX6 Recorder (MX6) containing a DC (3461) preamplifier and two pulsemonitor (3611) preamplifiers. One 3611 preamplifier was set on AC mode and the other on DC mode.

A Solatron 7055 Microprocessor Digital Voltmeter (DVM)

A Solatron Data Transfer Unit (DTU)

A Devices Instantaneous Ratemeter type 2751 (ratemeter)

A Comark Electronic Thermometer type 1608 (thermometer)

A Pye Lynx Automatic Camera (camera)

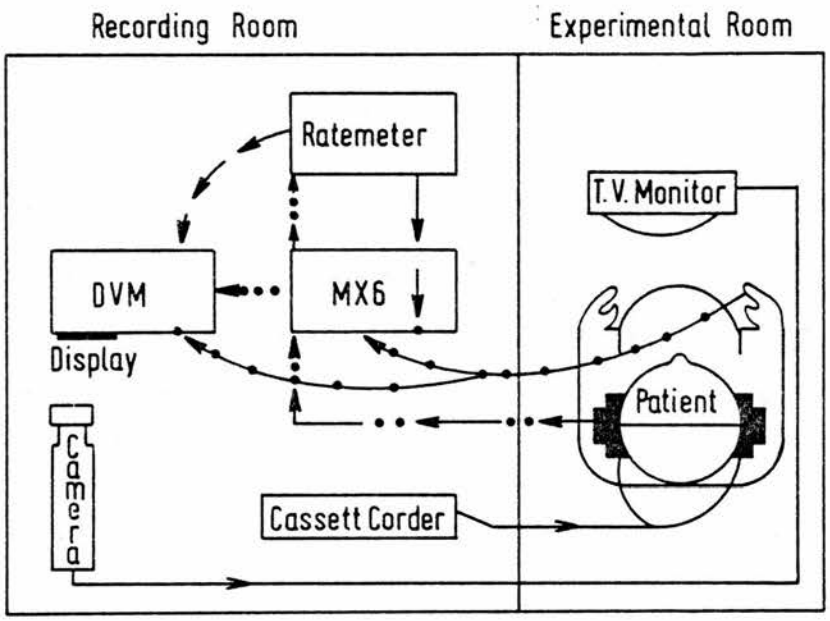
A Sony Stereo Cassette-corder TC-158SD (tape recorder).

Finger temperature was obtained using a NiCr/NiAl thermocouple probe sensitive to $\frac{1}{100}$ °C, attached to the volar aspect of the third phalange on the third finger on the patient's right hand. A Devices light reflectance plethysmograph was placed above the zygomatic arch on the external superficial temporal artery at the point of maximum engorgement. The placement of the plethysmograph was on the side of the patient's head that was associated with the painful phase of the migraine. The patients wore Beyer Dynamic headphones which, in addition to providing a means of communication, helped to protect the plethysmograph from extraneous light. The body of the headphones were placed over the ear, only the light sponge outer rim overlapped the plethysmograph so as not to disturb its placement.

Finger temperature monitored by the thermocouple probe was fed into the thermometer and thence to the 3611 preamplifier (DC mode) in the MX6; a second thermocouple probe was fed directly into the DVM via the DTU. Signals from the light reflectance plethysmograph were fed into the 3611 preamplifier (AC mode) of the MX6, whereby a wave trace of temporal artery pulsations was obtained. Output from the 3611 preamplifier (AC mode) was also transmitted to the ratemeter whereby a calibrated heart rate signal (beats per minute) were fed into the 3461 preamplifier in the MX6. In this way permanent records

of finger temperature changes, temporal artery pulse amplitude and heart rate could be obtained for every patient on every session.

DIAGRAMATIC REPRESENTATION OF THE BIOFEEDBACK EQUIPMENT



Finger Temperature Information

Heart Rate Information

Temporal Artery Amplitude Information

Combined Heart Rate / Temporal Artery Amplitude Information

FEEDBACK : MICROPROCESSOR PROGRAMMES AND PROGRAMME DESCRIPTIONS

Finger temperature, temporal artery amplitude and heart rate information was fed into the DTU which was linked to the DVM. All operations were measured as dc voltage: 'the results being the average value of the input during a selected period'.

The following descriptions are taken from the Solatron Technical Manual (1977).

FINGER TEMPERATURE BIOFEEDBACK

Programme Used	Description
8 (thermocouple) Option 3	Enables measurements to be displayed directly in ^{o}C. For the NiCr/NiAl thermocouple. Ambient temperature is entered into the programme and the measurement is linearised and displayed in ^{o}C.
9 (time)	This measurement does not carry out measurement processing, but brings the measurement and processing under the control of the voltmeters built in clock. When a time is selected, the voltmeter presents the answer at the end of the run without regard to the results of the previous one.

The patient was presented with a display of finger temperature accurate to $\frac{1}{100}\,^{o}C$. The feedback was updated at 1 second intervals.

TEMPORAL ARTERY AMPLITUDE BIOFEEDBACK

Programme Used	Description
5 (Max/Min)	The programme can determine the maximum-to-date, the minimum-to-date or the peak-to-peak value of a long series of measurements.
Option 3	The peak-to-peak value. This option gives an algebraic subtraction of the minimum from the maximum.
7 (statistics)	Calculates trends; mean, variance, and standard deviation, all statistical analysis calculations; also root-mean-square values. The option selected governs what is displayed during the running of the programme; the user can recall all results from memory at the end of a run.
Option 1	Gives the average to date value, subject to continuous updating while the programme is running. At the end of each measurement run the average value of the measurements is displayed.
9 (time)	As with the finger temperature biofeedback condition. This programme is regarded as particularly useful when used with programme 7 (statistics) as it allows measurements to be made over a fixed time period and an average reading to be obtained. When a run time is selected, the voltmeter will present an answer at the end of the run, then restart a further run without regard to the results of the previous one.

The patient was presented with a value analogous to variations in the mean size of the temporal artery pulsations. These values were

the updated average of the measurements, presented at 15 second intervals. The slight variations in pulse amplitude found across subjects and within subjects across sessions meant that pulse amplitude readings had to be taken at different plethysmograph light intensities, to gain clear signals. Although the same patient may have shown varying size of pulse amplitudes across sessions, within session recordings were all obtained on the same light intensity. The presentation of updated signals at 15 second intervals provided a method of overcoming the following deficiencies:

1. presenting the patient with rapidly fluctuating and thus potentially confusing feedback;
2. of not being able to monitor magnitude of change, as would be the case if binary feedback was given (Koppman, McDonald and Kunzel , 1974 and Feuerstein, Adams and Bieman ,1975), or of having 'idiosyncratic responses' (such as simultaneous changes in blood pulse volume, blood volume and frequency of vasospasms) interfere with the feedback signal (Feuerstein and Adams ,1977).

Friar and Beatty (1976) presented feedback after every 20 pulse beats: thus the 15 second intervals used in this study would represent a similar relationship between number of beats and presentation of feedback.

HEART RATE BIOFEEDBACK

Programme used	Description
7 (statistics)	As with temporal artery amplitude feedback.
Option 1	Gives a continuous updating while the programme is running. At the end of each measurement run the average value of the measurements is displayed.
9 (time)	As with the finger temperature and temporal artery amplitude biofeedback conditions. When a run time is selected, the voltmeter will present an answer at the end of the run, then restart a further run without regard to the results of the previous one.

The patient was presented with a value representing the average heart rate (in beats per minute). The feedback was updated and presented at 15 second intervals.

Heart rate biofeedback was chosen as a placebo treatment in this project as it was considered to fulfil the following criterion:

> "A placebo is a procedure with no intrinsic therapeutic value, performed in controlled studies to determine the efficacy of other clinical procedures".
> (Dorland's Medical Dictionary ,1977).

The reasons for the choice of heart rate decrease biofeedback are as follows:

1. No study has attempted to develop control of heart rate as a method of treating migraine and there is no evidence to suggest that it would be of therapeutic value.

2. Heart rate biofeedback did not require the placement of extra transducers and did not produce any direct physiological effect upon finger temperature or pulse amplitude responses.
3. Heart rate biofeedback studies would suggest that there is no difference between types of biofeedback display (Blanchard, Scott, Young and Haynes , 1974) or schedules of display (Hatch , 1980), in eliciting heart rate decreases. It was therefore considered unnecessary to construct a separate means of display for heart rate biofeedback.
4. The pulsatile nature of the pain often associated with migraine headaches gives a credible association between heart rate and migraine headaches.
5. A decrease in heart rate indicated a decrease in arousal; thus a biofeedback task consistent with lowering arousal may be construed by patients as a relevant treatment approach.
6. As it is possible to produce heart rate decelerations comparable with those accepted as significant with biofeedback by instructing subjects to lower heart rate (Lang and Twentyman ,1976), some of the placebo condition patients would experience "success" independent of their biofeedback performance. The effect of the perceived control may maintain the patient's motivation in regular treatment session attendance.

Finger temperature, temporal artery amplitude or heart rate were then shown on a light emitting diode (LED) display on the DVM. The LED display of the DVM was transmitted by a Pye Lynx automatic camera to a TV monitor in the subject's room.

PROGRESSIVE RELAXATION CONDITION

Patients in the Progressive Relaxation condition were presented with relaxation exercises (abbreviated from Jacobson , 1938) over the headphones from a pre-recorded tape on the cassette recorder. No feedback of any nature was presented to patients in this treatment condition.

PROCEDURE

Assessment

Assessment interviews were conducted in the Out-patient Departments of a General Hospital or in the University of Edinburgh Department of Psychiatry. The aim of the interview was to obtain detailed information about the characteristics of the patient's headaches using a modified version of Waters (1970) headache questionnaire. The completion of each questionnaire was supervised by the experimenter thus ensuring the immediate clarification of any ambiguities concerning the questions or the patient's responses. On completion of the questionnaire, each patient was given the following information:

"A study into the treatment of headaches is being conducted at the University Department of Psychiatry; inclusion in the study will be based upon the results of the questionnaire."

Patients whose headaches were not considered to be of the type under investigation were informed of the fact as soon as possible. Their referring doctor was also informed and no further contact with the patient regarding the study was made.

Patients whose headaches were diagnosed as being of the type under investigation were informed of their inclusion in the study and then assigned to one of two groups; a waiting list group or an experimental group.

The patients assigned to the waiting list group were asked to continue with the treatment prescribed by their doctor and to keep detailed records of their headache frequency, intensity, duration and type and amount of medication taken. This recording procedure was conducted for the period of one year, after which they were offered a treatment for their headaches.

The patients assigned to an experimental group were also required to monitor headache activity and medication consumption as outlined above, and were offered a treatment which would necessitate their attendance at the Department of Psychiatry on a regular basis over a number of weeks. The treatment they were offered was a non-drug and non-psychiatric form of treatment intended to supplement and not replace any drug regime the patient was already using.

Each patient was then given a demonstration headache record form and instructions of how to record headache intensity, duration and medication consumed. (See Appendix 1.3).

The patients who did not want to be considered for inclusion in the project were immediately removed from the list of potential experimental patients. Those patients who expressed an interest in participating in the project were informed that a letter providing further details would be sent to them in due course. If their headaches were diagnosed as being of the type under investigation, a supply of headache forms accompanied the letter.

All patients were assured of the following; it was their prerogative to withdraw from the study at any point should they so wish. That the study was being conducted with the consent of their doctor who would still maintain clinical interest and responsibility for them. That no psychiatric treatment was involved and that attendance at a

Department of Psychiatry did not imply that their headaches were in any way unreal or imaginary.

Following the assessment interview, the headache questionnaires were rated by the experimenter. Patients who did not receive a diagnosis of classic migraine were informed that their headaches were not of the variety under investigation and that no further contact regarding the project would be made with them. (Appendix 1.4). The referring doctor was informed of the patients' exclusion from the experiment, reasons for exclusion were also presented. The referring doctor was offered suggestions regarding alternative treatment approaches; including relaxation classes and individual counselling. The alternative treatments were not carried out by the experimenter and no evaluation of these treatments has been attempted in this study.

The patients included in the treatment conditions were randomly allocated to one of the five experimental conditions (four treatment and a waiting list condition). The patients allocated to the waiting list condition were informed of their allocation by letter. (Appendix 1.5). The letter confirmed that the patient would be offered a treatment for their headaches after a period of one year but that during the intervening year an accurate record of their headache activity and medication consumption was of great importance. A second demonstration headache form was enclosed along with eight headache record forms for the patient's use. The patient was told that a similar number of headache forms would be sent to them at monthly intervals along with a stamped addressed envelope so that contact with the experimenter could be maintained. The letter stressed the importance of maintaining an accurate record of headache activity, medication consumption and details of changes in medication should it

occur, and that notification of a headache free period was as important as careful monitoring of headache activity.

The patients allocated to the treatment conditions were also informed by a letter outlining details of the study but not of the treatments. An appointment and eight headache record forms were included. (Appendix 1.6).

BASELINE PERIOD

During the three month baseline period, patients in the treatment conditions were requested to attend the Department at monthly intervals (at the end of the first and second months). The aim of the baseline sessions (acclimatisation sessions) was to acquaint the patients with the Department, the type of equipment used in the project and experimental procedures, in an attempt to reduce any anxieties the patients may have had regarding inclusion in the study and the treatments. Both "acclimatisation" sessions were of one hour and followed the same standard format.

Patients were seated in the treatment room where a brief discussion regarding completed headache forms preceded a description of the functions of the light reflectance plethysmograph and the thermocouple probes. The plethysmograph and the probes were then attached to the patient who was asked to remain awake, relaxed and still whilst a 20-minute recording phase was completed. On completion of the recording phase, the MX6 recordings of the finger temperature, temporal artery amplitude and heart rate recordings were shown to the patient. Questions regarding the MX6 recordings were answered by the experimenter, but no details of the relevance of the recordings for treatment purposes were given to the patients. Biofeedback was not

administered to any patient at any stage of the acclimatisation sessions. Recordings taken during the session were used for demonstration purposes only and were not included in the analysis. On termination of both acclimatisation sessions, patients were given eight headache forms and an appointment for their next attendance.

TREATMENT PHASE

At the end of the three month baseline period, patients were given appointments for the treatment phase of the experiment. On the first treatment session, patients were informed of the type of treatment they were to receive. Written rationales for treatment were provided for each patient (Appendix 1.7). Apart from the first session when discussion of the rationales preceded the treatment phase of the session, the following format was adopted on each treatment session:- On arrival, the patients were seated in the experimental treatment room. An initial discussion period of 15 minutes was allowed for each patient prior to the administration of the baseline phase of the treatment. The discussions were limited to 'headache' related topics and at no time were domestic or personal issues encouraged as topics of conversation. After the initial discussion, the apparatus was attached to the patient and the following instructions were given.

BIOFEEDBACK PATIENTS

"I want you to sit quietly for 20 minutes, after which time the television screen in front of you will display your (finger temperature/ temporal artery pulse wave/heart rate). When you see the numbers on the screen, I want you to try and (increase/decrease/decrease) them. Don't be concerned if you are unable to obtain immediate control as

it may take a little time before you see any effects. Please do not move unnecessarily, touch the sensitive probes or change the way you are breathing in an attempt to gain control as all of these strategies will hinder your attempts. There is reason to believe that the skill is best shown when calm and comfortable. Even if you have achieved control, it is often difficult to maintain and you may find that you lose the skill between sessions. At no time will you receive a shock or unpleasant sensation. If you feel uncomfortable and have to move, do so and change position but do not keep moving around longer than is necessary. If the equipment slips, or you become uncomfortable, or you would like to finish the session, then don't hesitate to call me. I will be in the room adjacent at all times during the session."

PROGRESSIVE RELAXATION PATIENTS

"I want you to sit quietly for 20 minutes, after which time you will hear a short tape of relaxation exercises played to you over the headphones. Please carry out the relaxation exercises as instructed. When the tape is ended, I will leave you to maintain a relaxed and tranquil state for 15 minutes. At no time will you receive a shock or unpleasant sensation. Please do not move unnecessarily, touch the sensitive probes or fall asleep. If you feel uncomfortable and have to move, do so and change position but do not keep moving around longer than is necessary. If the equipment slips, or you become uncomfortable, or you would like to finish the session then don't hesitate to call me. I will be in the room adjacent at all times during the session".

The 15 minute biofeedback-recording phase was thus started after the patient had been seated in the experimental room for at

least 35 minutes (15 minute discussion and 20 minutes adaptation period). This procedure would fulfil Yates's (1980) stringent criteria for minimising the effects of drift in the feedback-recording phase of the session. For all patients, irrespective of the treatment condition, finger temperature, temporal artery pulse amplitude and heart rate was recorded for the last five minutes of the 20 minute adaptation phase and for the 15 minutes of the treatment phase. A pre-experimental baseline was thus established at the beginning of every treatment session for every patient in every treatment condition.

Patients were given no suggestions or instruction as to how they might acquire feedback control. On completion of the first treatment session, patients were instructed to practice methods of complying with the treatment demands using mental rehearsal. It was suggested that mental rehearsal should be practiced in the following manner :

"Every day, sit down in a quiet, darkened room. Make sure that you are comfortable, warm and are not going to be disturbed or distracted for at least 20 minutes. Try to picture yourself in this experimental room and reproduce those feelings of control which you achieve whilst you are here". This suggestion was reinforced on every visit to the Department and patients were informed that this procedure may facilitate skill acquisition and enhance therapeutic benefits of the treatment. At no time were domestic or personal issues regarded as a means of therapeutic concern and although general conversation was encouraged as an aid to reducing anxiety, all possible consideration was taken in maintaining the above format. Patients were however encouraged to discuss the treatment with their families

and parents of younger patients were invited to attend the sessions with their children in an attempt to allay any anxieties present in the parents which may have been transmitted to their children.

Each experimental condition patient was expected to complete ten treatment sessions within the three month treatment period. The treatment sessions were thus offered on a once weekly basis, at times convenient to each patient but within normal working hours. Lunch time attendance was discouraged.

FOLLOW-UP PERIOD

On completion of the three month treatment period (ten treatment sessions) all patients were reminded of the importance of continued contact with the experimenter over the six month follow-up period. Emphasis was placed on the need for continued home rehearsal of the treatment on a regular daily basis, and the need for the accurate recording of headache activity and medications used. Patients were asked to inform the experimenter of months when headaches were absent. The patients were sent eight headache record forms, and a stamped addressed envelope every month for the duration of the six month follow-up period. At the end of the six month follow-up period, patients were informed that contact with them would be terminated.

MEASURES OF THERAPEUTIC BENEFIT

Seven indices of change were employed:

Headache frequency : The number of headache forms returned per month.

Headache intensity : The maximum intensity of pain experienced during the headache attack (rated on a 5-point scale).

Headache duration : The length of time the headache was experienced.

If a headache lasted for more than 24 hours but less than 48 hours, the two headache forms were treated separately for frequency, intensity and duration measures. If the headache lasted for more than 48 hours but less than 72 hours, the three headache forms were treated separately for frequency, intensity and duration measures, etc.

Index of headache activity : Frequency of headaches per month x maximum intensity of each headache x duration of each headache.

Analgesic score : Analgesic potency was rated in the following way: Aspirin (score 1), paracetamol (score 2), combination analgesics; aspirin and/or paracetamol and/or codeine (score 3), distalgesics (score 4), codeine, morphine and cyclyzine (score 5).

Thus if a patient takes two aspirin (2 x 1), two combination analgesics (2 x 3) and a codeine (1 x 5) during the course of a headache, the analgesic score = (2 x 1) + (2 x 3) + (1 x 5) = 13.

Vasoconstrictor medication : As vasoconstrictor drugs can be taken orally, parenterally or as suppositories; alone or in combined forms, the exact dosage on each administration is difficult to determine accurately, hence frequency of administration rather than amount was used as an index of measurement.

Prophylactic medication : The number of tablets taken as a prophylactic measure for the prevention of migraine attacks.

Although the use of valium and a range of anti-emetic drugs were observed during the experimental period, only the categories of drugs mentioned above were used in the analysis.

The above method of collecting follow-up information and the means of evaluating the information were employed to fulfil the criteria for evaluating therapeutic change as proposed by Bakal and

Kaganov (1976) and Adams, Feuerstein and Fowler (1980).

An index of headache activity was devised to indicate the degree of overall discomfort experienced by the patient during each headache. Blanchard, Theobald, Williamson, Silver and Brown (1978) used a 'Headache Index' of weekly headache ratings to measure changes in headache activity. They stated that it was a sensitive measure of headache activity and similar to the 'headache index' reported by Budzynski, Stoyva and Adler (1970). It was however an average of rated intensity and consequent incapacitation caused by the headache, and not as in this study a score of frequency x intensity x duration of headaches. There are no reports of an 'index of headache activity' as used in this study, in previous work.

Sargent, Green and Walters (1972, 1973a and 1973b) used a 7-point analgesic scale to record analgesic consumption, which Blanchard et al (1978) also adopted as being a "more sensitive measure of change than a mere pill count". The criterion of rating analgesic potency is however not specified and in the absence of such a scale the above 5-point scale was used in this study. The 5-point scale was constructed under the supervision of a pharmacist (Boots Pharmacy Department). The scale was intended to be a conservative measure of change more sensitive than pill counting, as used by Turin and Johnson (1976), Mitch, McGrady and Iannone (1976), Stambaugh and House (1977) and Warner and Lance (1975).

SUMMARY

The design of the study involved the collection of a group of headache patients from which classic migraine headache patients were diagnosed. The collection and assessment was carried out over the

period of a year (January 1979 to January 1980). The diagnosed classic migraine headache patients were randomly allocated to one of the four treatment conditions and a waiting list control condition. The conditions consisted of a finger temperature biofeedback condition, a temporal artery pulse amplitude biofeedback condition, a progressive relaxation condition and a heartrate biofeedback condition. The patients in the finger temperature biofeedback condition were given feedback of finger tip temperature and instructions to increase the temperature; the patients in the temporal artery pulse amplitude biofeedback condition were given feedback of pulse artery amplitude of the superficial temporal artery and instructions to decrease the amplitude. The progressive relaxation condition patients were given no feedback but were administered abbreviated relaxation instructions. The heart rate biofeedback condition (placebo condition) received feedback of heart rate and given instructions to decrease heart rate. The waiting list control condition patients received no treatment.

On initial contact with the patients and prior to allocation to the treatment and waiting list condition, patients were given an assessment questionnaire. The waiting list condition patients were informed that treatment would be offered to them after the period of a year, but that during the waiting period regular checks on their medication consumption and headache activity would be made. For the remaining subjects, allocation to the treatment conditions preceded a three month baseline, three month treatment and six month follow-up period.

At the end of the first month and the second month of the baseline period, patients in the four conditions attended for baseline

recording sessions. The patients were not informed about the treatment they would receive until the start of the treatment period. On each of the baseline visits, patients were familiarised with the apparatus and recording equipment that was to be used during the treatment sessions. Treatment was started at the beginning of the fourth month. The treatment was given on a regular weekly basis, at times convenient to the patient but within regular hours of the working week. Patients were discouraged from lunch hour attendance. The six month follow-up period was started on conclusion of the treatment period. During the 12 month experimental period, indices of headache activity and medication consumption were recorded for patients in all treatment conditions and the waiting list condition.

CHAPTER 10

RESULTS.

RESULTS SECTION

RESULTS OF PHYSIOLOGICAL INFORMATION

Data Reduction

Statistical analysis of the physiological information was undertaken for the 32 patients who completed the ten treatment sessions. Of the 32 'completers', eight were in the finger temperature condition, eight in the temporal artery amplitude condition, seven in the relaxation condition and nine were in the heart rate (placebo) condition.

Within every treatment session, a 20 minute stabilisation phase was followed by a 15 minute treatment phase. Baseline physiological recordings were taken during the last five minutes of the stabilisation phase (pre-experimental baseline). Recordings obtained during the pre-experimental baseline phase were used to evaluate the magnitude and direction of change that occurred during the 15 minute treatment phase of the session.

Finger temperature data

For the purpose of statistical analysis, data from the 15 minute treatment phase was divided into three five-minute periods.

During the five minute pre-experimental baseline phase and the 15 minute treatment phase (composed of three five-minute periods), finger temperature was examined every 30 seconds and the changes from the 'stability point' finger temperature calculated. Stability point finger temperature was taken as that temperature achieved by the patient at the end of the 20 minute stabilisation phase. The ten 30-second temperature values (five minute pre-experimental baseline phase) were summed algebraically, as were each of the ten 30-second temperature values in each treatment period. The pre-experimental

baseline phase value was then subtracted from each of the three five-minute period values of the treatment phase. Thus for each treatment session, three values were obtained, each representing the magnitude and direction of the temperature change from the pre-experimental baseline occurring during the three five-minute periods of the treatment session. All finger temperature information is expressed as the mean change observed at each 30 second interval of the treatment phase.

Temporal artery amplitude data

As with finger temperature data, the 15 minute treatment phase was divided into three five-minute periods.

Temporal artery amplitude recordings were examined every 30 seconds throughout the pre-experimental baseline phase and the treatment phase (composed of three five-minute periods). At each of the 30 second intervals, pulse waves were examined over a five second epoch. During each five second epoch, the maximum and the minimum pulse amplitude were recorded. The maximum and minimum pulse amplitudes were summed algebraically over the pre-experimental baseline phase and the three five-minute treatment phase periods. Mean and standard deviation values of the temporal artery pulse waves were thus calculated for the pre-experimental baseline phase and the three five-minute periods during the treatment phase.

As has been stated, slight variations in pulse amplitude size across subjects and within subjects across sessions meant that pulse amplitude recordings had to be taken at different plethysmograph light intensities to gain clear signals. The light intensity control of the Devices 3611 Pulse monitor preamplifier provides only a coarse

control of gain. As there is no indication that a direct relationship exists between recordings taken on different lamp intensity settings, relative difference scores were calculated for the three five-minute treatment periods with respect to the pre-experimental baseline phase. This form of transformation gives an index of change independent of initial levels by treating the differences as relative to the level of score (Bromley , 1971). All temporal artery information is expressed as the mean change observed during within session periods.

Heart rate data

Heart rate (beats per minute (bpm)) were obtained for each of the five minutes in the pre-experimental baseline phase and each of the three five-minute periods of the treatment phase. Magnitude and direction of heart rate changes were calculated for each five minute period within the treatment phase with respect to the pre-experimental baseline. All heart rate information is expressed as mean change in beats per minute (bpm).

The dependent variables were:

Changes in finger temperature (^{o}C), relative difference (RD) changes in mean temporal artery pulse amplitude, relative difference (RD) changes in the standard deviation of temporal artery pulse amplitude, and heart rate changes (bpm).

Changes during the within session periods relative to the pre-experimental baseline were calculated for every patient in every session of the four treatment conditions.

Analysis of physiological information

Physiological data was analysed using a 4 x 10 design (4

between treatment conditions and 10 within treatment sessions) with repeated measures on the within (treatment session) variable. Since three within session period measures were obtained, a 3 Way Analysis of Variance was computed for each of the above dependent variables, 4 between (treatment conditions) x 10 within (treatment sessions) x 3 within (treatment periods), in which there were repeated measures on the last two factors.

Computations were completed using the B.M.D.P. (Biomed) and S.P.S.S. programmes. All raw, mean and standard deviation scores are contained in Appendix 2.

FINGER TEMPERATURE INFORMATION

Table 1

Table 1 is a summary table from the 3 Way Analysis of Variance examining the effects of treatment conditions, sessions and periods on finger temperature change. (Appendix 2.2)

Table 1

Finger temperature changes for all subjects between treatment conditions, across treatment sessions and within treatment session periods

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	2 Tail Probability
Treatment	1106.24	3	368.75	0.75	0.53
Error	13728.77	28	490.31		
Period	292.67	2	146.33	3.76	0.03
Period x Treatment	185.27	6	30.88	0.79	0.58
Error	2178.40	56	38.90		
Session	3398.56	9	377.62	1.50	0.15
Session x Treatment	8054.94	27	298.33	1.19	0.24
Error	63273.48	252	251.09		
Session x Period	595.92	18	33.11	1.01	0.44
Session x Period x Treatment	1798.75	54	33.31	1.02	0.44
Error	16472.09	504	32.68		

3 Way Analysis of Variance

The results from Table 1 show that there was no significant treatment effect upon finger temperature change. The finger temperature feedback condition demonstrated no greater change in finger temperature than the other treatment conditions. Previous research has suggested that the 'skill' of finger warming can be acquired easily; within three to four sessions (Gardner and Keefe 1976, Ohno, Tanaka, Tayeka and Ikemi 1977), thus a gradual increase in temperature change over sessions would be expected. A 'learning' effect of this type may cause a treatment x session interaction. As a treatment x session effect was not apparent, it would appear that there is no indication that a learning process was taking place.

A significant within period effect ($F = 3.76$, 2df., $p < 0.03$) suggests that the finger temperature changes during the three five-minute within session periods differed significantly. The within period difference was however a feature of all treatment conditions and all sessions, as the period x treatment and period x session effects failed to reach significance. Finger temperature feedback therefore does not appear to have produced significant changes in finger temperature.

Table 2

Mean finger temperature changes within treatment sessions (per 30′ epoch)

Finger Temp	Temp Art Amp	Relaxation	Heart Rate
+0.10°C	-0.08°C	-0.19°C	+0.03°C

Although the finger temperature condition displayed a higher mean change, the treatment condition differences failed to reach a level of significance.

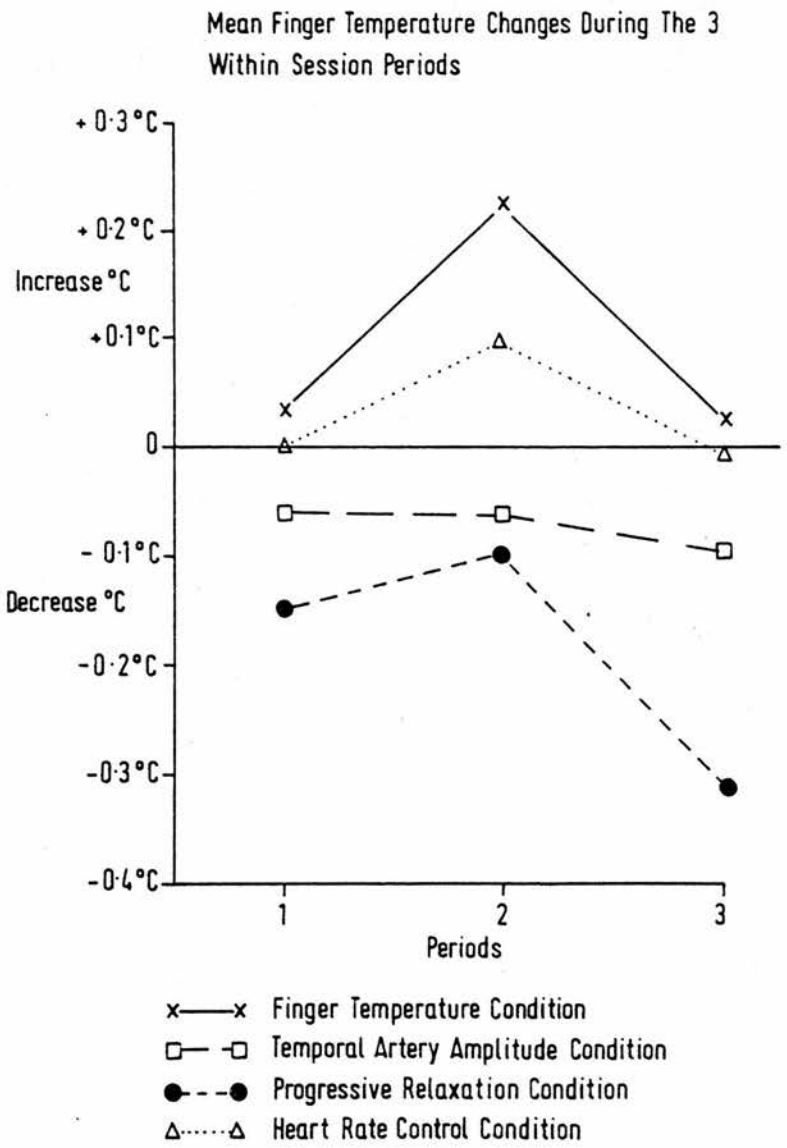
The results from the Pilot Study (Chapter 8) suggest that downward 'drift' of finger temperature may take place in an ambient temperature of 22±1°C. The decreases shown by the temporal artery amplitude and progressive relaxation conditions may have been due to the effects of drift. The greater decrease evident for the progressive relaxation condition may have been due to the combined effects of drift and the 'abbreviated progressive relaxation' exercises that preceded the 15 minute treatment phase. (Lynch and Schuri 1978).

The slight increase in temperature shown by the heart rate condition may also be within the limits of drift; this however is a speculative point in the absence of supportive evidence.

Graph 1

Mean temperature changes for the 3 within session periods.

MEAN TEMPERATURE CHANGES FOR THE 3 WITHIN SESSION PERIODS

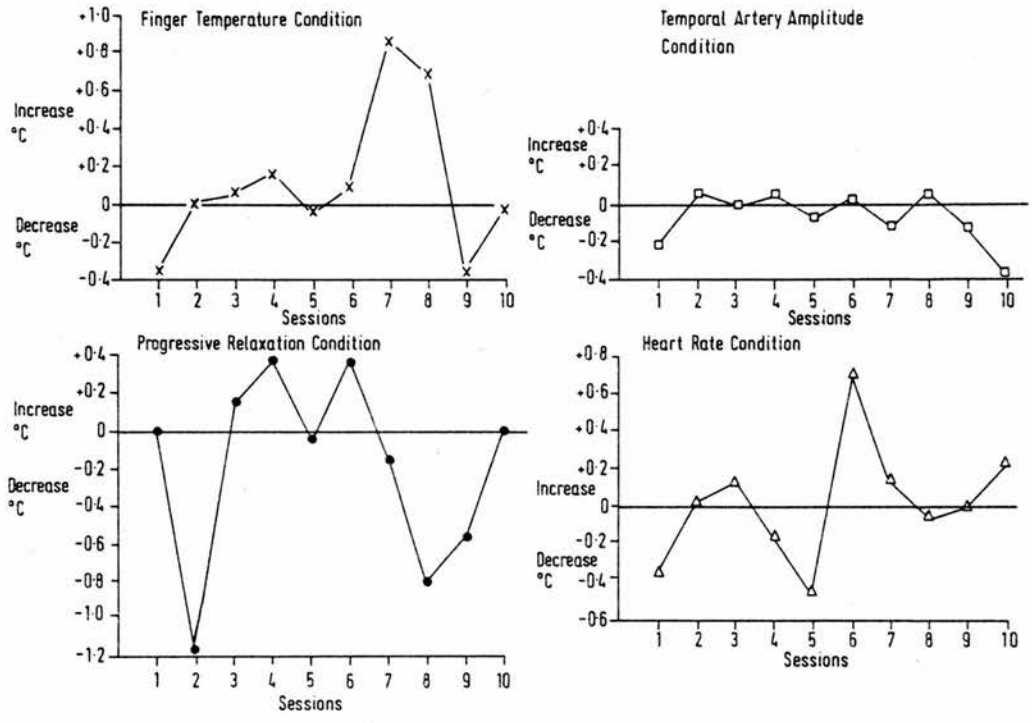


It is evident from Graph 1 that the significant 'periods' effect was due to a general tendency for finger temperature to decrease during the third period, relative to the second. This decrease may be due to the effects of drift, as it was a constant effect across all treatment conditions. Although the finger temperature condition showed an increase from baseline across all periods, feedback does not militate against the 'drift' effect during the third period even though there is a considerable mean increase in finger temperature during the second within session period.

Graph 2

Graph 2 shows the mean finger temperature changes (30 second epoch) across treatment sessions for each of the treatment conditions.

FINGER TEMPERATURE CHANGES (PER 30 SECOND EPOCH) WITHIN TREATMENT SESSIONS



Stability point finger temperatures

Stability point finger temperature was that temperature attained by each patient at the end of the 15 minute stabilisation and five minute pre-experimental baseline phase, prior to the 15 minute treatment phase. It represented the stable level of responding for the patient within the session. Thus for each patient a stability point temperature was recorded within every session at the start of every treatment phase. As the non-significant treatment effect (Table 1) may have been due to group differences in stability point temperatures, rather than an inability to increase finger temperature during the feedback session, stability point finger temperatures were investigated to determine whether the finger temperature condition showed elevated temperatures in anticipation of feedback.

Table 3 is a summary table of a 2 Way Analysis of Variance, between conditions and across sessions for stability point temperatures. (Appendix 2.3).

Table 3

Stability point temperatures between treatment conditions and within treatment sessions

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	2 Tail Probability
Treatment	103.19	3	34.39	0.72	0.55
Error	1342.05	28	47.93		
Session	78.43	9	8.71	0.96	0.48
Treatment x Session	290.71	27	10.77	1.18	0.25
Error	2294.97	252	9.11		

Two Way Analysis of Variance.

The 2 Way Analysis of Variance (repeated measures) between treatment conditions and across sessions showed no significant condition, sessions or condition x session effects. If a treatment condition effect were apparent, it may have indicated that finger temperature stability point values were different across conditions, suggesting that temperature changes were taking place prior to the onset of feedback. A treatment x sessions effects may have indicated that changes prior to the onset of feedback were taking place across sessions, perhaps as a function of the practice afforded by earlier sessions. As neither a treatment nor a treatment x session effect was observed, it appears that no pre-feedback finger temperature changes occurred in any treatment condition.

TEMPORAL ARTERY PULSE AMPLITUDE INFORMATION

The temporal artery amplitude information was analysed as Relative Difference (RD) change scores. The RD scores enabled relative changes to be measured, independent of absolute values (Bromley 1971). An RD score was calculated for each of the three five-minute within session periods with respect to the pre-experimental baseline. A positive RD score indicated an increase in the treatment period pulse amplitude relative to the pre-experimental baseline. Conversely, a negative RD score indicated a decrease in the treatment period pulse amplitude relative to the pre-experimental baseline. (Appendix 2.4).

Table 1 is a summary table from a 3 Way repeated measures Analysis of Variance examining the effects of treatment conditions, sessions and periods on mean temporal artery pulse amplitude (RD scores).

Table 1

Temporal Artery Pulse Amplitude (relative difference scores) between treatment conditions, within treatment sessions and within treatment session periods

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	2 Tail Probability
Treatment	1.69	3	0.57	1.62	0.21
Error	9.76	28	0.35		
Period	0.11	2	0.06	2.17	0.12
Period x Treatment	0.14	6	0.02	0.87	0.52
Error	1.47	56	0.03		
Session	3.08	9	0.34	6.05	0.00
Session x Treatment	0.93	27	0.03	0.61	0.94
Error	14.24	252	0.06		
Session x Period	1.15	18	0.06	1.03	0.43
Session x Period x Treatment	2.18	54	0.04	0.65	0.98
Error	31.34	504	0.06		

3 Way Analysis of Variance

The results from Table 1 show that there was no treatment condition effect upon changes in mean temporal artery amplitude (RD scores). The temporal artery feedback condition therefore demonstrated no greater change than the other treatment conditions. There was a highly significant across session effect ($F = 6.05$, 9df., $p < 0.01$) which indicated that mean changes in amplitude differed across the treatment sessions. A lack of a significant treatment x session effect would suggest that the changes were not attributable to a particular treatment condition, but were constant across all treatment conditions.

Table 2.

Mean temporal artery amplitude changes within treatment sessions

Finger Temp	Temp Art Amp	Prog Rel'n	Heart Rate
+0.05	+0.12	+0.16	+0.15

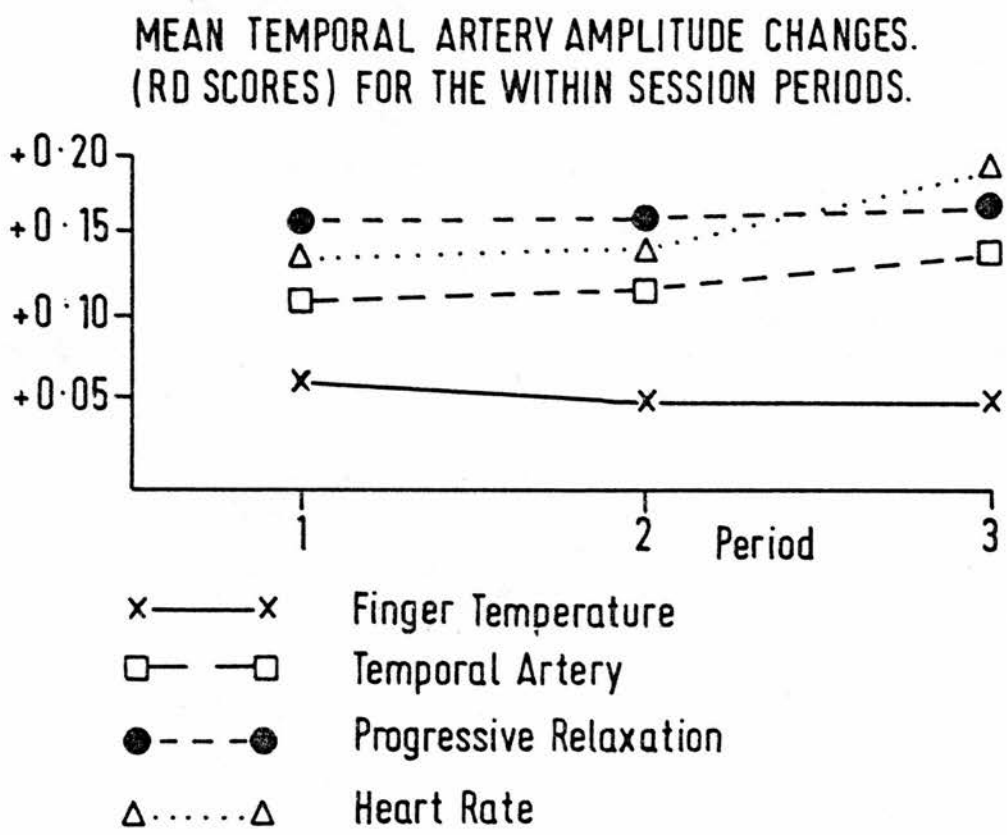
Table 2 shows that all treatment conditions showed slight increases in mean artery amplitude during the treatment phase in comparison to the pre-experimental baseline phase of the sessions. The slight increases in artery amplitude suggest reduced levels of arousal, and the observed increase for the relaxation condition may support this view. Whilst it appears that feedback does not prevent slight temporal artery amplitude increases, it may facilitate a reduction in the amount of vasodilation that would normally occur as a result of relaxation. The change shown by the finger temperature condition cannot however be explained with current information.

In the absence of a treatment effect, it was considered

necessary to confirm the accuracy of the relationship between the pulse wave tracings and the presented feedback information (see page 172).

Graph 1

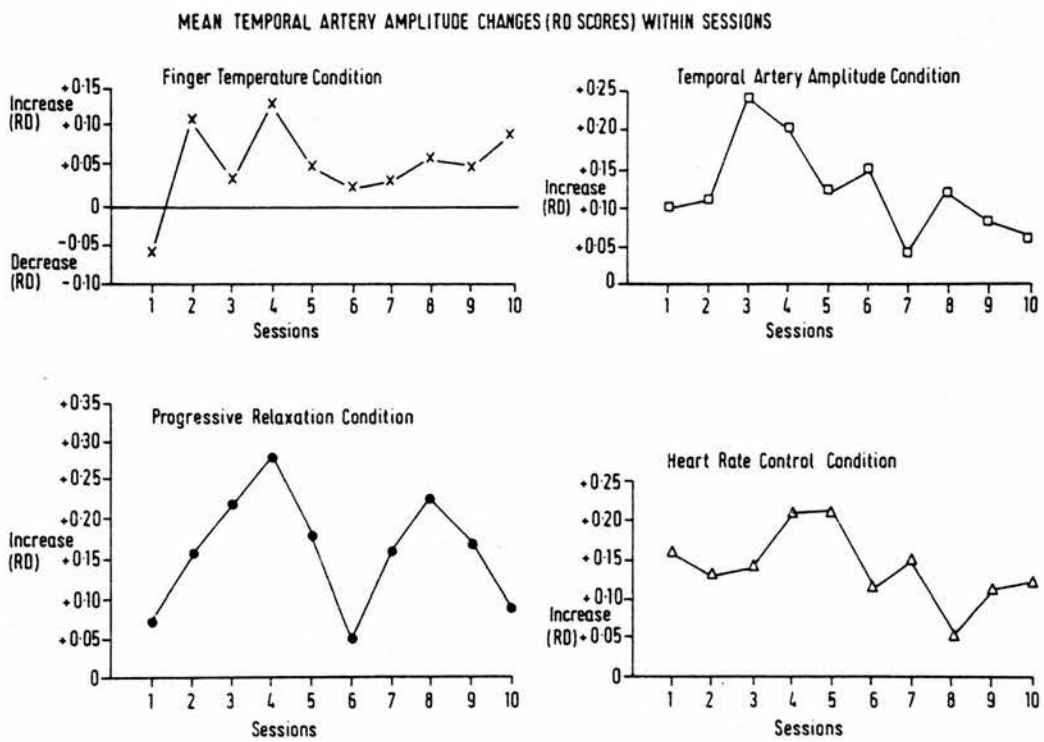
Mean temporal artery pulse amplitude changes (RD scores) for the 3 within session periods



Graph 1 shows the mean period changes across sessions. The finger temperature condition was the only condition to show a slight decrease in mean amplitude, albeit small and non-significant.

Graph 2.

Graph 2 shows the mean changes across sessions for each treatment condition.



TEMPORAL ARTERY AMPLITUDE : ACCURACY OF FEEDBACK

The results show that the temporal artery amplitude condition failed to demonstrate a change in mean amplitude [illegible]. In previous studies binary feedback had been used; in the present study, however, analogue feedback was presented to the patients. Although there is no information on the comparative efficacy of these two approaches, analogue feedback would on a priori grounds be expected to produce more efficient learning (Hume 1977). As there was no evidence to suggest that the presence of feedback afforded greater control of temporal artery activity, an investigation into the relationship between changes in artery amplitude and the feedback display was conducted.

Three subjects - one male and two female - took part in this study. Their ages ranged from 24 - 31 years (mean 26.3 years). All were from University staff and were free of peripheral vascular abnormalities. None of the subjects had been involved in any other part of this study.

Each subject was seated in the experimental room for a period of 45 minutes. The light reflectance plethysmograph was placed on the left temporal artery at the point of maximum beat engorgement (approximately 1 cm above the zygomatic arch). Finger temperature and heart rate were not recorded during the session. The subjects did not receive feedback of temporal artery amplitude (which was displayed on the DVM at six second intervals) but the display was recorded by the experimenter. During the 45 minute experimental period, temporal artery amplitude was recorded on 'low' intensity gain for 15 minutes, 'medium' gain for 15 minutes and 'high' gain for 15 minutes. The

intensity of gain was counterbalanced across the three subjects. Apart from the above modifications for experimental purposes, the presentation and computation of the temporal artery information was the same as in the main treatment study.

The coefficient of correlation was calculated for the two measures (display and permanent record) for each intensity gain sample and across all intensity gain samples. (Appendix 2.7).

The following table shows the coefficients of correlation for each subject on each intensity gain.

Intensity gain	Subject 1 n = 10	Subject 2 n = 10	Subject 3 n = 10
Low	$r=+0.9$ ($p<0.01$)	$r=+0.9$ ($p<0.01$)	$r=+0.8$ ($p<0.01$)
Medium	$r=+0.8$ ($p<0.01$)	$r=+0.9$ ($p<0.01$)	$r=+0.9$ ($p<0.01$)
High	$r=+0.9$ ($p<0.01$)	$r=+0.9$ ($p<0.01$)	$r=+0.9$ ($p<0.01$)

An overall correlation across all subjects and all intensity gains ($n = 90$) was $r = +0.9$ ($p<0.001$).

The results indicate that there is a high correlation between mean temporal artery amplitude (permanent record) and the feedback display. This would show that the feedback was an accurate representation of change and the degree of change that occurred during the treatment session.

The light reflectance plethysmograph is a sensitive transducer and detection can be influenced by movement and muscle tension. For this reason patients in the main study were instructed to remain as still as possible and not to disturb the plethysmograph. Although movement and muscular 'artifacts' may have interfered with the feed-

back display, the length of the 'stabilisation' period and the instructions to remain still would reduce the frequency of artifact interference. The present evidence would indicate that the feedback display was an accurate analogue signal of the mean size of temporal artery amplitude.

The inability of the temporal artery amplitude condition to demonstrate a significant decrease in amplitude may therefore indicate one of two things: firstly, feedback did not enable control of temporal artery amplitude to be gained. Secondly, the amount of change that occurred during the feedback presentation could not be further effected with the aid of feedback. These are however points for further discussion.

TEMPORAL ARTERY PULSE AMPLITUDE (RD of standard deviation scores)

As stated in the introduction, mean and standard deviation changes in temporal artery pulse amplitude were obtained for the pre-experimental baseline and each of the three five-minute within session periods. The mean values were obtained to show changes in the 'central tendency' of the amplitude and, as has been stated, there is a high correlation between the means calculated for analysis and the feedback information presented. Previous research has however implied changes in cerebral blood flow levels from the number of artery pulses which exceeded pretrial criteria. The pretrial criterion was not a fixed level but adjusted at frequent intertrial intervals. (Koppman, McDonald and Kunzel 1974; Feuerstein and Adams 1977 and Christie and Kotses 1973). Frequency counts may not, however, reflect mean amplitude changes, but may reflect a change in the variability (standard deviation) of the pulse amplitudes.

The standard deviation (SD) values were thus obtained to investigate the amount of variance, and changes in variance that occurred in the pulse amplitude during the treatment phase of each session. (Appendix 2.5).

Table 1 is a summary table of the results from a 3 Way repeated measures Analysis of Variance of standard deviation changes (RD scores) in temporal artery pulse amplitude between treatment conditions, across sessions and within periods.

Table 1.

Standard deviation changes in temporal artery pulse amplitude (RD scores) between treatment conditions, across sessions and within periods

Temporal Artery Pulse Amplitude Information. Relative Difference Scores of Standard Deviation Values

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability
Treatment	1.42	3	0.47	1.0	0.41
Error	12.71	27	0.47		
Period	0.62	2	0.31	3.62	0.03
Period x Treatment	0.34	6	0.06	0.67	0.67
Error	4.59	54	0.08		
Session	1.09	9	0.12	0.48	0.89
Session x Treatment	6.37	27	0.24	0.93	0.57
Error	61.55	243	0.25		
Session x Period	0.62	18	0.03	0.46	0.97
Treatment x Session x Period	3.29	54	0.06	0.80	0.84
Error	36.89	486	0.08		

3 Way Analysis of Variance

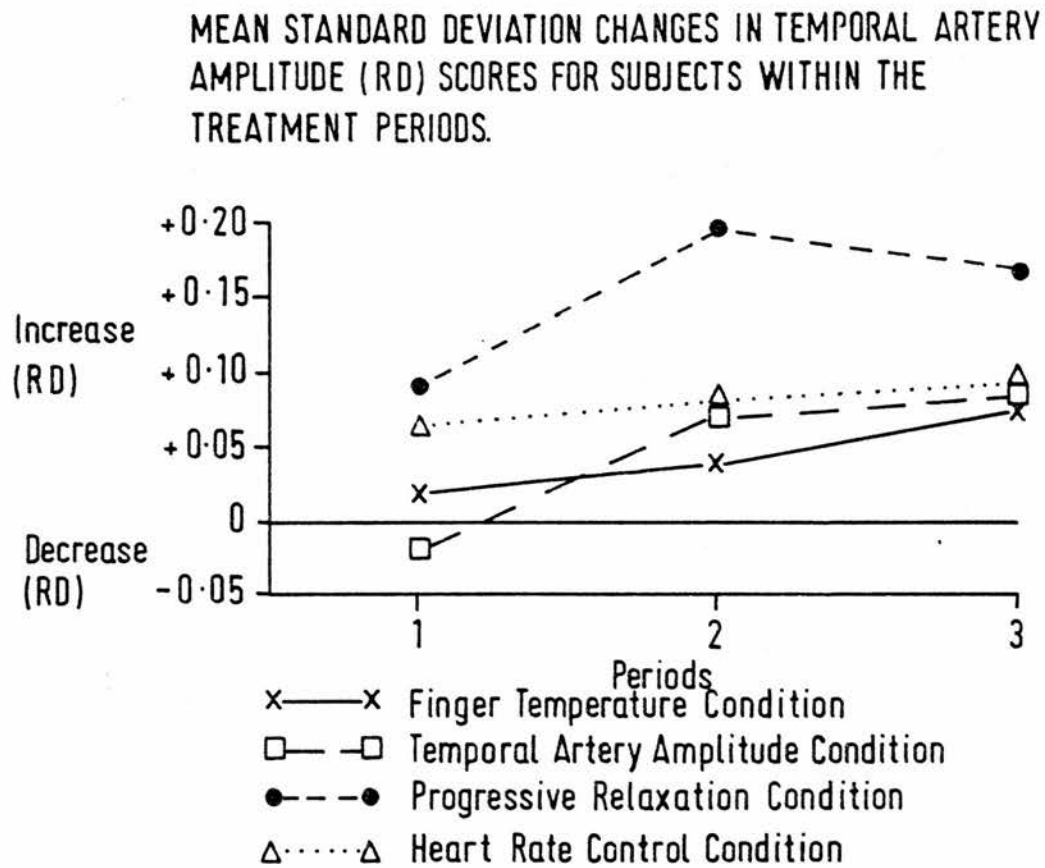
The results from Table 1 showed that as with mean changes in temporal artery amplitude, there were no significant treatment condition effects. The temporal artery amplitude feedback group therefore failed to demonstrate significant changes in artery amplitude variability. There was a significant period effect ($f = 3.63$, 2df., $p<0.03$), but in the absence of a period x treatment or period x session effect, it would appear that the differences

within the periods was constant between all conditions and across all treatment sessions.

Graph 1 shows the standard deviation changes in temporal artery amplitude (RD scores) for all subject within the treatment periods.

Graph 1

Mean standard deviation changes in temporal artery amplitude (RD scores) for subjects within the treatment periods



It is evident that the temporal artery amplitude condition showed the greatest reduction in variability during the first within session period. The reduced variability was not however maintained and slight increases within the second and third periods were noted. As with the mean changes, the relaxation condition showed the greatest increase in variability across the periods.

Table 2.

Mean standard deviation changes in temporal artery amplitude within all session (RD scores)

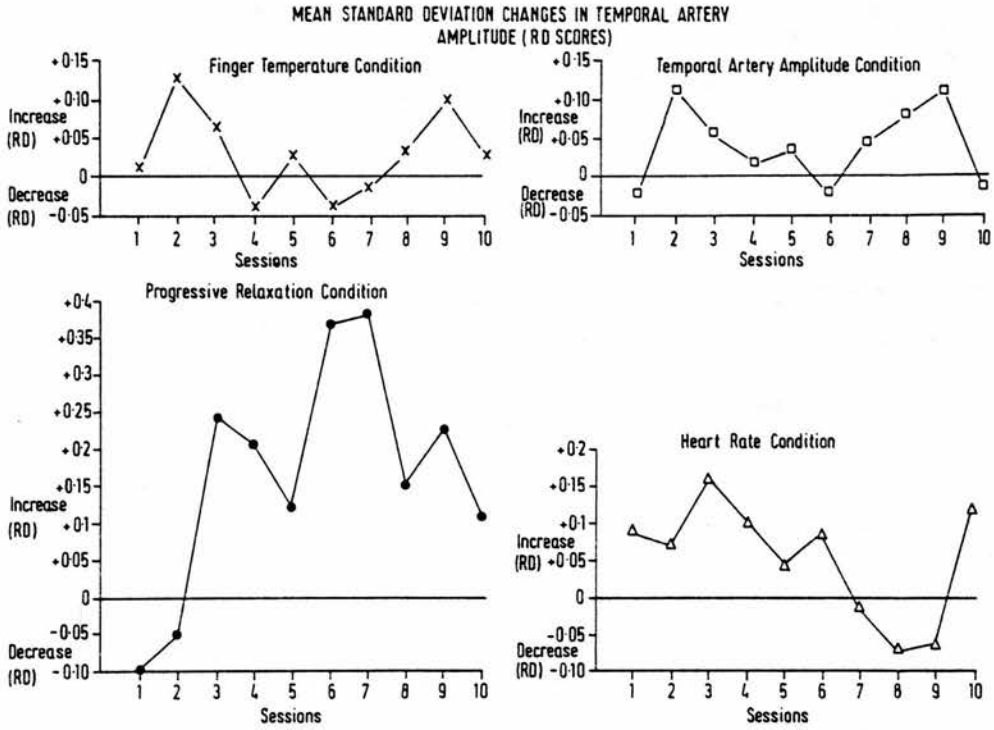
Finger Temp	Temp Art Amp	Prog Rel'n	Heart Rate
+0.04	+0.05	+0.14	+0.08

Although, as Table 2 shows, the mean standard deviation changes in the finger temperature and temporal artery amplitude conditions are very similar, neither group showed a change below the pre-experimental baseline.

Graph 2 shows the mean standard deviation changes (RD scores) in temporal artery amplitude across sessions for each treatment condition. The finger temperature and temporal artery amplitude conditions showed similar changes across the treatment sessions. As with 'mean' changes, the standard deviation changes observed across the treatment sessions are increased for the Progressive Relaxation condition. The increases observed for the relaxation condition may be consistent with a reduction in arousal, although this is a speculative point in the absence of corroborative information.

Graph 2

Mean standard deviation changes in temporal artery amplitude (RD scores)



HEART RATE INFORMATION

Table 1 is a summary table of the 3 Way repeated measures Analysis of Variance of heart rate changes, between treatment conditions, across treatment sessions and within periods. (Appendix 2.6).

Table 1.

Mean heart rate changes across treatment conditions, treatment sessions and within treatment session phases

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability
Treatment	19345.16	3	6448.39	3.10	0.04
Error	58243.29	28	2080.12		
Period	2925.82	2	1462.91	9.58	0.00
Period x Treatment	1248.86	6	208.14	1.36	0.25
Error	8555.59	56	152.78		
Session	4132.22	9	459.14	1.14	0.33
Session x Treatment	19037.45	27	705.09	1.76	0.01
Error	101161.58	252	401.43		
Period x Session	925.54	18	51.42	0.86	0.63
Period x Session x Treatment	3291.17	54	60.95	1.02	0.44
Error	30108.15	504	59.74		

3 Way Analysis of Variance.

The results from Table 1 show that a significant treatment effect occurred ($F = 3.10$, 3 df., $p<0.04$). This indicated significant differences in heart rate changes between the treatment conditions.

As a significant treatment x session effect also occurred ($F = 1.76$, 27 df, $p < 0.01$) the differences in treatment conditions was not constant, but varied over the treatment sessions. There was also a significant period effect ($F = 9.58$, 2 df., $p < 0.01$) showing that changes within the periods also occurred. As there were no period x treatment or period x sessions effects, it can be assumed that the changes within periods were constant between each treatment condition and within each session.

The significant treatment x session effect was investigated further to determine which treatment conditions were responsible for the significant heart rate changes, and within which sessions the changes occurred.

A One Way Analysis of Variance was carried out on each of the ten treatment sessions, investigating the between treatment condition heart rate changes for each session. The ten One Way ANOVA's were calculated to identify the extent of between treatment condition differences for each treatment session. The results of the ten One Way ANOVA's showed that significant treatment effects occurred for session 1 ($F = 4.41$, 3 df, $p < 0.03$) and session 10 ($F = 3.49$, 3 df, $p < 0.03$). There were no significant treatment effects in any of the intervening eight sessions.

Table 2 is a summary table of the results of the One Way ANOVA for between treatment condition heart rate changes.

Table 2.

Mean heart rate changes between treatment conditions on session 1

Source	Degrees of Freedom	Sum of Squares	Mean Squares	F Ratio	F Prob
Between Groups	3	3019.60	1006.53	3.41	0.03
Within Group	28	8260.64	295.02		
Total	31	11280.24			

1 Way Analysis of Variance.

A Posteriori Contrast Test. (least significance difference test. Alpha level 0.05)

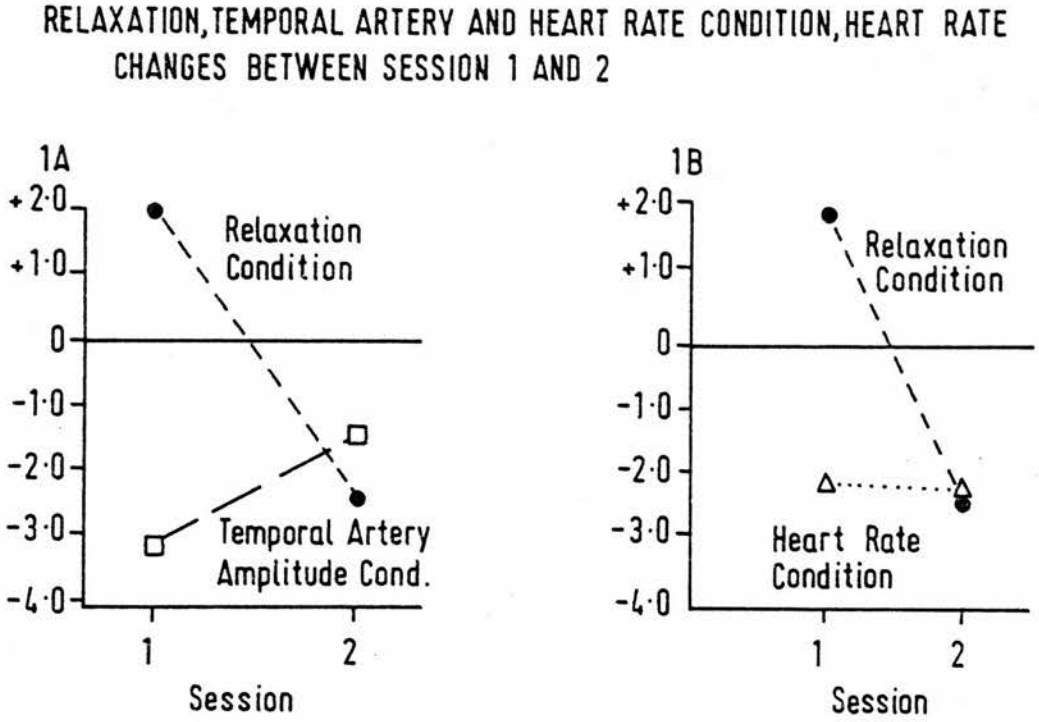
Mean		TAC	HR	FT	REL
-15.49	Temporal artery pulse amplitude condition (TAC)				
-10.41	Heart rate condition (HR)				
1.36	Finger temperature control condition (FT)				
9.97	Progressive relaxation condition (REL)	*	*		

(*) Denotes pairs of groups significantly different at the 0.05 level)

The 'a posteriori contrast test' shows that the changes were due to the mean heart rate values for the temporal artery and heart rate conditions being lower than for the progressive relaxation condition. (See Graphs 1A, 1B.)

Graph 1.

Relaxation, temporal artery and heart rate condition, heart rate changes between session 1 and 2



In the absence of a significant treatment effect (1 WAY ANOVA) for session 2, it is indicated that there was a significant change in heart rate for the Progressive Relaxation condition between sessions 1 and 2.

Table 3 is the summary table of the results of the 1 Way Analysis of Variance between treatment condition heart rate changes on session 10.

Table 3.

Source	Degrees of Freedom	Sum of Squares	Mean Squares	F Ratio	F Prob
Between Groups	3	2091.00	697.00	3.48	0.03
Within Groups	28	5604.32	200.15		
Total	31	7695.33			

1 Way Analysis of Variance.

A Posteriori Contrast Test (least significance difference test, Alpha level 0.05)

Mean		REL	HR	TAC	FT
-24.09	Progressive relaxation condition (REL)				
-13.13	Heart rate condition (HR)				
-09.20	Temporal artery pulse amplitude condition (TAC)			*	
-00.80	Finger temperature control condition (FT)			*	

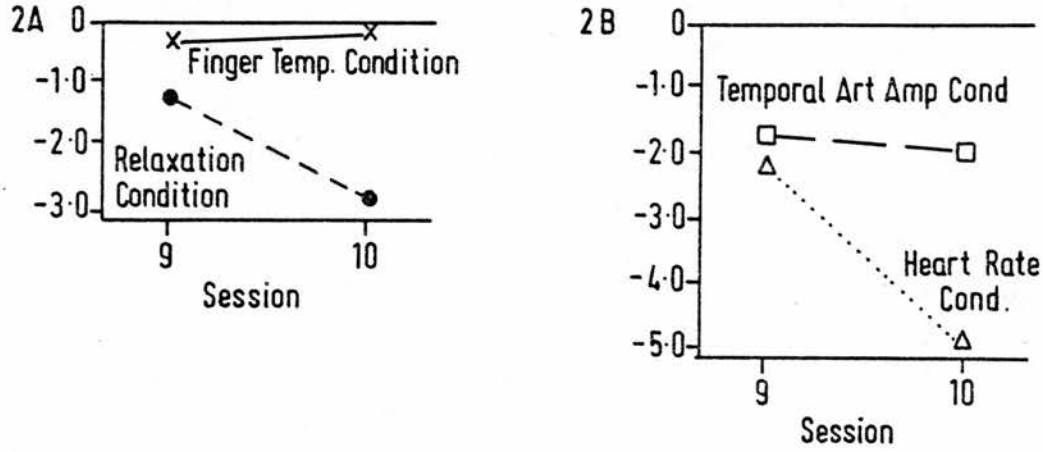
(*) Denotes pairs of groups significantly different at the 0.05 level.

The 'a posteriori contrast test' shows that the differences were due to the mean heart rate values for the progressive relaxation condition being lower than the finger temperature condition and the heart rate condition being lower than the temporal artery amplitude condition. (See Graphs 2A, 2B.)

Graphs 2 (A & B)

Heart rate changes for the finger temperature, temporal artery, heart rate and relaxation conditions between sessions 9 and 10.

HEART RATE CHANGES FOR THE FINGER TEMPERATURE, TEMPORAL ARTERY, HEART RATE AND RELAXATION CONDITIONS BETWEEN SESSIONS 9 AND 10



In the absence of any other significant condition effects within sessions 2 through to 9, it can be assumed that the treatment x session effects took place on session 1 and 10. The significant changes in heart rate were due to the progressive relaxation and heart rate conditions. In session 1, heart rate for the relaxation condition was significantly higher than for the other conditions; it however decreased on session 2. During session 10, significant decreases in heart rate were shown by the progressive relaxation and heart rate conditions.

Table 4.

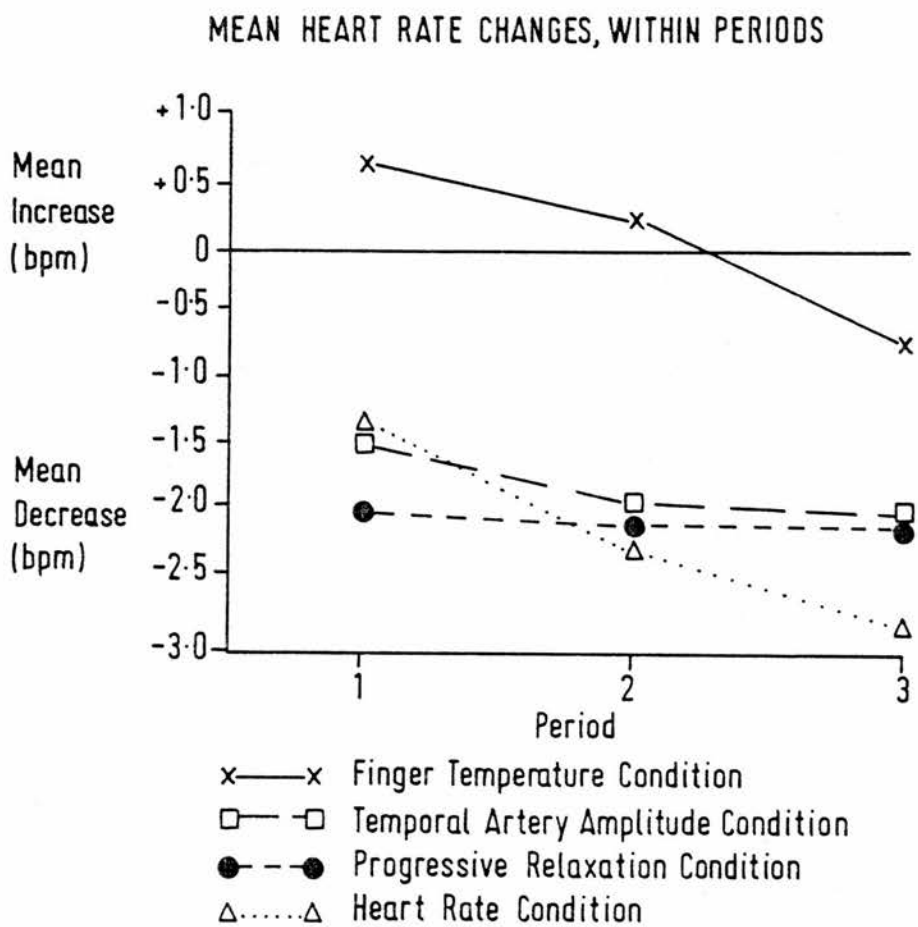
Mean heart rate changes within treatment sessions (bpm)

Finger Temp	Temp Art	Prog Rel'n	Heart Rate
+0.09	-1.67	-2.01	-2.10

Table 4 indicates that sessions 1 and 10 reflect the general superiority of progressive relaxation and heart rate biofeedback in effecting decreases in heart rate. The non-significant treatment effect suggested that feedback did not produce greater changes than were achieved by the Progressive Relaxation condition. There was therefore no incremental utility in feedback over relaxation in producing heart rate decreases.

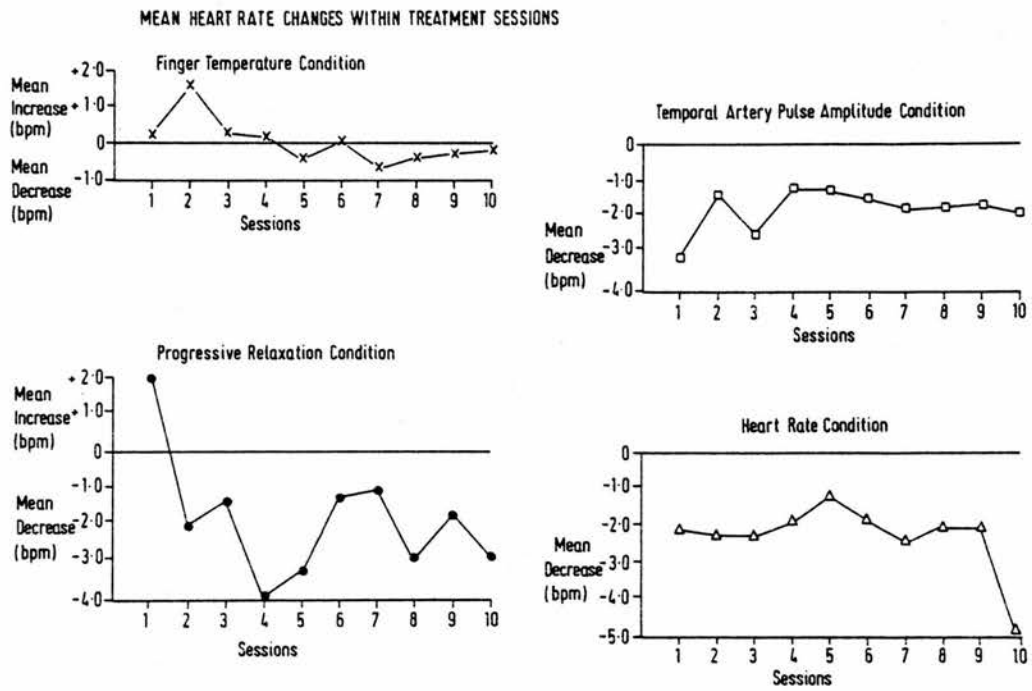
Graph 3 shows the mean changes in heart rate within session periods. A slight decrease in heart rate is noted for all conditions within periods. However the greatest decrease was achieved by the heart rate condition, albeit non-significant.

Graph 3.



Graph 4.

Graph 4 shows the mean changes in heart rate (bpm) across sessions for each treatment condition.



RESULTS OF THERAPEUTIC CHANGE

Data Reduction

Seven indices of therapeutic change were recorded over the three month baseline period, three month treatment and six month follow-up periods. The indices were: frequency of headaches per month, maximum intensity of each headache, the duration of the headache, an index of headache discomfort (frequency x intensity x duration), an analgesic index per month, frequency of use of vaso-constrictor drugs per month and the number of prophylactic drugs taken per month. All raw, mean and standard deviation scores are contained in Appendix 3.

Initial observations of 'headache frequency' during the three month baseline period showed that a reduction occurred; across all treatment conditions, during the second month (Appendix 3.1). This reduction was preceded by the patient's first visit to the Department (for the first acclimatisation session) during which no treatment was administered and was followed by an increase in 'headache frequency' during the third month. A Two Way Analysis of Variance between the four treatment conditions and within the three baseline months revealed a significant difference in frequency within the baseline months ($F = 4.64$, 2 df, $p < 0.01$).

Table 1.

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	2 Tail Probability
Treatment	193.86	3	64.62	3.07	0.04
Error	576.54	27	21.02		
Month	82.54	2	41.27	4.64	0.01
Month x Treatment	76.04	6	12.67	1.42	0.22
Error	480.43	54	8.89		

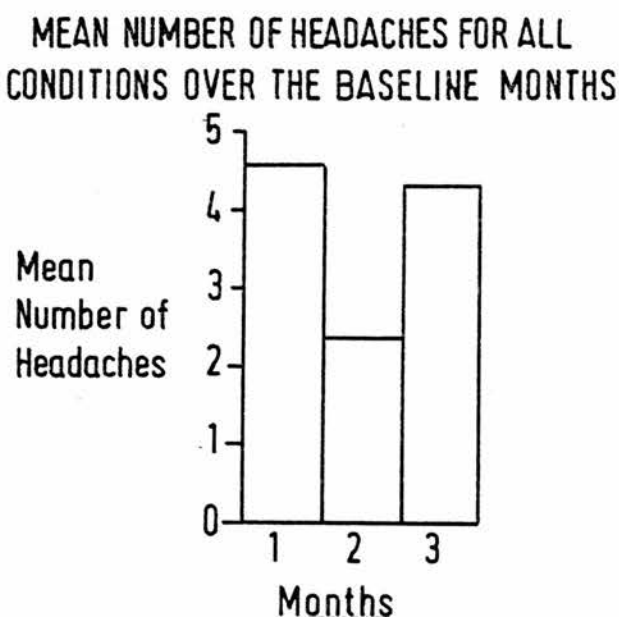
A 'treatment' condition effect was also observed ($F = 3.07$, 3 df, $p < 0.04$). In the absence of a treatment x month interaction, the differences in headache frequency during each 'baseline month' were assumed to remain constant. Therefore within month changes are constant for each treatment condition.

Table 2.

Finger Temp	Temp Art	Prog Rel'n	Heart Rate
99	136	34	114

Graph 1 shows the mean frequency of headaches over each baseline month for patients in all treatment conditions.

Graph 1.



It was hypothesised that the decrease in headache frequency observed during the second month of the baseline period was due to non-specific treatment effects of attending for the first 'acclimatisation' session. It was therefore concluded that month three would be representative of a stable level of headache activity; the uncontrolled effects of the acclimatisation sessions, and of self monitoring having the least influence at the end of the baseline period. Month three was therefore used as the baseline month to evaluate the clinical change during the following months.

As the initial baseline levels of responding differed between the treatment conditions for frequency of headaches ($F = 3.07$, 3 df, $p < 0.04$), relative difference (RD) scores were calculated for each of the seven indices of change. The relative difference scores indicated relative changes over the follow-up months with respect to baseline, the scores being independent of between treatment condition differences in baseline values.

In order to assess changes in headache activity for each treatment condition and the waiting list control condition, the following approach was adopted:

Indices of change for month three (end of the baseline period) were compared with the indices recorded from month seven (immediately on completion of treatment), month 10 (four months after the completion of treatment) and month 12 (six months after the completion of treatment). Relative Difference scores were therefore obtained for each of the follow up months to assess the immediate effects of treatment, the effects after four months and after six months. A positive relative difference value indicated an improvement whilst

a negative relative difference value indicated a worsening of the symptom with respect to baseline. The amount of the value related to the magnitude of change.

Since measures of therapeutic change were calculated for three follow-up months relative to baseline values, change was represented as five between (treatment conditions) x three within (months) design with repeated measures on the within (months) variable.

Statistical analysis of clinical information was undertaken for the 29 patients who had completed the ten treatment sessions and had notified the experimenter of headache activity on every month of the 12 month duration of the study. Of the 29 patients, eight were in the finger temperature condition, eight were in the temporal artery amplitude condition, five were in the progressive relaxation condition, five were in the heart rate condition and three were in the waiting list condition.

FREQUENCY OF HEADACHES

For the purposes of statistical analysis and for the graphical presentation of the results, 'MONTH 1' is the relative difference value for the changes recorded on month 7 (immediately after treatment) compared with the baseline. 'MONTH 2' is the relative difference value for the changes recorded on month 10 (four months after the completion of treatment) compared with the baseline and 'MONTH 3', the relative difference values recorded on month 12 (at the end of the study).

Table 1.

Table 1 is a summary table of the results of a 2 Way repeated measures ANOVA frequency between treatment conditions and within the three follow-up months. (Appendix 3.2).

Frequency of headaches between treatment conditions within the follow-up months

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability
Treatment	45.13	4	11.28	6.47	0.00
Error	41.86	24	1.74		
Month	1.30	2	0.65	0.79	0.46
Month x Treatment	2.52	8	0.31	0.38	0.93
Error	39.67	48	0.83		

2 Way Analysis of Variance

There was a significant difference between the treatment conditions. As there was no difference between the follow-up months

and there was no month x treatment interaction, it would appear that the difference in treatment conditions was constant over the follow-up months.

In order to establish which treatment conditions differed over the follow-up period, One Way Analyses of Variance were computed between each of the five conditions for each of the three follow-up months.

Graph 1.

Graph 1 represents the mean relative difference values in frequency of headaches at month 1 (end of treatment period with respect to baseline).

FREQUENCY OF HEADACHES. MONTH 1. BETWEEN TREATMENT CONDITIONS

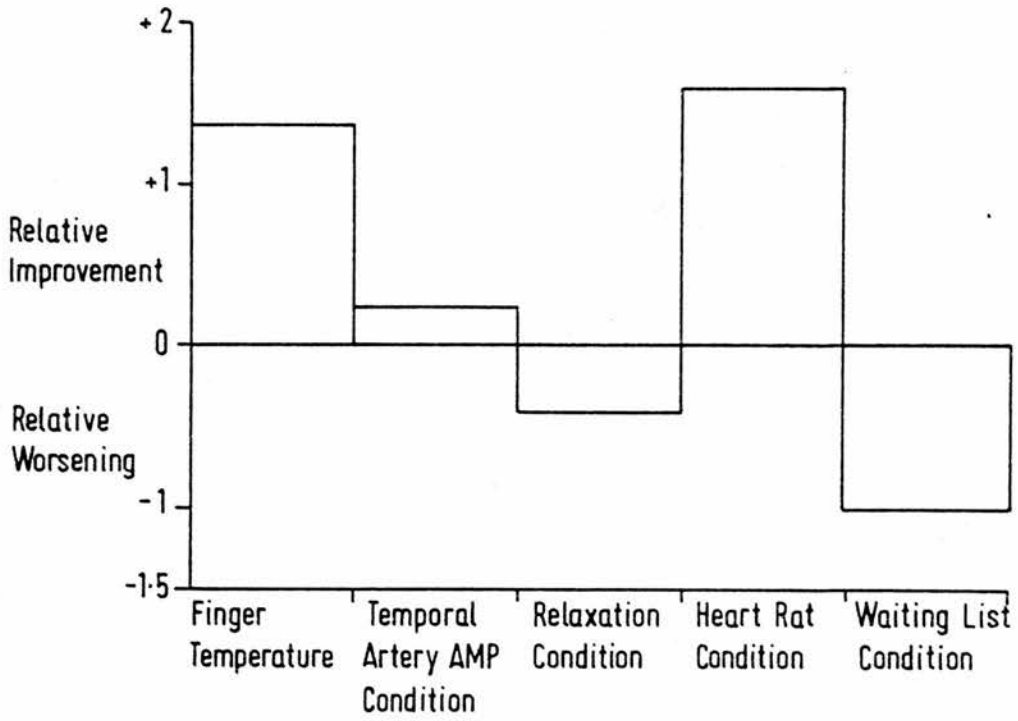


Table 2.

Table 2 is the summary table for the One Way Analysis of Variance between the treatment conditions at month 1.

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability
Equality of cell means	23.39	4	5.84	7.0991	0.01
Error	19.77	24	0.82		

A significant difference between the treatment conditions occurred during month 1. Table 3 is a table of probabilities for the T values between the treatment conditions on month 1.

Table 3.

Condition	FT	TA	RL	HR	WL
Finger temperature (FT)	1.00				
Temporal artery (TA)	0.02	1.00			
Relaxation (RL)	0.00	0.22	1.00		
Heart rate (HR)	0.67	0.02	0.00	1.00	
Waiting list (WL)	0.00	0.05	0.37	0.00	1.00

Table 3 shows that the significant differences in headache frequency found at month 1 were due to the finger temperature and heart rate treatment conditions showing significantly greater improvement over the relaxation and temporal artery amplitude condition and the waiting list condition. However there was no difference in the improvement (reduction in headache frequency) between the heart rate and the finger temperature treatment conditions.

Graph 2.

Graph 2 represents the mean relative difference values in frequency of headaches at month 2 (four months after the end of treatment with respect to baseline).

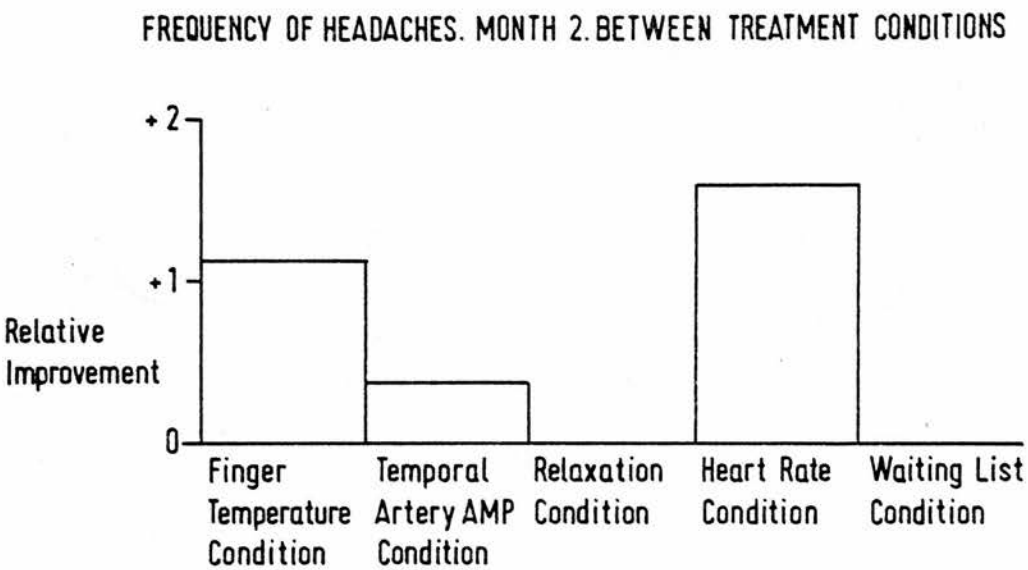


Table 4

Table 4 is the summary table for the One Way Analysis of Variance between the treatment conditions at month 2. No significant difference between the treatment conditions was found.

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability
Equality of cell means	10.26	4	2.56	1.71	0.18
Error	35.95	24	1.49		

Graph 3.

Graph 3 represents the mean relative difference values in frequency of headaches at month 3 (six months after the end of treatment with respect to baseline).

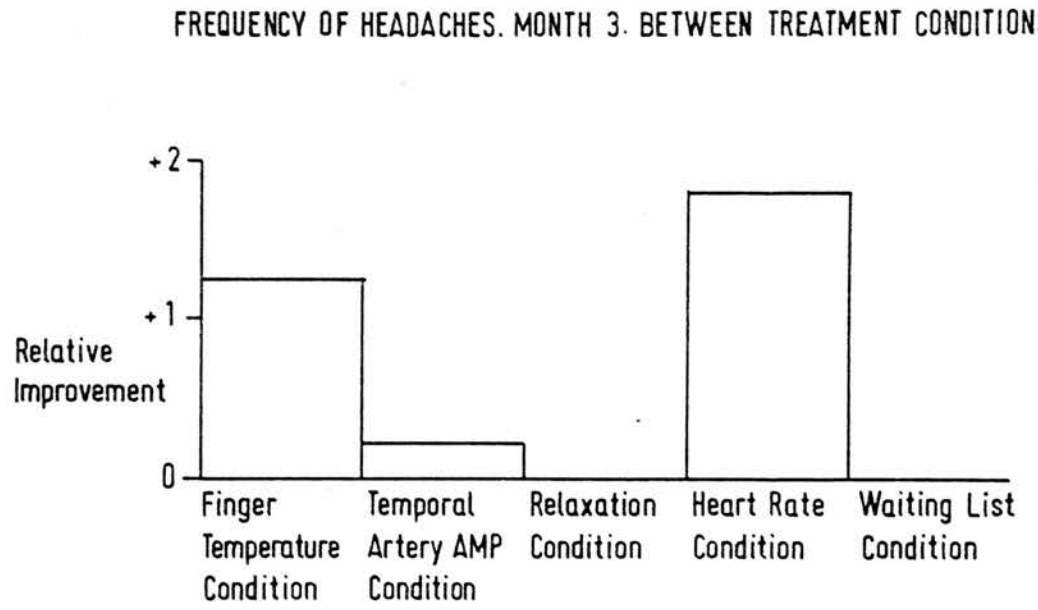


Table 5

Table 5 is the summary table for the One Way Analysis of Variance between the treatment conditions at month 3.

Source	Sum of Squares	Degrees of Freedom	Mean Square	F Value	Tail Probability
Equality of cell means	13.99	4	3.49	3.25	0.03
Error	25.80	24	1.08		

A significant difference between the treatment conditions occurred during month 3. Table 6 is a table of probabilities for the T values between the treatment conditions on month 3.

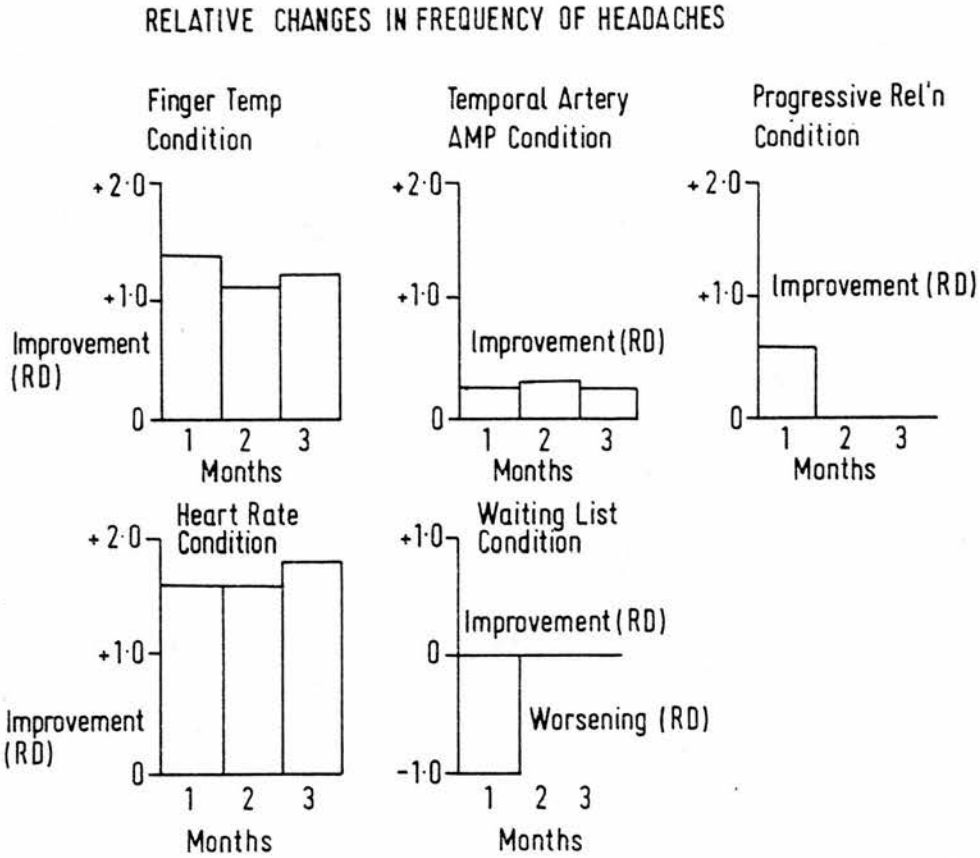
Table 6

Conditions	FT	TA	RL	HR	WL
Finger temperature (FT)	1.00				
Temporal artery (TA)	0.07	1.00			
Relaxation (RL)	0.05	0.68	1.00		
Heart rate (HR)	0.36	0.01	0.01	1.00	
Waiting list (WL)	0.09	0.72	1.00	0.03	1.00

Table 6 shows that the significant differences in headache frequency found at month 3 were due to the heart rate condition showing significantly greater improvement over the temporal artery amplitude, the relaxation and the waiting list conditions. Although the heart rate condition showed a greater improvement than the finger temperature control condition, the difference does not reach significance.

Graph 4.

Graph 4 shows the mean relative difference values for frequency of headaches over the three follow-up months for the finger temperature, temporal artery amplitude, relaxation, heart rate and waiting list conditions respectively.



INTENSITY OF HEADACHES

Table 1.

Table 1 is the summary table of a 2 Way repeated measures ANOVA between treatment conditions and within the three follow-up months. (Appendix 3.3).

Intensity of headaches between treatment conditions, within the follow-up months.

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability
Treatment	37.53	4	9.38	5.86	0.00
Error	38.42	24	1.60		
Month	0.58	2	0.29	0.45	0.64
Month x treatment	3.55	8	0.44	0.69	0.69
Error	30.84	48	0.64		

2 Way Analysis of Variance.

There was a significant difference between the treatment conditions. As there was no difference between the follow-up months and no month x treatment interaction, it would appear that the difference in treatment conditions was constant over the follow-up months.

In order to establish which treatment conditions differed over the follow-up period, One Way Analyses of Variance were computed between each of the five conditions for each of the three follow-up months.

Graph 1.

Graph 1 represents the mean relative difference values in the intensity of headaches at month 1 (end of the treatment period with respect to baseline).

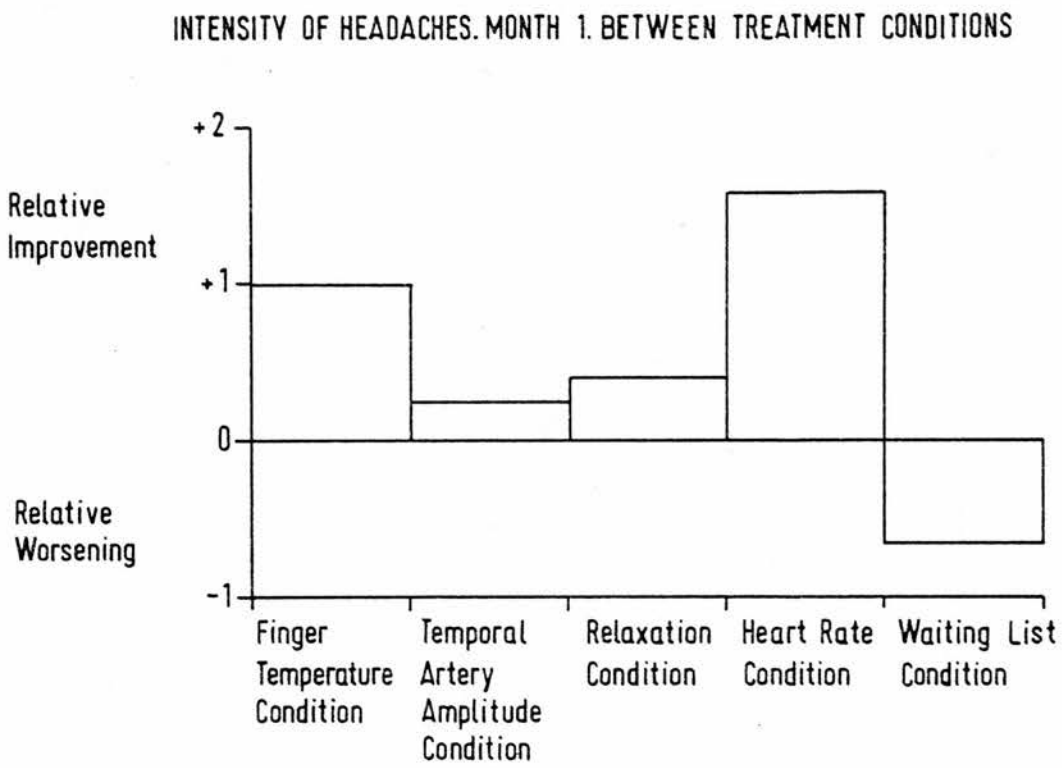


Table 2

Table 2 is the summary table for the One Way Analysis of Variance between the treatment conditions during month 1.

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability
Equality of cell means	12.26	4	3.07	3.58	0.02
Error	20.57	24	0.86		

A significant difference between the treatment conditions occurred during month 1. Table 3 is a table of probabilities for the T values between the treatment conditions on month 1.

Table 3.

Condition	FT	TA	RL	HR	WL
Finger Temperature (FT)	1.00				
Temporal artery (TA)	0.12	1.00			
Relaxation (RL)	0.27	0.78	1.00		
Heart rate (HR)	0.27	0.02	0.05	1.00	
Waiting list (WL)	0.01	0.16	0.13	0.00	1.00

Table 3 shows that the significant differences in headache intensity found at month 1 were due to the heart rate treatment condition showing significantly greater improvement over the temporal artery amplitude and waiting list conditions. The heart rate condition however does not differ significantly from the finger temperature condition and just fails to reach statistical significance over the relaxation condition.

Graph 2.

Graph 2 represents the mean relative difference values in intensity of headaches at month 2 (four months after the end of treatment with respect to baseline).

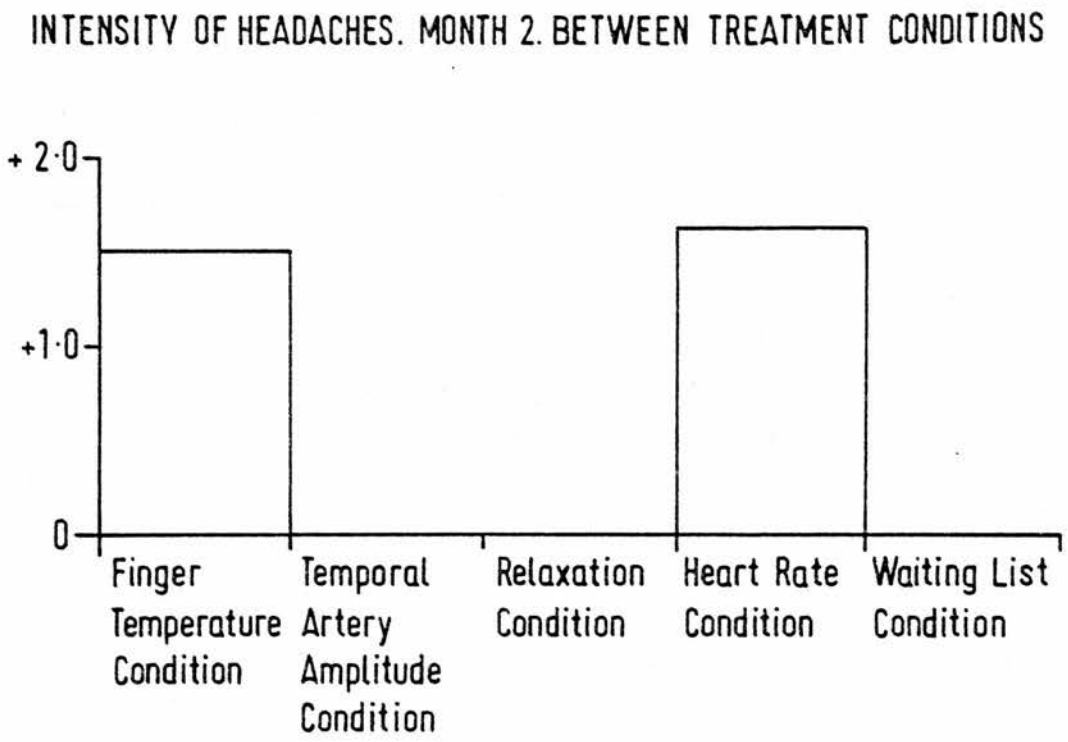


Table 4.

Table 4 is the summary table for the One Way Analysis of Variance between the treatment conditions at month 2.

Source	Sum of Squares	Degrees of Freedom	Mean Square	F Value	Tail Probability
Equality of cell means	17.01	4	4.25	4.05	0.01
Error	25.20	24	1.05		

One Way Analysis of Variance.

Table 5.

Table 5 is the table of probabilities for the T values between the treatment conditions on month 2.

Condition	FT	TA	RL	HR	WL
Finger temperature (FT)	1.00				
Temporal artery (TA)	0.01	1.00			
Relaxation (RL)	0.02	1.00	1.00		
Heart rate (HR)	0.87	0.01	0.02	1.00	
Waiting list (WL)	0.04	1.00	1.00	0.04	1.00

Table 5 shows that the significant differences in headache intensity found at month 2 were due to the finger temperature and heart rate treatment conditions showing significantly greater improvement over the relaxation, temporal artery amplitude and waiting list condition. However, there was no difference in improvement (reduction in headache intensity) between the heart rate and the finger temperature treatment conditions.

Graph 3.

Graph 3 represents the mean relative difference values in intensity of headaches at month 3 (six months after the end of treatment with respect to baseline).

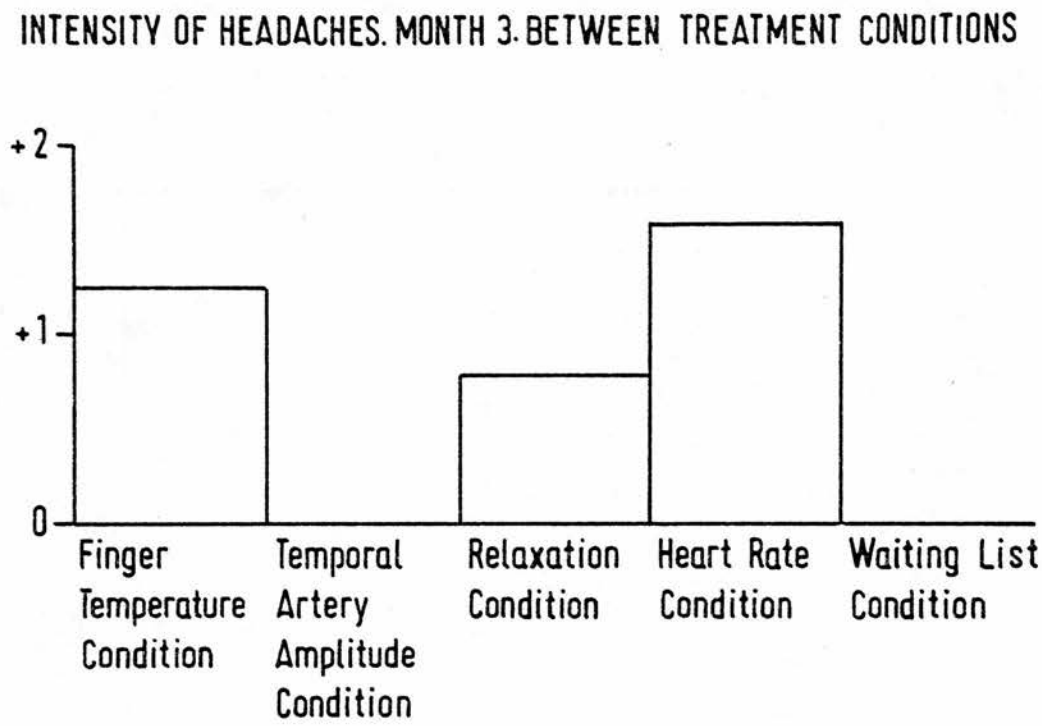


Table 6.

Table 6 is the summary table for the One Way Analysis of Variance between the treatment conditions at month 3.

Source	Sum of Squares	Degrees of Freedom	Mean Square	F Value	Tail Probability
Equality of cell means	11.81	4	2.95	3.02	0.04
Error	23.50	24	0.98		

One Way Analysis of Variance.

A significant difference between the treatment conditions occurred during month 3. Table 7 is a table of probabilities for the T values between the treatment conditions on month 3.

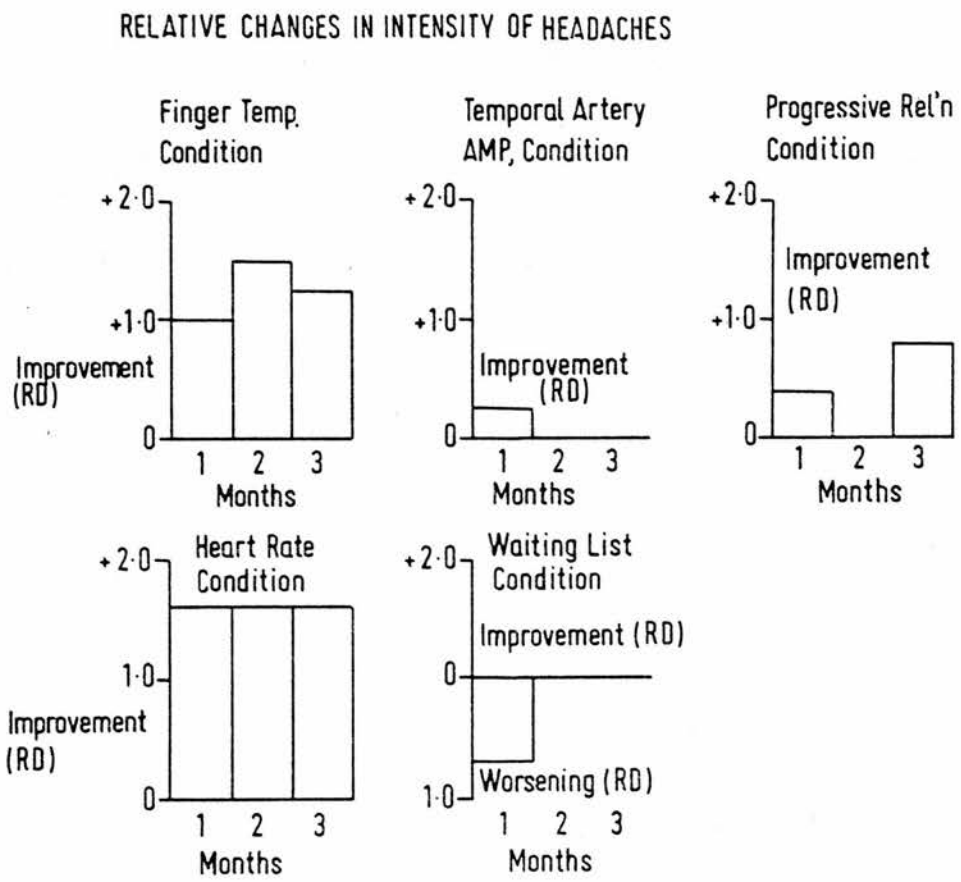
Table 7.

Condition	FT	TA	RL	HR	WL
Finger temperature (FT)	1.00				
Temporal artery (TA)	0.02	1.00			
Relaxation (RL)	0.43	0.17	1.00		
Heart rate (HR)	0.54	0.01	0.21	1.00	
Waiting list (WL)	0.07	1.00	0.28	0.04	1.00

Table 7 shows that the significant differences in headache intensity found at month 3 were due to the heart rate condition showing significantly greater improvement over the temporal artery amplitude and waiting list conditions. However, the improvement found in the heart rate condition was not significantly greater than found in the finger temperature and relaxation conditions.

Graph 4.

Graph 4 shows the mean relative difference values for intensity of headaches over the three follow-up months for the finger temperature, temporal artery amplitude, relaxation, heart rate and waiting list conditions respectively.



DURATION OF HEADACHES

Table 1.

Table 1 is the summary table of the 2 Way repeated measures ANOVA between treatment conditions and within the three follow-up months. (Appendix 3.4).

Duration of headaches between treatment conditions, within the follow-up months

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability
Treatment	38.61	4	4.65	7.08	0.00
Error	32.72	24	1.36		
Month	0.31	2	0.16	0.23	0.79
Month x treatment	0.97	8	0.12	0.18	0.99
Error	32.66	48	0.68		

2 Way Analysis of Variance.

There was a significant difference between the treatment conditions. As there was no difference between the follow-up months and no month x treatment interaction, it would appear that the difference in treatment conditions was constant over the follow-up months. In order to establish which treatment conditions differed over the follow-up period, One Way Analyses of Variance were computed between each of the five conditions for each of the three follow-up months.

Graph 1.

DURATION OF HEADACHES. MONTH 1. BETWEEN TREATMENT CONDITIONS

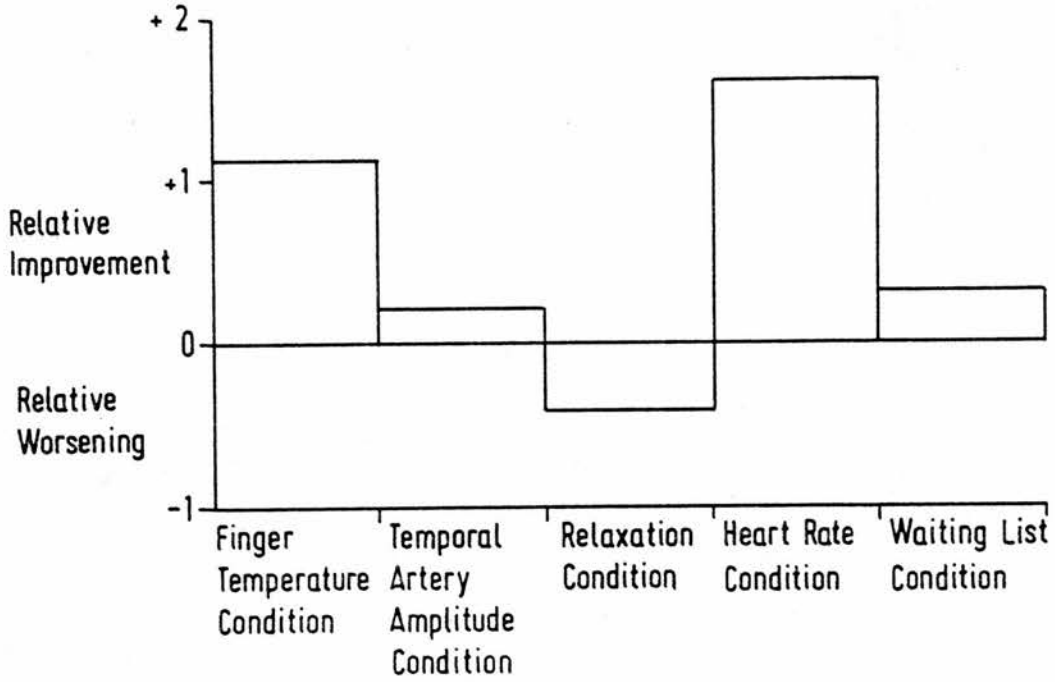


Table 2.

Table 2 is the summary table for the One Way Analysis of Variance between the treatment conditions during month 1.

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability
Equality of cell means	13.39	4	3.35	4.60	0.01
Error	17.44	24	0.73		

A significant difference between the treatment conditions occurred during month 1. Table 3 is a table of probabilities for the T values between the treatment conditions on month 1.

Table 3.

Condition	FT	TA	RL	HR	WL
Finger temperature (FT)	1.00				
Temporal artery (TA)	0.05	1.00			
Relaxation (RL)	0.00	0.19	1.00		
Heart rate (HR)	0.34	0.01	0.00	1.00	
Waiting list (WL)	0.18	0.89	0.25	0.05	1.00

Table 3 shows that the significant differences in the headache duration found at month 1 were due to the heart rate condition showing significantly greater improvement in headache duration than the temporal artery amplitude and relaxation conditions. However, no significant differences were found between the heart rate, the finger temperature and the waiting list conditions.

Graph 2.

Graph 2 represents the mean relative difference values in duration of headaches at month 2 (four months after the end of treatment with respect to baseline).

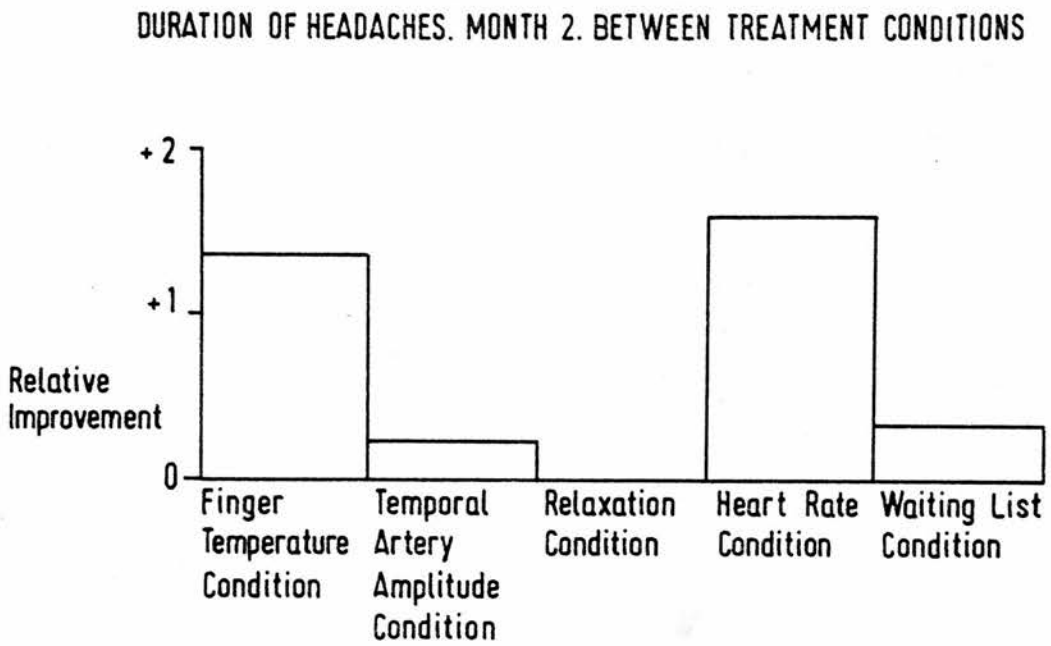


Table 4.

Table 4 is summary table for the One Way Analysis of Variance between the treatment conditions at month 2.

Source	Sum of Squares	Degrees of Freedom	Mean Square	F Value	Tail Probability
Equality of cell means	12.07	4	3.02	3.41	0.02
Error	21.24	24	0.89		

One Way Analysis of Variance.

Table 5.

Condition	FT	TA	RL	HR	WL
Finger temperature (FT)	1.00				
Temporal artery (TA)	0.03	1.00			
Relaxation (RL)	0.02	0.65	1.00		
Heart rate (HR)	0.68	0.02	0.01	1.00	
Waiting list (WL)	0.12	0.89	0.63	0.08	1.00

Table 5 shows that the significant differences in headache duration found at month 2 were due to the heart rate and finger temperature conditions showing significantly greater improvement over the temporal artery amplitude and relaxation conditions. However, there was no difference in improvement (reduction in headache duration) between the heart rate, finger temperature and waiting list conditions.

Graph 3.

Graph 3 represents the mean relative difference values in duration of headaches at month 3 (six months after the end of the treatment with respect to baseline).

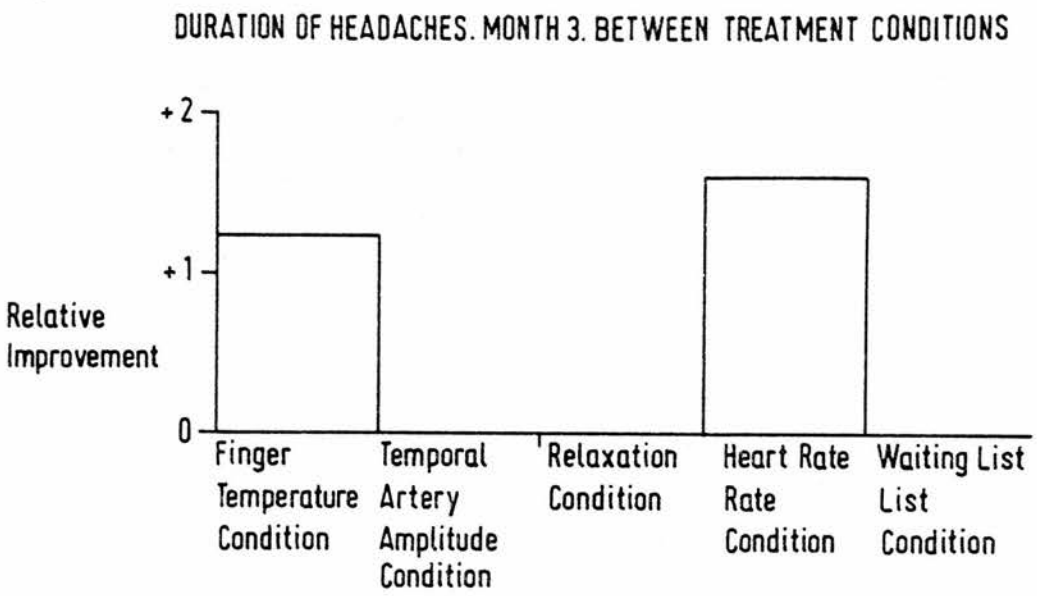


Table 6.

Table 6 is the summary table for the One Way Analysis of Variance between the treatment conditions at month 3.

Source	Sum of Squares	Degrees of Freedom	Mean Square	F Value	Tail Probability
Equality of cell means	14.13	4	3.53	3.17	0.03
Error	26.70	24	1.11		

One Way Analysis of Variance.

A significant difference between the treatment conditions occurred during month 3. Table 7 is a table of probabilities for the T values between the treatment conditions on month 3.

Table 7.

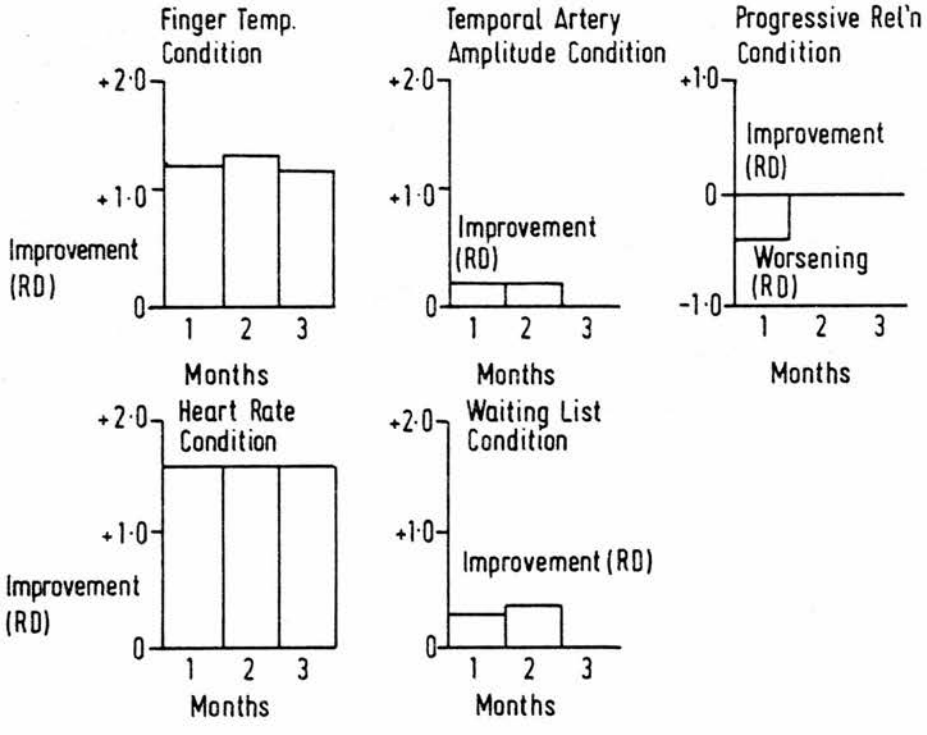
Condition	FT	TA	RL	HR	WL
Finger Temperature (FT)	1.00				
Temporal artery (TA)	0.03	1.00			
Relaxation (RL)	0.05	1.00	1.00		
Heart rate (HR)	0.57	0.01	0.02	1.00	
Waiting List (WL)	0.09	1.00	1.00	0.05	1.00

Table 7 shows that the significant differences in headache duration found at month 3 were due to the heart rate condition showing significantly greater improvement over the temporal artery amplitude, the relaxation and the waiting list conditions. The improvement found in the heart rate condition was not significantly greater than found in the finger temperature condition.

Graph 4.

Graph 4 shows the mean relative difference values for duration of headaches over the three follow-up months for the finger temperature, temporal artery amplitude, relaxation, heart rate and waiting list conditions respectively.

MEAN CHANGES IN DURATION OF HEADACHES



INDEX OF HEADACHE ACTIVITY

Table 1.

Table 1 is a summary table of the results of a 2 Way repeated measures ANOVA of relative changes in the 'index of headache activity' between treatment conditions and within the three follow-up months. (Appendix 3.5).

Index of headache activity between treatment conditions, within the follow-up months

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability
Treatment	37.92	4	9.48	5.53	0.00
Error	41.18	24	1.72		
Month	3.08	2	1.54	2.16	0.13
Month x treatment	3.22	8	0.40	0.56	0.80
Error	34.29	48	0.71		

Two Way Analysis of Variance.

There was a significant difference between the treatment conditions. As there was no difference between the follow-up months and no month x treatment interaction, it would appear that the difference in treatment conditions was constant over the follow-up months. In order to establish which treatment conditions differed over the follow-up period, One Way Analyses of Variance were computed between each of the five conditions for each of the three follow-up months.

Graph 1.

Graph 1 represents the mean relative difference values in 'index of headache activity' values at month 1 (the end of the treatment period with respect to baseline).

INDEX OF HEADACHE ACTIVITY. MONTH 1. BETWEEN TREATMENT CONDITIONS

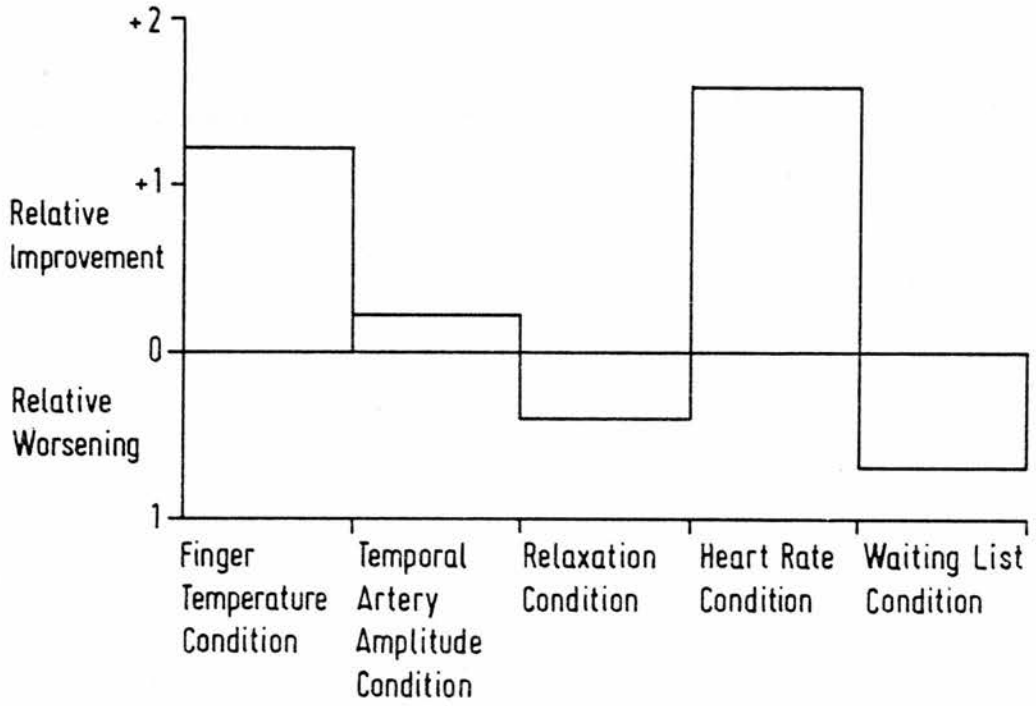


Table 2.

Table 2 is the summary table for the One Way Analysis of Variance between the treatment conditions during month 1.

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability
Equality of cell means	19.11	4	4.78	5.71	0.00
Error	20.07	24	0.84		

A significant difference between the treatment conditions occurred during month 1. Table 3 is a table of probabilities for the T values between the treatment conditions on month 1.

Table 3.

Condition	FT	TA	RL	HR	WL
Finger temperature (FT)	1.00				
Temporal artery (TA)	0.04	1.00			
Relaxation (RL)	0.00	0.22	1.00		
Heart rate (HR)	0.51	0.02	0.00	1.00	
Waiting list (WL)	0.00	0.15	0.69	0.00	1.00

Table 3 shows that the significant differences in the 'index of headache activity' values found at month 1 were due to the finger temperature and heart rate conditions showing significantly greater improvement than the temporal artery amplitude, the relaxation and the waiting list conditions. There was however no significant difference found between the heart rate and finger temperature conditions.

Graph 2.

Graph 2 represents the mean relative difference values in 'index of headache activity' values at month 2 (four months after the end of treatment with respect to baseline).

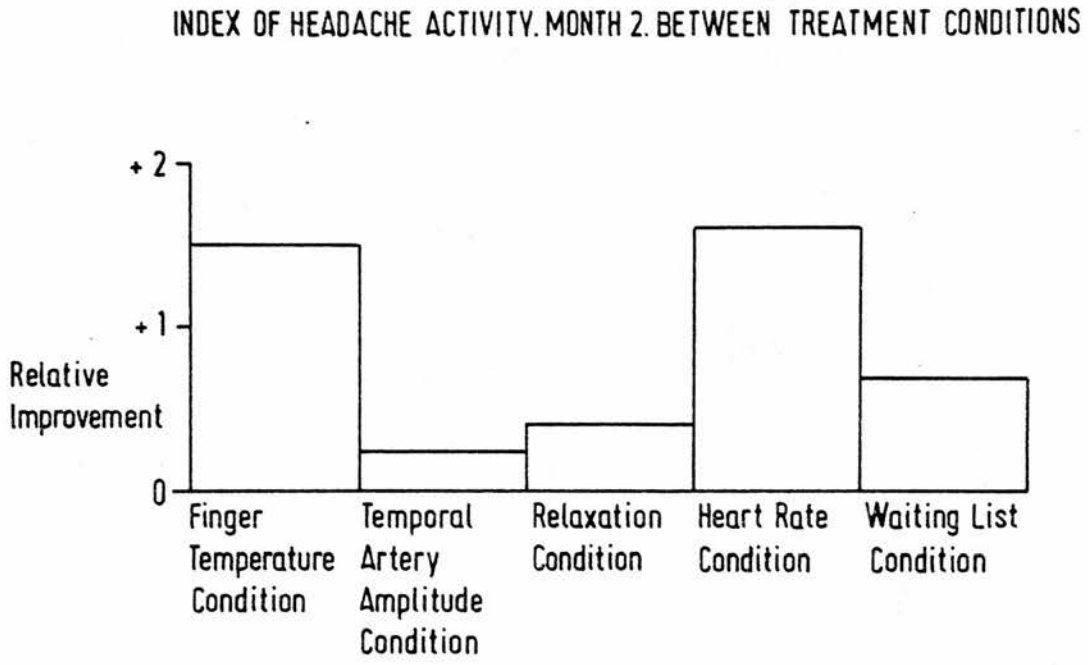


Table 4.

Table 4 is the summary table for the One Way Analysis of Variance between the treatment conditions at month 2. No significant differences were found to exist between the treatment conditions.

Source	Sum of Squares	Degrees of Freedom	Mean Square	F value	Tail Probability
Equality of cell means	10.12	4	2.53	2.13	0.11
Error	28.57	24	1.19		

One Way Analysis of Variance.

Graph 3.

Graph 3 represents the mean relative difference values in 'index of headache activity' values at month 3 (six months after the end of the treatment with respect to baseline).

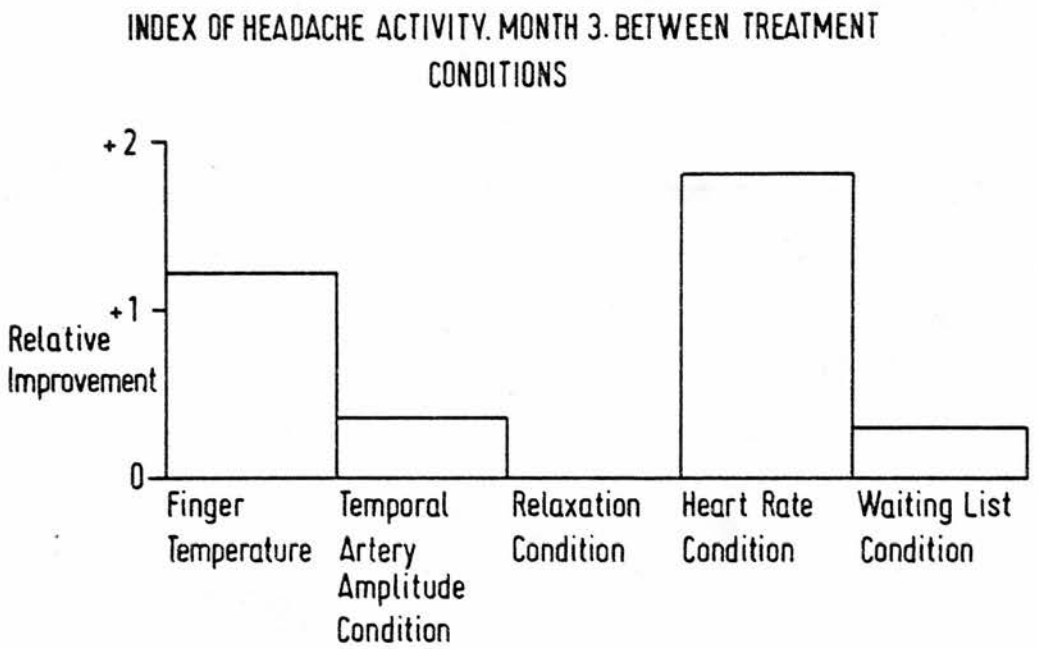


Table 5.

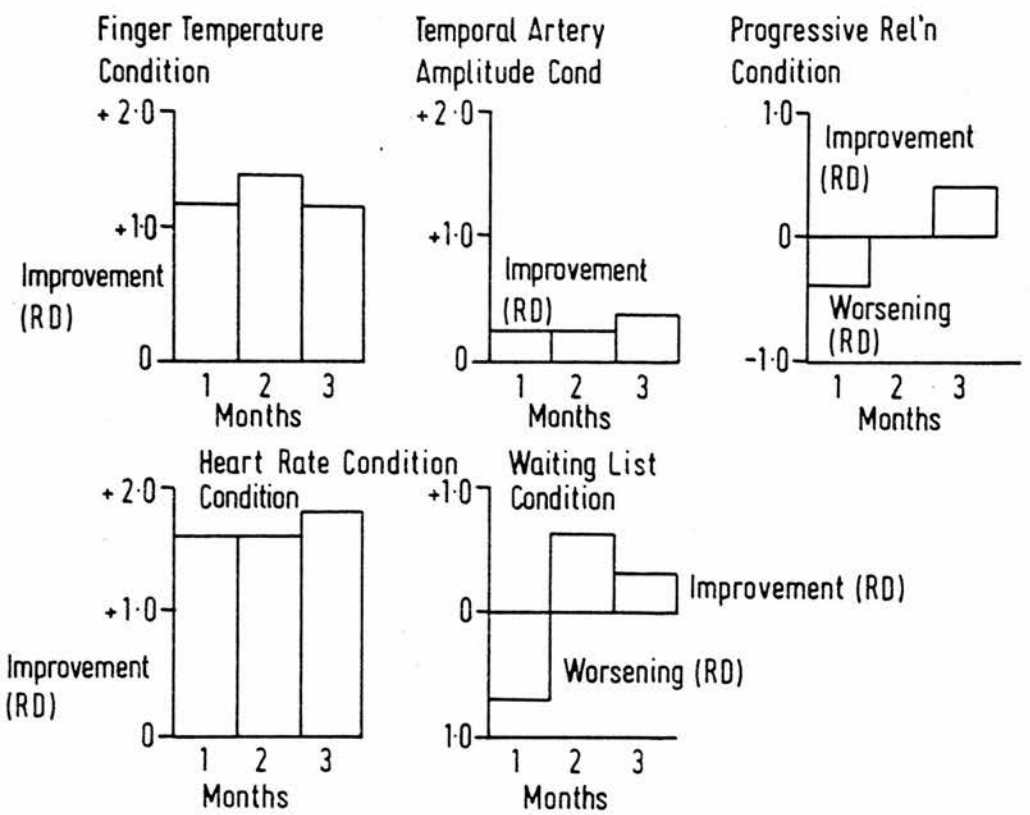
Table 5 is the summary table for the One Way Analysis of Variance between the treatment conditions at month 3.

Source	Sum of Squares	Degrees of Freedom	Mean Square	F Value	2-Tail Probability
Equality of cell means	11.92	4	2.97	2.66	0.06
Error	26.84	24	1.11		

The probability value just failed to reach significance.

Graph 4.

Graph 4 shows the mean relative difference values for 'index of headache activity' over the three follow-up months for the finger temperature, temporal artery amplitude, relaxation, heart rate and waiting list conditions respectively.



ANALGESIC INDEX PER MONTH

Table 1.

Table 1 is the summary table of the 2 Way repeated measures ANOVA of relative changes in 'analgesic index' values between treatment conditions and within the three follow-up months. (Appendix 3.6). No significant differences on any of the levels was found.

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability
Treatment	17.31	4	4.3	1.4	0.27
Error	74.35	24	3.09		
Month	2.01	2	1.00	2.33	0.11
Month x treatment	1.86	8	0.23	0.54	0.82
Error	20.69	48	0.43		

Two Way Analysis of Variance.

FREQUENCY OF THE USE OF VASOCONSTRICTOR DRUGS

Table 1.

Table 1 is the summary table of the 2 Way repeated measures ANOVA of relative changes in 'frequency of use of vasoconstrictor drugs' between treatment conditions and within the three follow-up months. (Appendix 3.7).

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability
Treatment	10.71	4	2.68	2.30	0.09
Error	27.96	24	1.16		
Month	2.23	2	1.12	3.29	0.05
Month x treatment	2.99	8	0.37	1.11	0.38
Error	16.27	48	0.34		

Two Way Analysis of Variance.

Table 1 shows that there was a difference in the frequency of the use of vasoconstrictor drugs over the three follow-up months ($F = 3.29$, 2 df, $p < 0.05$). In the absence of a treatment or treatment x month effect, it can be concluded that the changes over the follow-up months was constant for all groups.

Table 2.

Mean changes in the use of vasoconstrictor drugs (RD scores) for the three follow-up months

Month 1	Month 2	Month 3
+0.07	0.00	+0.07

Table 2 shows that the frequency of use of vasoconstrictor drugs, increased slightly from the baseline levels, on months 1 and 3. On month 2, a return to baseline frequency was observed.

NUMBER OF PROPHYLACTIC DRUGS TAKEN PER MONTH

Table 1.

Table 1 is the summary table of relative changes in 'number of prophylactic drugs taken per month' values between treatment conditions and within the three follow-up months. (Appendix 3.8). No significant differences on any of the levels was found.

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability
Treatment	1.49	4	0.37	0.77	0.56
Error	11.66	24	0.49		
Month	0.25	2	0.12	0.67	0.51
Month x treatment	1.66	8	0.20	1.12	0.37
Error	8.91	48	0.19		

2 Way Analysis of Variance.

MULTIPLE REGRESSION ANALYSIS

The results of this study show that no treatment condition effected significant changes upon any of the physiological variables. It may therefore be concluded that biofeedback did not enable patients to change the physiological response under observation. There were however significant differences between the treatment conditions on the measures of clinical outcome. As a result of this anomaly an investigation into the relationship between the clinical changes and the changes in physiological measures for each individual patient was undertaken.

A multiple regression analysis was used to determine the effects, and the magnitude of the effects of more than one independent variable (physiological changes) on the dependent variable (clinical outcome), (Kerlinger, 1973).

Information from the 26 patients to have completed the ten treatment sessions, and to have maintained continuous contact over the 12 month experimental period was used for analysis. Of the 26 patients, eight were in the finger temperature condition, eight were in the temporal artery amplitude condition and five were in each of the heart rate and progressive relaxation conditions.

The dependent variable.

The dependent variable was a clinical outcome measure for each patient. As the 'index of headache activity' score provided a sensitive measure of overall clinical change, a mean change score was calculated for each patient (mean of the change over the three month follow-up).

The independent variables

As significant 'period' effects occurred more frequently than 'session' effects for changes in the four physiological variables, the mean change for each of the three within session 'periods' was calculated for each patient on each physiological variable. Thus FT 1, FT 2 and FT 3 were finger temperature changes during periods 1, 2 and 3 respectively, across the ten treatment sessions. Mean temporal artery amplitude (TM) changes, standard deviation of temporal artery amplitude (TS) changes and heart rate (HR) changes were similarly estimated. The age of each patient (AGE) and the length of time they had suffered from migraine headaches (DURI) were also included as independent variables.

Details of each patient's age and length of illness were obtained from the assessment questionnaires (Appendix 1.1). The questionnaires were used primarily for diagnostic purposes, although they also contained some personal information about the characteristics of each patient's headaches. The stringent screening procedure used to select patients for the present study resulted in the patient's showing a specific range of diagnostic characteristics. As there was only a small amount of variability in the range of symptoms, diagnostic information was not included in the multiple regression analysis. Also, due to the small number of patients used in the multiple regression analysis, the inconsistencies in the rate of reporting some aspects of personal information, and the questionable accuracy of retrospective information, no further 'personal characteristics' information was included for analysis.

The multiple regression analysis was used to analyse the

relationship between the dependent variable (clinical outcome) and a set of independent variables (physiological changes, age of patient and duration of illness). (Appendix 3.9).

Used as a descriptive tool, multiple regression analysis allows the linear dependence of one variable on others to be summarised and decomposed. Thus instead of focusing on prediction of the dependent variable and its overall dependence on a set of independent variables, the relationship between the dependent variable and particular independent variables can be made. Multiple regression techniques can then be used to determine the magnitude of direct and indirect influences that each variable has on other variables.

The test of statistical significance of the multiple regression analysis is based upon the comparison of variances (or mean squares). S.P.S.S. provides the following information in the computation of the multiple regression analysis. Firstly, a matrix of the correlation coefficients between all variables included for analysis; secondly analyses of variance based on the individual independent variables with the dependent variable. Each analysis of variance is ordered in a step-wise sequence, the highest F values positioned at the beginning of the procedure. Each F ratio gives the level of association between the regression of the dependent variables on the independent variable. Thirdly, a matrix of the multiple correlation coefficients (R) is provided between all variables. The multiple correlation co-efficients (R) show the highest possible correlation between a least squares linear composite of the independent variables and the observed dependent variable. The symbol 'R' can be interpreted like any ordinary coefficient of correlation, except that

the values of 'R' range from 0 to 1.00, unlike 'r' which range from -1.00 through to 1.00 (Kerlinger, 1973).

The sign of 'R' indicates the direction of the relationship, whether positive or negative, while the absolute value of 'R' can be used as an index of the relative strength of the relationship. However, since R^2 indicates the proportion of the variation in the dependent variable explained by each independent variable, it has a clearer interpretation than 'R' as an index of the strength of the relationship. Thus for example if $R^2 = 0.28$, this would indicate that 28% of the variance in the dependent variable is explained by the independent variable/s. The symbol R^2 is accordingly called a 'coefficient of determination'.

PERIOD 1

Table 1 is the matrix of correlation coefficients for temporal artery amplitude/mean changes (TM1), temporal artery amplitude/ standard deviation changes (TS1), heart rate changes (HR1) and finger temperature changes (FT1) compared with clinical outcome (HIN). The changes in physiological variables were the average of all 'period 1' changes over the ten treatment sessions.

	FT1	TM1	TS1	HR1	HIN
FT1	1.00				
TM1	-0.24	1.00			
TS1	0.03	0.46	1.00		
HR1	0.25	0.04	0.02	1.00	
HIN	0.24	-0.40	-0.30	0.26	1.00

Table 2 is a summary table of the linearity of regression for each independent variable on the dependent variable. The 'step-wise' multiple regression procedure gives the coefficient of determination values (R^2) for each independent variable alone and in combination. The R^2 values show the amount of variance in the dependent variable accounted for by the independent variables. The 'change in R^2' values show the effects and the magnitude of the effects of each independent variable upon the dependent variable.

	R^2	Change in R^2	Beta
TM1	0.16	0.16	-0.31
HR1	0.24	0.07	+0.25
TS1	0.26	0.02	-0.16
FT1	0.27	0.01	+0.10

Table 2 shows that 26.5% of the variance in clinical outcome

can be accounted for by the combined effects of the four physiological variables during period 1. However, of this, 16.2% can be accounted for by TM1 alone ($F = 4.65$, 1 df, $p < 0.05$). The remaining 10.3% of the variance of HIN can be accounted for by the combination of the remaining three variables, none of which reach statistical significance.

PERIOD 2

Table 1 is the matrix of correlation coefficients for TM, TS, HR and FT compared with clinical outcome (HIN). The physiological variables were the average of all 'period 2' changes over the ten treatment sessions.

	FT2	TM2	TS2	HR2	HIN
FT2	1.00				
TM2	-0.25	1.00			
TS2	-0.04	0.63	1.00		
HR2	0.36	-0.28	-0.16	1.00	
HIN	0.16	-0.54	-0.44	0.41	1.00

Table 2 is a summary table of the coefficient of determination values (R^2) for each independent variable alone and in combination. The 'change in R^2' values show the magnitude of the effects of each independent variable upon the dependent variable.

	R^2	Change in R^2	Beta
TM2	0.29	0.29	-0.36
HR2	0.37	0.07	+0.30
TS2	0.39	0.02	-0.18
FT2	0.39	0.00	-0.05

Table 2 shows that 39.2% of the variance in HIN can be accounted

for by the combined effects of the four physiological variables during period 2. However, 29.6% of this variance is accounted for by temporal TM2 alone. ($F = 10.07$, $df = 1$, $p < 0.01$). The remaining 9.6% of the variance of HIN can be accounted for by the combined effects of the three remaining variables. No effects of the three remaining variables reach statistical significance.

PERIOD 3

Table 1 is the matrix of correlation coefficients for TM, TS, FT and HR compared with HIN. The physiological variables were the average of all 'period 3' changes over the ten treatment sessions.

	FT3	TM3	TS3	HR3	HIN
FT3	1.00				
TM3	-0.34	1.00			
TS3	-0.08	0.49	1.00		
HR3	0.02	-0.25	0.04	1.00	
HIN	0.04	-0.61	-0.39	0.35	1.00

Table 2 is a summary table of the coefficient of determination values (R^2) and the 'change in R^2' values for each independent variable upon the dependent variable.

	R^2	Change in R^2	Beta
TM3	0.37	0.37	-0.53
HR3	0.41	0.04	+0.23
FT3	0.44	0.03	-0.15
TS3	0.46	0.02	-0.15

Table 2 shows that 45.5% of the variance in clinical outcome can be accounted for by the combined effects of the four physiological variables. As with periods 1 and 2, TM changes account for the

greatest proportion of the variance associated with clinical outcome (37.02%, $F = 14.11$, 1df, $p < 0.01$). The remaining 7.48% of the variance is accounted for by the combined changes of the other independent variables, none of which reach levels of statistical significance.

During the three within session periods, the coefficients of determination (R^2) are consistently greater for mean temporal artery amplitude changes (TM) than for the other physiological variables. This would show that changes in clinical outcome are largely accounted for by changes in TM. The correlation coefficient matrices show that the association between TM and clinical outcome (HIN) is negative; thus an improvement in HIN is associated with decreases in mean temporal artery amplitude. There is no other physiological variable which approaches levels of significance to account for the variance in clinical outcome.

Mean temporal artery amplitude change from period 3 (TM3) was included in a multiple regression analysis with age of the patients (Age) and duration of illness scores (DURI). Although heart rate changes (HR) failed to reach levels of significance in the three within session period analyses, HR3 was included as the fourth independent variable on the basis of being the second largest contributor to the variance of HIN. The dependent variable was HIN.

Table 1 is the matrix of correlation coefficients for TM3, HR3, AGE, DURI and HIN.

	AGE	TM3	DURI	HR3	HIN
AGE	1.00				
TM3	-0.34	1.00			
DURI	-0.18	0.00	1.00		
HR3	-0.11	-0.25	0.02	1.00	
HIN	0.27	-0.61	-0.23	0.35	1.00

Table 2 is a summary table of the coefficients of determination values (R^2) and the 'change in R^2' values for each independent variable upon the dependent variable.

	R^2	Change in R^2	Beta
TM3	0.37	0.37	-0.52
DURI	0.42	0.05	-0.22
HR3	0.47	0.05	+0.24
AGE	0.48	0.01	+0.08

Table 2 shows that 47.5% of the variance in clinical outcome can be accounted for by the combined effects of the four independent variables. TM3 changes account for 37% of the variance associated with clinical outcome. ($F = 14.11$, 1 df, $p < 0.001$). The remaining 10.5% of the variance is accounted for by the combined changes of the other independent variables, none of which reach levels of significance.

SUMMARY OF RESULTS

The results will be summarised in terms of the hypotheses outlined in Chapter 9.

1. Finger temperature changes in the finger temperature control condition do not reach a level of significance. Finger temperature elevation is not demonstrated across the treatment sessions and feedback fails to modify a tendency, displayed by all conditions, for temperature to rise and fall over the within session periods.
2. Temporal artery amplitude (mean and standard deviation) changes are not significantly effected by the presence of feedback. Although the mean and standard deviation changes are slightly increased in the Progressive Relaxation condition, they do not reach significance.
3. Heart Rate changes also fail to reach levels of significance across all sessions for the heart rate condition.
4. For the Progressive Relaxation condition, heart rate decreases; temporal artery amplitude (mean and standard deviation) shows slight increases and finger temperature decreases. None of these changes however reach levels of significance. The decrease in finger temperature may be due to the combined effects of the isometric exercises that preceded the treatment phase, and drift.
5. Clinical improvement cannot be related to a 'group' analysis of physiological changes. A multiple regression analysis of individual physiological changes, age of each patient and duration of illness (independent variables) and mean change in 'Index of headache activity' (dependent variable), showed that mean

temporal artery amplitude changes accounted for the greatest proportion of variance in clinical outcome. A decrease in mean temporal artery amplitude was associated with clinical improvement.

6. The comparative clinical benefits of finger temperature and heart rate feedback was greater than the benefit associated with temporal artery amplitude feedback. The relaxation condition appeared to have derived little clinical benefit from their treatment.

CHAPTER 11

DISCUSSION.

DISCUSSION

The discussion will be divided into four sections. Section 1 (physiological information) will compare the physiological results obtained from each experimental condition with results from previous studies. Section 2 (clinical outcome) will compare the clinical changes observed during the follow-up with the clinical changes reported in previous studies. Section 3 (multiple regression analysis) will present a multiple regression analysis of the relationship between physiological changes and clinical outcome. The concluding section will relate the results of this study to theories of migraine headache and methodological aspects of biofeedback. Points of departure for future research will be suggested throughout all sections of the discussion.

PHYSIOLOGICAL INFORMATION

1. Finger temperature condition.

The results of this study show that the finger temperature condition patients failed to demonstrate significant increases in finger temperature compared with the other treatment conditions. As there were no differences in 'stability point' temperatures between the treatment conditions, it is concluded that the non-significant results were due to the inability of the patients to increase finger temperature, rather than that finger temperature changes were taking place in anticipation of finger temperature feedback.

The present study has attempted to overcome some of the methodological deficiencies apparent in earlier studies. For example, some previous studies - notably those of Sargent, Green and Walters (1972, 1973a and 1973b) - investigated differential temperature changes

between the forehead and fingers. It was however unclear whether temperature changes were due to changes in both forehead and finger blood flow or predominantly one or other of these sites. Although Sargent et al (1973b) and Keefe (1975) established that differential temperature changes were primarily due to increases in finger temperature rather than forehead temperature decreases, some subsequent studies have investigated temperature control using instructions to increase and decrease finger temperature on successive within-trial sessions (Keefe, 1975; Keefe and Gardner, 1979). Significant changes were however found to be largely due to temperature decreases during 'decrease' instruction trials, rather than temperature increases and decreases when instructed. It was therefore difficult to establish whether control had been achieved at all, given the influences of uncontrolled factors, such as drift. The present study investigated 'unidirectional' finger temperature control to overcome the above difficulty, and is consistent with the recommendation of Yates (1980) who suggested that before differential control is investigated, comprehensive studies of 'unidirectional' control should first be undertaken.

A second major difficulty is obtaining information of physiological change which is relatively free of uncontrolled influences. In order to minimise the influences of uncontrolled factors during feedback, a stabilisation period is usually incorporated before any measurements are taken. In the present study a 20 minute stabilisation period preceded every feedback/recording period. Many studies have neglected to include details of the length of stabilisation periods or have used short stabilisation periods. Hunter, Russell, Russell

and Zimmerman (1976) used a three minute baseline period from which to investigate experimental period changes; the baseline period was also used as the stabilisation period. Hunter et al (1976) instructed children to increase finger temperature while holding the thermometer bulb between thumb and forefinger. Although small increases in temperature were observed, given the short stabilisation period, the effects of naturally occurring variations cannot be excluded. It should also be mentioned that although the children were discouraged from rubbing and pressuring the thermometer bulb, insulation effects and movement artifacts cannot be ruled out.

The lack of properly controlled procedures when investigating finger temperature control, and the absence of control groups, may be responsible for the wide range of reported results. Taub and Emurian (1976) reported large increases and decreases in finger temperature with the aid of feedback. Attempts to replicate Taub et al's (1976) findings have however been unsuccessful. Lynch and Schuri (1978) found that subjects showed reliable decreases from baseline temperatures, which persisted following instructions to reverse temperature changes. Roberts, Schuler, Bacon, Zimmerman and Patterson (1975) reported large and reliable performance and learning effects for differential hand-hand temperature feedback, but Lynch and Schuri (1978) stated that attempts to replicate Roberts et al (1975) study had also been unsuccessful. Despite giving the subjects four times as much training than in the original experiment, processing and computing the information by identical methods, control was not demonstrated.

In the present study, the finger temperature condition demon-

strated a mean increase in temperature of 0.1°C above baseline. While many studies have neglected to include details of temperature changes Reading and Mohr (1976) regarded a rise of 0.1°C as a demonstration of successful control. The Reading et al (1976) study, as with many others, was uncontrolled.

Surwit , Shapiro and Feld (1976) stated that a mean decrease of 2°C was shown by subjects instructed to decrease finger temperature. Subjects instructed to increase finger temperature were unable to do so. These experimenters considered that the failure to obtain voluntary control of temperature increases may have been due to a 'ceiling effect' since the average starting temperature of their subjects was 33-34°C, and hence little opportunity for temperature increase was available. In a second study of the same report, Surwit et al (1976) attempted to overcome the ceiling effect by reducing room temperature from 22.5°C, as in their first study, to 19.5°C. However, once again, subjects were unable to increase finger temperature, even though the average starting temperature had been decreased to around 30°C. In the present study, the average starting temperature of all patients was 33.4°C, and was 33.7°C for the finger temperature condition patients. As the room temperature was similar to Surwit's et al (1976) original experimental temperature of 22.5°C, decreases in finger temperature may have been anticipated. The small temperature increases demonstrated in this study may therefore suggest that some volitional control had been achieved (at least by Reading and Mohr's 1976 criterion); this must however remain speculative.

In summary, the results of the present study show that biofeedback has little effect upon finger temperature changes. As stated,

the high stability point finger temperatures, and the small capacity for further increases displayed by finger temperature, may account for the results. In the light of the pilot study findings that feedback may counteract the effects of drift, small increases in finger temperature may still indicate a degree of volitional control.

2. Temporal artery amplitude condition (CVMR).

In the present study, maximum and minimum temporal artery pulse amplitude measurements were taken at 30 second intervals over the five minute pre-experimental baseline and the three five minute periods of each session. From these recordings, measurements of central tendency of the amplitude (mean) and the spread or variability of the amplitude changes (standard deviation) were obtained.

The results show that there were no significant changes due to treatment effects in either the means or the standard deviations. The results therefore suggest that temporal artery amplitude feedback did not enable volitional decreases in temporal artery amplitude to be achieved.

As previously mentioned, little is known about the reactivity of the temporal artery. An increase in sympathetic nervous system activity is assumed to cause vasoconstriction, whilst a decrease in sympathetic activity is assumed to cause vasodilation of the temporal artery. This would appear to be a rather simplistic model of temporal artery reactivity as Sokolov (1963) stated that vasoconstriction may occur when a stimulus is novel or alerting. The relationship between the different types of stimuli in terms of their effects upon sympathetic arousal, and the consequent changes in temporal artery are still to be elucidated. However given the above information, it may be

the case that the state of passive concentration encouraged during biofeedback may facilitate vasodilatation of the temporal artery. Vasodilatation is however antagonistic to the change required during temporal artery feedback.

There are only a small number of experiments concerned specifically with changes in temporal artery amplitude; some studies infer a direct relationship between temporal artery amplitude changes and blood volume changes in the forehead (Friar and Beatty, 1976; Zamani, 1974). Given the possible influence of arteriovenous shunts (Heyck, 1971) and the lack of significant association between forehead blood volume changes and temporal artery amplitude changes (Attfield and Peck, 1979), the generality of forehead blood flow changes to temporal artery activity is questionable. One point of interest is that Friar and Beatty (1976) controlled for the effects of forehead blood flow feedback using a group of migraineurs given pulse amplitude feedback from the fingers. The fingers were considered to be an irrelevant site of control. The results showed that the experimental condition subjects demonstrated a decrease in forehead blood flow, whilst at the same time a decrease in finger pulse amplitude occurred (and probably finger temperature). Headache activity for the experimental group was found to improve. This result would appear to contradict the results of Johnson and Turin (1975) and Turin and Johnson (1976) who found that a decrease in finger temperature was accompanied by a significant worsening of headache activity.

Other studies of CVMR have placed the plethysmograph over the temporal artery at a point above the zygomatic arch (as in this study).

At this point, artery pulsations are easily detectable and the relationship between temporal artery activity and migraine can be more appropriately investigated.

Feuerstein and Adams (1977) measured blood volume pulse changes, blood volume changes and frequency of vasospasms; the origin of the vasospasm is not made clear in this study. Results showed that changes in one measure were not always associated with changes in the others. It was also noted that one subject gained control over temporal artery responses by contracting forehead muscles. As there were only two migraine headache patients in this experiment, the generality of the results about CVMR control would appear limited. Feuerstein, Adams and Beiman (1976) gave CVMR feedback and EMG feedback to an elderly patient with a history of both tension and migraine headaches. The frequency of vasospasm activity was found to decrease during EMG feedback. When CVMR feedback was presented, the frequency of vasospasm activity further decreased and a decrease in pulse amplitude was noted. The decrease in pulse amplitude occurred in one session only (session 5 of 6 sessions given). It was therefore concluded that changes across trials were not systematic. The lack of a significant session x trial interaction would also limit conclusions regarding the demonstration of a learning effect. Feuerstein et al (1977) concluded that 'idiosyncratic responses' characterise the results of CVMR feedback, and that a minimum of 15 treatment sessions should be employed during feedback. However, Sturgis, Tollison and Adams (1978) used 15 treatment sessions and found that a reduction in temporal artery amplitude was associated with the termination of feedback and that no changes were found while feedback was being given.

The variable results of these studies would indicate the need for further research into the responsivity of the temporal artery, especially in the light of the number of vascular responses which have been observed. It is also proposed that the effects of the feedback stimulus and the methods of measuring changes in artery responses should be investigated further. In the present study, changes in the central tendency (mean) of the artery amplitude and changes in the variability of the temporal artery amplitude (standard deviation) were analysed separately. In previous studies a 'window discriminator' has frequently been used to monitor changes in artery amplitude. The temporal artery wave form is displayed on a recording device (an oscilloscope has frequently been used) and the window discriminator is placed over the display. The upper limit of the window is crossed by the increases in amplitude of the systolic peak of the waveform, while the lower limit of the window is set at the diastolic limb of the blood volume pulse. Feedback is presented to the subject if the artery amplitude fails to fall within the limits of the discriminator window. The limits of the discriminator window are then increased or decreased so that a progressive shaping of the artery responses can be achieved. However, Feuerstein and Adams (1977) stated that this type of measurement procedure may not give an accurate index of blood volume level but can be considered a measure of blood volume pulse variability. The following hypothetical example illustrates this point:

If, during a feedback trial the maximum height of the artery amplitude is 19 millimeters, the height of the window discriminator would be set accordingly. If during the trial period, some pulses

have a maximum amplitude of 20 millimeters and others an amplitude of 14 millimeters, a mean amplitude measurement of 17 millimeters would be obtained, and pulse beats exceeding the criterion would be observed. If after feedback, the amplitudes changed to 18 millimeters and 16 millimeters respectively, the mean size of the amplitude would remain 17 millimeters, however a reduction in the number of pulses exceeding the criterion would be observed. Under these conditions the variability of the amplitude responses would have decreased but not the overall mean size of the amplitude. The results of the present study would appear to support Feuerstein et al's (1977) observation. The mean artery changes shown by the temporal artery amplitude condition during period 1 did not decrease below baseline, while the standard deviation score did achieve a below baseline change (the only condition to achieve a below baseline change). Thus temporal artery amplitude feedback would appear to facilitate a decrease in the variability of the artery amplitude rather than an overall decrease in mean artery amplitude.

The second point concerns the effect of binary feedback as used in previous studies, compared with the analogue feedback as used in the present study. Although there is no evidence of the comparative efficacy of one or other of these methods, Hume (1977) stated that analogue feedback is, on a priori grounds, likely to produce more efficient learning. If, for example, the presentation of white noise (binary feedback) proved unpleasant to the subject, a transient decrease in temporal artery amplitude may result (Sokolov, 1963). These are speculative comments, but serve to indicate the need for further detailed research in this area.

3. Progressive relaxation condition.

Progressive relaxation exercises have often been used in combination with other procedures in the treatment of migraine, thus there have arisen questions about the role of relaxation in combined relaxation/biofeedback packages.

Mathew, Ho, Kralik and Claghorn (1979) stated that there is evidence to suggest that the therapeutic action of biofeedback is mediated through a 'relaxation response'. The inclusion of a progressive relaxation condition in the present study was designed to investigate whether specific physiological changes could contribute to the clinical changes of migraine headache patients. The results of the study show that the relaxation condition patients showed a slight decrease in heart rate and finger temperature and increases in the mean and standard deviation changes in temporal artery amplitude. None of the physiological changes reached levels of significance. The fact that the decrease in finger temperature was slightly in excess of the other treatment conditions may indicate that the effects of drift were combined with the effects of the isometric exercises which preceded the onset of the recording phase of the session (Lynch and Schuri, 1978).

Although the observed physiological changes may be consistent with the effects of reduced sympathetic activity, it may be precipitant to conclude that a 'relaxation response' had been demonstrated. Lacey and Lacey (1958) recorded changes in blood pressure, skin conductance, heart rate and heart rate variability, to various stimuli. They concluded that while measurements of a single locus variable might be a useful indicator of the effects of

different stimuli, there was a marked independence of various measures characteristic of each response. Christoph, Kron, Luborsky and Fishman (1978) measured heart rate, respiration rate and blood pressure to investigate whether a specific relaxation response could be observed. They concluded that there was no evidence to support the concept of the relaxation response in terms of only an across measure decrease in the physiological systems. Individual differences in the amount of response, the direction of response and the inter-relationships between the physiological measures were observed. Even within the most consistent measures, some subjects showed increases rather than decreases. The findings are therefore considered consistent with theories emphasising individual patterns of relaxation responses.

In a review of the effects of abbreviated relaxation instructions (as used in the present study), King (1980) stated that a clear picture fails to emerge with respect to the psychophysiological effects of abbreviated progressive relaxation in stress and non-stress situations. King (1980) further found that correlations between subjective ratings of anxiety and relaxation, and measured physiological variables during relaxation are low and non-significant. These low and non-significant correlations would support earlier observations by Hume (1977) who stated that there is little understanding of the inter-relationship between the cognitive and somatic components of relaxation.

4. Heart rate condition.

In the present study the slight decreases in heart rate are consistent with a decrease in sympathetic nervous system activity.

The fact that the mean decrease in heart rate is only slightly in excess of the decrease observed for the progressive relaxation condition would support the observation by Yates (1980) that decreases in heart rate are difficult to demonstrate, a factor probably attributable to the fact that resting level heart rate is close to the lowest physiological level attainable by normal subjects.

CLINICAL OUTCOME

The following is a brief summary of the clinical changes that occurred during the follow-up period of this study. The clinical changes for each treatment condition will then be related to the results of previous studies.

Clinical change was measured on seven scales; frequency of headaches, maximum intensity of headaches, headache duration, an index of headache activity (frequency x intensity x duration), an analgesic index, the frequency of vasoconstrictor drug administration and the number of prophylactic drugs taken.

It is evident from the results that significant differences in clinical change was obtained on four of the above seven scales; frequency of headache, intensity of headache, duration of headache and index of headache activity.

Frequency of Headache

The most consistently superior treatment conditions were the finger temperature control and heart rate control treatment conditions. The progressive relaxation and waiting list control conditions showed no improvement at all over the follow-up months. Patients in both conditions initially became worse during the first month of the

follow-up period, and returned to baseline levels during the second and third months. The temporal artery amplitude condition showed a consistent but non-significant improvement throughout the follow-up.

Intensity of Headaches

Again the most consistently superior treatment conditions were the finger temperature and heart rate treatment conditions. During the follow-up month 1, the waiting list condition showed a worsening in headache intensity; an improvement in all other treatment conditions was observed. During the second and third months, the temporal artery amplitude condition returned to baseline whilst the relaxation condition, after returning to baseline levels on the second month, showed a slight but non-significant improvement at the third month.

Duration of Headaches

For the headache duration measures, values for the first follow-up month indicated that a general improvement took place over all treatment conditions except for the relaxation condition, which became worse. Over the remaining follow-up months, the relaxation condition patients reverted to baseline levels. The temporal artery amplitude condition patients showed a slight improvement on the first and second follow-up months, and returned to baseline levels during the third follow-up month. The heart rate condition showed significantly greater improvement over all other conditions except for the finger temperature condition.

Index of Headache Activity

The index of headache activity scores reflect the findings observed in the frequency, intensity and duration measures. Finger

temperature and heart rate conditions show the most consistent improvement over the follow-up months. Temporal artery amplitude patients showed a slight, consistent, but non-significant improvement over the follow-up months. The relaxation condition patients tended to show a gradual improvement during the follow-up. In month 1, there was a worsening of headache activity; month 2 showed a return to baseline followed by a slight improvement in month 3. The waiting list patients showed slight improvement in month 2 after worsening on month 1; however this improvement was not maintained during month 3.

Other Measures

There were no significant condition x month interactions on the other clinical variables (medication indices) perhaps due to the small amount of medication that was consumed during the experimental period.

In summary, consistently greater improvement was shown for the finger temperature and heart rate conditions in comparison with the other treatment conditions over the follow-up months. Progressive relaxation condition patients in general showed little improvement, and even became worse during the first follow-up month (on intensity and duration measures). The relaxation condition results could be considered comparable to the waiting list control condition results. The temporal artery amplitude condition showed a slight, non-significant improvement that was consistent over the follow-up period.

1. Finger Temperature Condition

The finger temperature condition results would appear to be consistent with previous clinical studies. However, a dearth of

controlled studies makes accurate comparisons difficult.

Andreychuk and Skriver (1975) found 'handwarming' to be more effective in reducing headache activity than hypnosis and EEG (alpha wave) feedback; however there was no follow-up included. Diamond, Diamond-Falk and De Veno (1978) found that finger temperature combined with autogenic phrases was superior to relaxation instructions. The results of this study were however based on retrospective data, and not upon continued monitoring of headache activity. Peck (1980) found that finger temperature and EMG feedback provided some relief in headache symptomatology after a 24 week follow-up period. After 73 weeks, the patients' headache activity had reverted to baseline measures. Both finger temperature and EMG changes failed to reach significance in this study.

The results of the present study would appear comparable with the findings of Reading and Mohr (1976) who used a finger temperature increase of $0.1^{\circ}C$ as the criterion for successful elevation. Reading and Mohr (1976) found that improvement was maintained over a two month follow-up. This however was an uncontrolled study and 'expectancy' effects cannot be entirely excluded. The results of the present study would also be comparable with the findings of Mullinix, Norton, Hack and Fishman (1978) who showed that clinical benefits were unrelated to significant increases in finger temperature, although the absence of quantitive date makes further comparison impossible. In the present study, clinical benefit was mainly associated with the two conditions under which patients showed slight mean increases in finger temperature, viz finger temperature and heart rate conditions. However, the temporal artery amplitude

condition showed a slight decrease in mean finger temperature, but demonstrated consistent clinical improvement over the follow-up, albeit not significant.

2. Temporal artery amplitude condition (CVMR)

As with the finger temperature condition, a dearth of uncontrolled studies limits the comparability of the present results with previous work. Zamani (1974) stated that CVMR proved of greater therapeutic benefit than relaxation in the treatment of migraine headaches. Friar and Beatty (1976) compared an experimental group given feedback of temporal artery amplitude and instructions to try and reduce it, with a control group who received similar feedback but from the pulse amplitude of an index finger. The results showed that the experimental group demonstrated a significant reduction in artery amplitude which was associated with a reduction in major headaches. However neither study obtained information directly from the superficial temporal artery, but rather inferred artery changes from ramifications of the artery at the site of the forehead. Although the results of the present study would be consistent with the findings of Zamani (1974), Friar and Beatty (1976) gave no follow-up information.

Feuerstein and Adams (1977) and Feuerstein, Adams and Beiman (1975) compared EMG and CVMR feedback with migraine and tension headache patients and reported that CVMR was most effective in alleviating migraine headaches. As EMG feedback is aimed at facilitating the 'relaxation' of specific groups of muscles in the face, the results of these studies may be considered comparable to that of Zamani (1974) and the results of the present study. However as both Feuerstein and Adams (1977) and Feuerstein et al (1975)

incorporated only nine and eight week follow-up periods respectively, conclusions regarding the comparative effectiveness of the treatments must remain open to question.

Bild (1976) concluded that CVMR was more effective than EMG feedback in the treatment of migraine headaches. Both treatment conditions were found to be more effective than no-treatment although a lack of diagnostic criterion and a limited follow-up (45 days) would render the results equivocal. The lack of a placebo treatment condition may also lead to speculation about the influence of 'expectancy' effects.

In summary, conclusions regarding the clinical efficacy of CVMR feedback are tentative. The above studies are characterised by a number of methodological deficiencies, including the lack of adequate controls, an absence of diagnostic criteria and short follow-up periods.

3. Progressive relaxation condition

In contrast to the plethora of biofeedback literature, very little work has been reported on the effectiveness of progressive relaxation instructions as a treatment for migraine headaches. One of the major reasons for this is that progressive relaxation has seldom been used alone, but rather to supplement other treatment procedures such as group therapy (Hay and Madders, 1971) and EEG and hypnosis (Stambaugh and House, 1977). Progressive relaxation also has a number of variants, some including a greater 'muscular relaxation' component (Jacobson, 1938), while others have greater meditative components (transcendental meditation; Benson, Klemchuk and Graham, 1974), and meditative and self instruction components (autogenic

training; Schultz and Luthe, 1969).

Although relaxation training of various kinds has shown encouraging results, the lack of controlled follow-up studies limits conclusions about its efficacy. Perhaps the main advantage of progressive relaxation over other approaches is in terms of cost effectiveness; since relaxation training can be conducted in groups, it would seem a promising area for further research (Blanchard, Ahles and Shaw, 1979).

In general, where progressive relaxation has been used as a single independent variable controlling for the effects of biofeedback treatments, it has shown inferior performance. Beasley (1976) found that a combination of finger temperature and EEG feedback with autogenic phrases and relaxation instructions was superior to combined progressive relaxation with autogenic phrases. The single effect of relaxation alone was not however investigated. Zamani (1974) compared progressive relaxation with CVMR responses from the forehead. As previously stated, Zamani (1974) found that feedback was superior to progressive relaxation. Mitchell and Mitchell (1971) compared relaxation with combined relaxation and desensitisation (single model versus a multiple model approach) and found that the combined relaxation and desensitisation package (multiple model) was superior to relaxation alone (single model).

One of the few studies which have found relaxation to be of comparable benefit to biofeedback was conducted by Blanchard, Theobald, Williamson, Silver and Brown (1978). Although the progressive relaxation condition showed a slight improvement over finger temperature biofeedback during the last week of the treatment period, the difference

was not maintained after one month. Blanchard et al (1978) incorporated a 'no-treatment' control condition over the baseline and treatment periods of the experiment. They did not include headache information from the no-treatment group over the follow-up period. As a general decrease in headache activity characterised all groups during the baseline and treatment period of the experiment, it is difficult to determine whether the treatment conditions eventually derived any greater benefit than was experienced by the no-treatment control condition.

In the present study, the progressive relaxation condition derived little clinical benefit from their treatment. This may be due to the stringent selection procedure used to include patients into the study. If headaches are viewed on a continuum of vascular involvement, the patients in the present study may be seen as representing the extreme of the continuum, having a high degree of vascular involvement and a small tension component in their headaches. The present findings would therefore be consistent with Reading and Mohr (1976) who suggested that those subjects reporting tension-like headaches found the handwarming technique largely ineffective in controlling headaches.

4. Heart Rate Condition

The heart rate condition demonstrated an increase in clinical benefit that was consistently superior to all other treatment conditions except for finger temperature biofeedback. As a relationship between heart rate control and migraine has not previously been demonstrated, the results of the present study cannot be compared with any previous work in the area.

The findings of the present study may be considered similar to those of Mullinix, Norton, Hack and Fishman (1978) who found that false feedback of finger temperature increase facilitated clinical improvement in migraine headache. The actual change in finger temperature was not however significantly above baseline measures. The same clinical improvement was found for a second group of migraine subjects who demonstrated successful elevation of finger temperature with the aid of true feedback. Mullinix et al (1978) concluded that the results suggested that 'biofeedback techniques are useful in treating patients with migraine, and the mechanism, presumably a placebo effect, is independent of peripheral skin temperature'.

In the present study, placebo effects cannot be completely excluded. Shapiro (1960) cited a number of examples in which placebo effects produced significant clinical improvement in a variety of ailments, and the effects were found to be maintained over considerable periods of time. There are however a number of points which would appear inconsistent with use of the placebo as an explanation for the results of the present study. Firstly, the clinical improvement shown by the attention placebo condition (heart rate feedback) is inconsistent with hypothesis 6 chapter 9. This hypothesis stated that clinical change expected for patients in the heart rate condition would be similar to changes shown by patients in the no-treatment, waiting list control condition. As this was not the case, the results would therefore appear to be independent of any 'experimenter expectancy' effects that may have been manifest during the experimental period. Secondly, Wilkinson, Neylan and Rowsell (1974) stated that in any clinical trial there will be about a 30% placebo reaction, thus any

new treatment for migraine must show an improvement rate in excess of 50% before it can be considered really satisfactory. In the present study, the finger temperature condition and the heart rate conditions showed clinical benefit in excess of 65% and 75% respectively on follow-up measures of the 'index of headache activity' scale. Although the design of the present study may minimise the effects of non-specific treatment factors, the unknown effects of the 'differential placebo power' of the treatment conditions would make this a vital area for further research.

In conclusion, the between condition analysis of the physiological results shows that biofeedback does not enable subjects to gain significant control over the physiological parameter displayed. The observed non-significance of the physiological changes however presents an anomolous situation in the light of the clinical changes obtained between the treatment conditions over the follow-up months. It would therefore appear that some distinguishing feature, not shown by 'group' analysis of the information, may be apparent that would account for changes in clinical outcome. In order to investigate this aspect of the results, a multiple regression analysis was carried out. The multiple regression analysis was designed to evaluate the relative influence of each independent variable (physiological changes, age of patient and duration of illness) alone and in combination, upon a clinical change score. The analysis also generated other useful data, such as intercorrelations between the physiological changes.

MULTIPLE REGRESSION ANALYSIS

The average change for each of the three within session periods was obtained for the ten treatment sessions and used for analysis.

Period 1 (Independent variables : finger temperature, mean temporal artery amplitude score, standard deviation of temporal artery amplitude changes and heart rate changes. Dependent variable : Index of headache activity.)

In period 1, the physiological variables accounted for 27% of the variance in clinical change. The mean temporal artery amplitude change alone accounted for 16% of the variance, and was the only significant factor of the four physiological variables.

Period 2

In period 2 (variables as in period 1), the four physiological variables accounted for 39% of the variance in clinical change. As in period 1, the mean temporal artery amplitude changes accounted for the greatest change (30%) and was the only independent variable to attain significance.

Period 3

In period 3 (variables as in period 1), the four physiological variables accounted for 46% of the variance in clinical outcome. The mean temporal artery amplitude change accounted for 37% of the clinical outcome score, and was again the only independent variable to attain statistical significance.

In the fourth multiple regression analysis the independent variables consisted of mean temporal artery amplitude changes (Period 3), heart rate changes (period 3), the age of the patient and the length of migraine history of each patient. The dependent variable was again the 'index of headache activity' score. The results showed that the four independent variables accounted for 48% of the variance in clinical change; the mean temporal artery amplitude score alone accounted for 37% of the variance. The temporal artery amplitude score was again

the only independent variable to attain significance.

The results of the multiple-regression analysis showed that mean temporal artery amplitude changes accounted for the greatest amount of variance in the clinical outcome. The consistently high negative correlation coefficients between clinical change and mean temporal artery amplitude change showed that an increase in clinical benefit was associated with a decrease in mean temporal artery amplitude.

CONCLUDING COMMENTS

Conducting a clinical research project has a number of inherent methodological and ethical difficulties. The major difficulties apparent in the present study were:

Patient Selection

The inclusion of a relatively small patient population, due to the availability of patients and the screening procedure adopted for the selection of patients.

Patient Compliance

Maintaining the patients' motivation during the baseline phase of the experiment to ensure accurate completion of the headache record forms and willingness to undertake treatment. The two pre-experimental baseline sessions (after months 1 and 2) were intended to reduce the patients' anxiety regarding the treatment and engender interest in completing the treatment phase of the experiment.

The 'change process' which takes place during biofeedback consists of two separate components; the presentation of the physiological information and the ability of the individual to effect change upon the physiological process. The physiological parameters under

investigation in this study (finger temperature, temporal artery amplitude and heart rate) have relatively small capacities for variation in the 'resting state' normal individual. The relative independence of within subject and across subject changes in physiological responses (Lacey and Lacey, 1958) may further add to the difficulty in obtaining consistent across condition changes and effect the patient's motivation for completing the treatment phase of the experiment. Presenting each patient with a rationale of the treatment at the beginning of the treatment phase of the experiment was intended to emphasise the salience of the treatment, and maintain motivation during the treatment phase of the experiment. An inability to change the displayed phsyiological parameter, or to discriminate changes, with or without the aid of feedback may have adversely effected patient motivation, especially in the case of the progressive relaxation patients who received no information regarding physiological changes throughout the treatment sessions.

Finally, patient motivation in maintaining regular contact with the experimenter during the follow-up phase of the experiment in order to accurately monitor headache activity. The use of 'postal headache forms' with reminder letters and stamped addressed envelopes was intended to maximise patient compliance during the follow-up.

The no-treatment control condition patients were also sent headache forms, a reminder letter and a stamped addressed envelope at monthly intervals for the 12 months of the study. Although all patients were offered a course of treatment at the end of the experimental period, the small number of patients to reply to this offer (three patients) would indicate the unsatisfactory nature of

this arrangement.

Given the above criticisms of the present study, and the nature of the present results, including the discrepancy between a lack of significant across condition changes in the physiological variables and significant clinical outcome change, there may be a need for greater emphasis upon single case design studies (Barlow, Blanchard, Hayes and Epstein, 1977) in biofeedback research.

Although changes in mean temporal artery amplitude were correlated negatively with finger temperature changes across all within session periods, the results failed to reach significance. There is therefore no significant relationship between changes in finger temperature and mean changes in temporal artery amplitude. The finding that finger temperature consistently contributed low and non-significant changes in clinical outcome supports the findings of Mullinix et al (1978). Furthermore, the findings appear to be contrary to Sargent et al's (1973b) rationale for finger temperature control as a treatment for migraine headaches. Although finger temperature may be a sensitive one variable index of autonomic arousal, the specificity of such control appears to be independent of the therapeutic benefit gained from the procedure.

Changes in heart rate and mean temporal artery amplitude correlated positively, although to a low and non-significant degree. The finding that changes in heart rate contributed small and non-significant changes in clinical outcome supports the inclusion of heart rate feedback as an attention placebo condition in the present study.

The differences in clinical outcome between the three biofeed-

back conditions may be explained with reference to somatic mediation aspects of biofeedback. Somatic mediation refers to the ability of the patient to discriminate levels of, and changes in, autonomic activity, related to the ability of the patient to bring these activities under increased voluntary control with or without feedback. Previous research on this aspect of biofeedback is small and mainly concerned with changes in heart rate. Epstein and Miller (1974) requested subjects to press a button whenever they thought their heart rate was above or below an average level. It was found that discrimination training (with biofeedback) led to successful discrimination of heart rate changes on withdrawal of the feedback. Similarly, Gainer (1978) found a marked improvement in headache activity after a patient had been taught to discriminate finger temperature changes which occurred prior to the onset of her headaches. Before discrimination training, the patient had displayed an ability to increase finger temperature, but had not managed to apply her 'skill' in time to stop the onset of the headache. The evidence of this study would suggest that although the patient may have been successful in elevating finger temperature prior to the onset of the headache, clinical outcome was independent of finger temperature change. It may therefore have been the case that maintained attention upon effecting change at the perceived site of control (fingers) may have mediated change in other physiological responses (temporal artery amplitude).

There have been no studies of patient ability to discriminate change in temporal artery activity. An inability to discriminate small changes in artery activity may reduce the patient's incentive to maintain attention during 'home rehearsal' and thus minimise the

direct influence of control or the indirect influence of other factors. Bakal (1978) stated that 'in general terms, the behavioural literature has brought about a recognition that the patient must assume an active role in his or her treatment'. An inability to discriminate changes in temporal artery amplitude may reduce the 'active component' necessary to effect change, whether the change is a direct or a mediated effect of the patient's active involvement. This may explain why the temporal artery amplitude demonstrated only slight improvement over the follow-up, albeit non-significant in comparison with both finger temperature and heart rate conditions.

The finding that the progressive relaxation condition demonstrated small increases (non-significant) in mean temporal artery amplitude, associated with little clinical benefit would support the vascular theory of Wolff (1963). It would also be consistent with observations that migraine headaches are characteristic of periods of relaxation and a reduction in the pressures of 'everyday life' (Pearce, 1971). Hence, the terms 'holiday headache', 'Friday night headache' and 'weekend headache' have become synonymous with migraine as it is at these times that the opportunity for relaxation is available. The emphasis placed upon 'facial' relaxation when using EMG feedback may explain the comparatively poor therapeutic benefit evident when treating migraine headaches. The vasodilatation consequent upon relaxation would reduce any potential vasoconstrictory responses facilitated by biofeedback.

In conclusion, given that 'migraine patients tend to respond with vasoconstriction in the cerebral arteries when subjected to a task situation (suggesting that the situation is a stressful one)'

(Yates, 1980), further vasoconstriction may be difficult to achieve, even with the aid of feedback. This observation would be consistent with the results of the present study. It would also explain the clinical benefit observed during finger temperature feedback studies where non-significant increases in finger temperature are associated with improvement in headache activity. A similar explanation might also serve to account for the therapeutic benefits associated with CVMR studies. However, these points remain speculative and future studies should attempt to clarify the situation using a variety of feedback and sensitive measurement procedures concentrating upon demonstrating an association between the type of feedback and one specific vascular response at a time.

The results of the present study suggest the need to develop more predictably effective ways of producing cerebral vasoconstriction, and then to compare its effects with that of drugs. Investigations of this nature would be desirable because of the specificity of the locus of action shown by biofeedback in this study, and also observations that biofeedback may compare favourably with the more diffuse and undifferentiated action of vasoconstrictor drugs. It is also apparent that less heed should be paid to diagnostic labels, but that measures of physiological change for each patient, before, during and after headache attacks should be taken and used to design biofeedback treatments accordingly. A series of single case, multiple baseline experiments may be the best method of investigation in the light of present results.

Summary

1. Between group analysis of the effects of finger temperature biofeedback, temporal artery amplitude biofeedback and heart rate biofeedback showed no significant changes in the physiological variables.

2. Finger temperature and heart rate biofeedback conditions showed significantly greater improvement than the other treatment conditions. The temporal artery amplitude condition showed a consistent, non-significant improvement during the follow-up. The progressive relaxation and waiting list conditions demonstrated little clinical improvement.

3. Multiple regression analysis showed no consistent correlation between changes in the physiological variables. Mean temporal artery amplitude change (decrease) accounted for the greatest amount of variance in clinical change.

APPENDIX 1

HEADACHE QUESTIONNAIRE

Number ☐☐☐☐☐

Surname ..... Mr. / Mrs/ Miss

Christian Names ..... ☐

Address .....

.....

Date of Birth ..... ☐☐

What is (or was) your(or if under 16, your father) main occupation?

Job .....

Industry .....

If you are a married woman, what is (or was) your husband's main occupation?

Job ..... ☐☐

Industry .....

1. Have you had a headache within the past year?

YES / NO (Please tick one) ☐

IF YOU HAVE HAD A HEADACHE DURING THE PAST YEAR, PLEASE ANSEER ALL THE QUESTIONS BELOW FOR YOUR HEADACHES DURING THE PAST YEAR ONLY. WE DO NOT WANT DETAILS OF ANY HEADACHES THAT HAPPENED MORE THAN ONE YEAR AGO.

IF YOU HAVE NOT HAD A HEADACHE DURING THE PAST YEAR, PLEASE DO NOT CONTINUE WITH THIS QUESTIONNAIRE.

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2. Are your headaches usually mild or severe; or do you get both mild and severe headaches?

Mild
Severe (Please tick one) ☐
Both

If you get BOTH mild and severe headaches:-

Are they different kinds of headache, that is can you clearly distinguish between them? YES / NO (Please tick one) ☐

IF YOU GET BOTH SEVERE AND MILD HEADACHES, PLEASE ANSWER ALL THE QUESTIONS FOR YOUR SEVERE HEADACHES ONLY.

DO NOT CONTINUE WITH THIS QUESTIONNAIRE IF YOU GET MILD HEADACHES ONLY.

3. Which one of these statements is nearest the truth for you?

My headaches are quite severe
My headaches are very severe (Please tick one) ☐
My headaches are terribly severe
My headaches are almost unbearable

CONSIDERING YOUR SEVERE HEADACHES ONLY

4. Which of these statements is nearest the truth for you?

	(Please tick one)
I hardly notice my headaches at all.	
My headaches rarely inconvenience me.	
My headaches sometimes distract me from what I am doing	
Sometimes I am unable to continue my normal activities because of my headaches.	☐
My headaches sometimes interfere a lot with what I am doing.	
I can hardly do anything when I have a headache.	
I am absolutely fit for nothing when I have a headache.	

5. How long do your headaches usually last? ..... ☐

6. (a) When you have a headache, do you usually have to:-

	(Please tick if applicable)	
lie down?		
rest?		☐
take things easy?		

IF YOU DO - For how long is this usually? ..... hrs.

(b) Have you missed work or school DURING THE PAST YEAR because of a headache?

	(Please tick one)	
YES		☐
NO		

IF YES, for how many days? .....

7. Do you get a headache:-

	(Please tick one)	
About once a year?		
Several times a year?		
About once a month?		☐
Several times a month	(HOW MANY?.....)	
About once a week?		
Several times a week?	(HOW MANY? .....)	

(SEVERE HEADACHES ONLY)

8. What medication do you usually take for your severe headaches?

Name .....

Amount .....

How often .....

9. Can you usually stop the pain by taking a walk or by moving your head?

	(Please tick one)	
YES		☐
NO		

10. Do your headaches usually make your nose run or feel congested?

	(Please tick one)	
YES		☐
NO		

11. Do your severe headaches make you feel as though you have a tight band around your head?

Never? (Please tick one)
Sometimes?
Usually?
Always?

12. Are your headaches throbbing or thumping?

Never? (Please tick one)
Sometimes?
Usually?
Always?

13. Where do you usually feel the headaches?

Behind one eye (Please tick one)
Temples
Forehead
Back of head
All one side
All over the head
If elsewhere, where .....

14. Are your severe headaches on one side only:-

Never? (Please tick one)
Sometimes?
Usually?
Always?

15. Before you get a headache do you know that one is coming?

YES (Please tick one)
NO

IF YOU DO, Please describe briefly what you notice?

.....

.....

.....

16. Before you get a severe headache do you have any difficulties with speech? YES / NO

If so, describe .....

Numbness or tingling sensations? YES / NO

If so, where .....

17. Do your severe headaches often last for more than two days?

YES / NO (Please tick one)

18. Are you headache free for long periods and then experience a number in a short space of time?

YES / NO (Please tick one)

19. Before you have a severe headache do you notice any changes in your sight?

YES / NO (Please tick one) ☐

IF YES, please describe briefly what you notice

.....

.....

.....

20. When you have a headache do you:-

(Please tick any that apply)

Lose your appetite? ☐
Feel dizzy?
Feel sleepy?
Hear ringing in your ears?
Find that light hurts your eyes?

21. When you have a headache do you:-

(Please tick any that apply)

Ever feel sick? ☐
Usually feel sick?
Ever vomit?
Usually vomit?
Always vomit?

22. Have you ever seen a doctor about the headaches?

YES / NO (Please tick one) ☐

IF YES, have you seen a doctor about the headaches during the past year? ☐

YES / NO (Please tick one)

23. Over the years have your headaches become:-

More frequent? (Please tick one) ☐
Less frequent?
or have you noticed no change?

24. Over the years have your headaches become:-

More painful? (Please tick one) ☐
Less painful?
or have you noticed no change?

25. Do you notice that your headaches come at a particular time of the month / week /day.

YES / NO (Please tick one) ☐

IF YES, please specify .....

.....

26. Do you think that your headaches have any connection with your diet?

YES / NO (Please tick one) ☐

IF YES, please specify .....

.....

27. Does anyone else in your family suffer from similar severe headaches?

YES / NO (Please tick one) ☐

IF YES, please specify .....

WE ARE MOST GRATEFUL TO YOU FOR HELPING IN THIS WORK

APPENDIX 1.2

MIGRAINE SYMPTOMS ON THE WATERS HEADACHE QUESTIONNAIRE: A STATISTICAL ANALYSIS

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ABSTRACT

98 patients, who had received a diagnosis of migraine from their general practitioners, completed the Waters Headache Questionnaire. The data were subjected to a Principal Components Analysis (with Varimax rotation). 13 factors were extracted, but none of them comprised those variables regarded as constituting "migraine features". The results are discussed in the context of recent studies which have raised several questions concerning the classification of headaches.

A considerable amount of clinical and research time has been devoted to the topic of migraine headaches, particularly in the last two decades. Friedman et al. (1) proposed a classification system based on symptom patterns of headaches, drawing on the experimental and clinical data available to them at that time. Briefly, they proposed, inter alia, the following headache entities: common migraine, classic migraine, cluster headache, hemiplegic and opthalmoplegic migraine, muscle contraction headache, and combined vascular and muscle contraction headache. The earlier experimental data relating to these entities were summarised by Dalessio (2).

Major advances in the study of headache have occurred within several disciplines. Biochemical abnormalities, particularly deficiencies in serotonin, have been observed (3); cerebral blood flow changes have been recorded during the prodromal and the headache phase of a migraine attack (4); and new psychological methods of treatment have been developed (5). However not all of the recent advances have been consistent with the earlier clinical "conventional wisdom". For example, Blau (6) was critical of the reports that the superficial temporal artery pulse amplitude was larger during the headache phase of the migraine attack, than during the non-painful phase. In a recent review, Morley (7) claimed that because of major deficiencies in experimental design and data analysis, it is impossible to make unequivocal interpretations of the evidence concerning cerebral vasomotor dysfunction in migraine. Bakal & Kaganov (8) found considerable overlap in the pathophysiology of migraine and muscle contraction headaches. Finally, it has been suggested that the varied headache symptom patterns may be simply a

function of differences in severity, rather than of differences in clinical entities (9).

It appears from the above that the problem of headache requires some rethinking and fresh analysis. One useful approach may be to reduce the heterogeneity in the classification of headaches, by examining how headache symptoms cluster together. If symptoms are correlated and form consistent patterns, the patterns so obtained may more precisely provide the defining characteristics of headache syndromes.

The statistical technique of factor analysis is ideally suited to examining the emergence and clustering of symptom patterns. On the basis of a matrix of correlations amongst variables (or symptoms), it simply attempts to examine what would be the optimum combination of variables that could best account for the variance in all the data. The combinations which emerge suggest whether some underlying pattern of relationship exists, such that all the data may be re-arranged or reduced to a smaller set of factors or components. In clinical applications, factor analysis may permit one to see how far symptoms cluster together to form clinical entities. If you start with a large number of variables (say, over thirty), typically it would be found that only four or five factors could sum up most of the information about the relative position of individuals, on the original variables. The factors extracted each account for a decreasing proportion of the remaining variance; at some point, further factors would account for very little extra variance and no more are extracted. It should be noted that factor analysis is a generic term for a large number of related techniques. The technique based most explicitly on

exact mathematical transformations of the original data, and which is particularly suitable for examining symptom factors, is that of principal components analysis. Often however this mathematical approach produces factors which are statistically, but not clinically, interpretable. In such cases, it is quite legitimate to carry out further transformations, or rotations, of the data, to produce simpler and more clinically intelligible factors. Such rotated factors often "line up" more precisely with the original variables, and this facilitates describing and labelling the extracted factors. Varimax rotation is the most commonly used method, and is the one best suited to the analysis of symptom patterns. Examples of the application of factor analysis to medical research have been discussed by Child (10) and factor analysis as a technique has been succinctly reviewed by Kim & Mueller (11).

It appears that only one study has been published describing a factor analysis of headache symptoms. Ziegler et al. (12) obtained seven main factors from 27 headache variables; no one factor comprised the expected "migraine features"; instead it was found that three distinct migraine factors emerged. Their results further challenged commonly held beliefs in a number of ways; for example, there was no strong evidence of a separate "cluster headache" entity.

It would appear that, despite these controversial findings, no further factor analytic study of headache symptomatology has been reported. The present study was intended to fill this gap. A major difference between this study and that of Ziegler et al., however, is that this study utilised a standard questionnaire, the Waters Headache Questionnaire, about which much is already known. For example it has been found that, of subjects given a

diagnosis of migraine based on questionnaire responses, 90% would also be diagnosed as having migraine in a clinical interview (13). Furthermore the items in the Waters Headache Questionnaire are not identical to those used by Ziegler et al. (12). For example, the Waters Headache Questionnaire obtains more precise information concerning the location of the pain, and enquires more closely into associated symptoms, such as appetite changes, feelings of dizziness and sleepiness, light hurting the eyes, etc. However the Waters Questionnaire does not, unlike in the Ziegler et al. (12) study, include items relating to family history of headaches, response to treatment and precipitants of the headache. The items of the Waters Headache Questionnaire are shown in the Appendix.

METHOD

Subjects

98 patients completed the Questionnaire; all were referred to a headache clinic by their general practitioners. Because the patients came from a variety of practices, it is impossible to state the precise diagnostic criteria used; however all subjects were referred with a diagnosis of migraine, and there is no reason to suspect that these patients are not representative of patients so diagnosed. There were 77 females, and 21 males; mean age was 36.8 years, with a standard deviation of 13.5, and a range of 10 to 64. They were predominantly from social classes 1 (20.4%), 2 (39.8%) and 3 (24.5%); only 15.3% were from social classes 4 and 5.

Procedure

All patients were asked to complete the Questionnaire as part of an assessment procedure, prior to commencing treatment by a variety of psychological methods, including relaxation and biofeedback. Only quantitative responses relating to symptomatology were analysed; first by simply examining frequency of endorsement of the items; second, by factor analytic methods, using principal components analysis (without iteration), and subsequently rotated by the Varimax method, with Kaiser normalisation. The computer programmes used in this analysis were part of the Statistical Package for the Social Sciences (SPSS) (14).

Results

1) The responses of the subjects to the items of the Waters Headache Questionnaire are shown in the Appendix.

2) Using Principal Components Analysis, factors were extracted until additional factors accounted for little extra variance; that is, until the eigen value reached a level of less than 1.0. Thirteen factors were found, which were then rotated according to the Varimax criterion (Kaiser Normalisation). After rotation, it was found that the 13 factors accounted for 71.38% of the total variance. Furthermore each of the 13 factors accounted for a reasonably similar, albeit low, proportion of the variance, with no large differences in ability to account for variance between the first factor extracted (6.62%) and the thirteenth (4.6%). Accordingly, all 13 factors are described here; only those symptoms which load (i.e. correlate) with the factor at +0.30 or above are reported. Both total variance and common variance (i.e. variance due just to the factors extracted) are reported for each factor. The details are shown in Table 1.

Table 1 about here

DISCUSSION

Perhaps the most notable finding from the principal components analysis was that no single factor emerged which encompassed the three "migraine features" described by Waters (15): warning sign, unilateral headache, and nausea/vomiting. This is quite consistent with the results of the factor analytic study of Ziegler et al. (12), who also reported that no factor emerged which contained the variables "ordinarily thought of as particularly characteristic of migraine". However, neither did the present study find any factors which closely resemble those obtained by Ziegler et al. (12). This is presumably because different variables were measured; nevertheless there was sufficient similarity in the content of the questionnaires to lead one to expect that some factorial congruence might emerge.

The findings are not however totally at variance with clinical lore. Factor 1 contained two of Waters' three migraine features (unilateral headache and nausea/vomiting); the remaining symptions loading on this factor are generally as expected: pain in the forehead or behind the eyes; light hurting the eyes; and low probability of pain being all over the head. The only anomalous finding was that frequency of headaches had a negative loading on this factor, although not substantially. Of all the factors to emerge, this one most closely resembles a "migraine" factor.

The second factor to emerge describes those headaches which are not particularly troublesome, are located bilaterally in the forehand and are without accompanying sensory or perceptual disturbances. However such headaches may or may not be severe (severity loading $=+0.022$). Of all the factors to

to emerge, this one most closely resembles a "muscle tension headache" component. However, if this were the case, one might expect that the variable "Are your headaches throbbing or thumping?" would be negatively loaded on this factor; but the loading was a mere -0.037. Also, it must be remembered that all subjects in the study had already been diagnosed as clearly suffering from migraine by their general practitioners. However, given that there are notorious difficulties in making a differential diagnosis between migraine and muscle tension headaches, that mixed vascular and tension headaches may occur (Friedman et al. (1)) and that subjects may suffer from both types of headaches at different times, it remains a possibility that this factor represents a muscle tension component in patients with a diagnosis of migraine.

The remaining factors bear no obvious resemblance to putative headache entities, but do raise some points of interest. In factor 3, the symptom of "Warning Signs" is not clearly linked to other clinical features of headache, apart from location and frequency; the presence of warning signs may therefore not be as closely linked with "migraine features" as others have suggested (16). Factor 7 describes an increase in frequency and pain intensity over the last year, which is independent of kind of headache. Of particular interest is factor 11 which largely describes the severity of headaches, and to a lesser extent the frequency. Severity appears to be independent of kind of headache, and of other headache features, since this is the only factor on which severity is highly loaded.

It should be noted that the patients in this study formed a reasonably homogeneous group in terms of chronicity, severity, and symptomatology. Accordingly the variance in the scores was necessarily restricted, and there remains the possibility that, if a wider range of variance had been introduced by using a more heterogeneous group of headache patients, a more clear-cut migraine factor structure may have emerged.

Waters (16) reported that severity is significantly associated with the presence of the three main headache features of warning, nausea/vomiting and unilateral headache. In order to test this, those patients reporting various combinations of the three features were examined for severity ratings. No significant differences emerged between any of the combinations; for example there was no significant difference between the mean severity (3.92) of those subjects reporting all the three features, and the mean (3.80) of those not reporting all three features, ($t = 0.256$, $p = 0.80$). The range of severity ratings was very narrow, from a mean of 3.714 for patients with "vomiting" as the only migraine feature; to a mean of 4.0 for patients with "warning sign" as the only migraine feature. All other combinations of the three migraine features were between these values. These findings are not consistent with the suggestion of Waters (15) that headache severity correlates with the presence of the three migraine features.

Walker (17) suggested that "psychogenic" headaches may be distinguished from migraine, in that they tend to be of longer duration. However others have found the opposite; that is, non-migrainous headaches tended to be of shorter duration (18). The present study found that duration was not closely related to any headache "type", a finding consistent with the results of Ziegler et al. (12).

It is clear that the results of studies on migraine symptomatology do not form a consistent pattern, in terms of the specific constellations of symptoms found. However there is some agreement in that many workers now query whether migraine is a separate clinical syndrome, with features clearly distinct from those of other headache syndromes. The available evidence is more consistent with the suggestion by Waters (15) that "migraine might be an extreme in a continuum rather than a completely distinct clinical entity". The underlying basis of this putative continuum may be the degree of vascular involvement, but more research is required before this can be firmly established.

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APPENDIX

Responses to the Waters Headache Questionnaire.

Symptom Item Endorsement

	% endorsement
(a) Are your headaches :	
Mild	0
Severe	9
Both	91
(b) My headaches are	
Very mild	7
Mild	13
Not usually severe	13
Quite severe	37
Very severe	12
Terribly severe	5
Almost unbearable	9
(c) I hardly notice my headaches at all	0
My headaches rarely inconvenience me	1
My headaches sometimes distract me from what I am doing	4
Sometimes I am unable to continue my normal activities because of my headaches	17
My headaches sometimes interfere a lot with what I am doing	15
I can hardly do anything when I have a headache	33
I am absolutely fit for nothing when I have a headache	30
(d) How long do your headaches last?	
Less than half a day	37
One day	33
More than one day	15
More than two days	15

(e) When you have a headache, do you usually have to -

Lie down	72
Rest	12
Take things easy	16

(f) Have you missed work during the past year because of a headache? 66

(g) Do you get a headache -

About once a year	2
Several times a year	12
About once a month	12
Several times a month	33
About once a week	8
Several times a week	33

(h) Are your headaches throbbing or thumping?

Never	9
Sometimes	22
Usually	29
Always	37

(i) Where do you usually feel the headaches?

Temples	27
Forehead/behind eyes	49
Back of head	20
Top of head	12
All one side	47
All over head	12
Elsewhere	6

(j) Are your headaches on one side only?

Never	9
Sometimes	22
Usually	28
Always	41

(k) Before you get a headache, do you know that one is coming? 79

(l) When you have a headache, do you notice any change in your sight? 64

(m) When you have a headache, do you

Lose your appetite	67
Feel dizzy	41
Feel sleepy	40
Hear ringing in your ears	14
Find that light hurts your eyes	83
Notice tingling, or any strange feeling in any part of your body	50

(n) When you have a headache, do you

Never feel sick or vomit	9
Ever feel sick	8
Usually feel sick	20
Ever vomit	35
Usually vomit	14
Always vomit	13

(o) Over the year, have your headaches become

More frequent	54
Less frequent	11
Or have you noticed no change	35

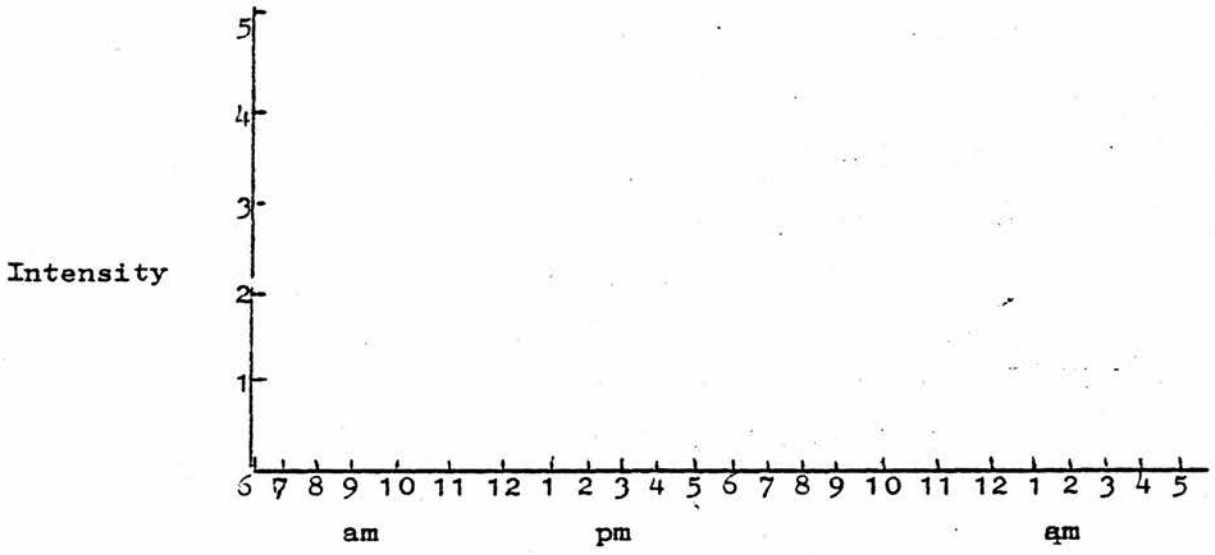
(p) Over the years, have your headaches become

More painful	44
Less painful	8
Or have you noticed no change	48

APPENDIX 1.3

NAME .....

DATE .....



1. Slight sensations in the head
2. Slightly painful
3. Definitely painful
4. Extremely painful
5. The worst pain possible

****

Medication: What

How much

When

Appendix 1.4

UNIVERSITY DEPARTMENT OF PSYCHIATRY
(ROYAL EDINBURGH HOSPITAL)
MORNINGSIDE PARK
EDINBURGH.
EH10 5HF

TELEPHONE No. 031-447 2011

Dear

Thank you for taking time to discuss your headaches with me. Unfortunately your headache does not appear to be of the type which would benefit from any of the treatments I am investigating. It has therefore been decided that you should continue with your present treatment and not take part in this research project. Dr. Price has been informed of this decision.

I would, however, like to thank you once again for the time spent in conversation with me and wish you the best for the future.

Yours sincerely,

M. Attfield,
Clinical Psychologist.

Appendix 1.5

UNIVERSITY DEPARTMENT OF PSYCHIATRY
(ROYAL EDINBURGH HOSPITAL)
MORNINGSIDE PARK
EDINBURGH.
EH10 5HF

TELEPHONE No. 031-447 2011

Dear

Thank you for taking the time to discuss your heaches with me. This research project is concerned with comparing different forms of treatment for migraine headache. It has been decided that you should continue the course of treatment prescribed for you by Dr. Price.

You will find 8 headache forms enclosed with this letter, please complete a form each time you have a headache. One form covers a 24 hour period, so if your migraine lasts for more than 24 hours you will need to complete 2 forms, and so on. A 'specimen' form is enclosed to refresh your memory on how to fill them in. Further forms will be supplied at monthly intervals and with each set a stamped addressed evenlope will be included to return completed forms. Please do not forget to inform me of headache free months, as this is also important information.

As the project will last for one year, I will send you a supply of forms every month for that period of time. When the study is complete, you will be offered a course of the most effective form of treatment. Finally, if you do not wish to be included in this study, please inform me.

Once again, thank you for your help.

Yours sincerely,

M. Attfield,
Clinical Psychologist.

Appendix 1.6

UNIVERSITY DEPARTMENT OF PSYCHIATRY
(ROYAL EDINBURGH HOSPITAL)
MORNINGSIDE PARK
EDINBURGH.
EH10 5HF

TELEPHONE No. 031-447 2011

Dear

Thank you for taking time to discuss your headaches with me. This research project is concerned with comparing different forms of treatment for migraine headache. The project will last for one year and will consist of the following three stages:

Stage 1. During the first three months a record of the frequency and severity of your headaches will be kept. I will see you on two occasions; at monthly intervals, so that we can get to know each other better and so that you can become familiar with the department.

Stage 2. The second three months will be the treatment phase of the project. During this phase, I will see you for one hour per week, for ten consecutive weeks.

Stage 3. During the final six months, I will again be recording your headaches to see if there have been any changes.

At no time will I require you to stop taking prescribed or preferred medication, I will however require you to keep accurate records of the medication taken. The treatment is not painful and a minimum amount of equipment will be used. The equipment that is used will be taped to the surface of the skin and at no time will you experience anything unpleasant.

Although I will be seeing you for the treatment sessions, it is important to remember that I will be supplementing any previously prescribed medication and not replacing it. Please find enclosed eight headache forms. Complete a form each time you have a migraine, one form covers 24 hours, so if your headache lasts for more than 24 hours you will need to complete two forms, and so on. Finally if you do not wish to be included in this study, please inform me, otherwise I will be contacting you to arrange an appointment.

Yours sincerely,

M. Attfield,
Clinical Psychologist.

APPENDIX 1.7

FINGER TEMPERATURE CONTROL - WHY SHOULD IT WORK?

In the human body there is a large system of arteries, veins and smaller blood vessels. Eight pints of blood are pumped around this system by the heart. These vessels are not rigid, arteries have muscular walls which can expand or contract slightly, regulating the amount of blood that flows through them to any particular part of the body. Apart from its many other functions, blood carries warmth to all parts of the body. Now we know that the pain of a migraine headache is due to an abnormal increase in the amount of blood flowing to the head. The blood vessels in the head therefore have to stretch to allow this extra blood to flow and this stretching is thought to cause the pain. Finger temperature control is thought to work in two main ways:

Firstly, learning to increase the flow of blood to the hands by increasing one's finger temperature may decrease the amount of blood which is being directed to the head, the arteries in the head will therefore no longer need to stretch and the headache should disappear.

Secondly, we know that an increase in finger temperature is a sign of a relaxed nervous system, at least that part of the nervous system that causes blood to be unevenly distributed around the body. Learning how to raise your finger temperature will relax the nervous system causing that redistribution of the blood which should bring relief from your headache.

Of course, achieving this state will take time and practice, so try to spend at least fifteen minutes a day, every day in practice.

APPENDIX 1.7

TEMPORAL ARTERY AMPLITUDE CONTROL - WHY SHOULD IT WORK?

In the human body there is a large system of arteries, veins and smaller blood vessels. Eight pints of blood are pumped around this system by the heart. These vessels are not rigid, arteries have muscular walls which can expand or contract slightly, regulating the amount of blood that flows through them to any particular part of the body. There is a lot of evidence to show that migraine headaches are associated with an abrupt increase in blood flow to the head which is thought to stretch the arteries and cause pain. One of the major arteries of the head associated with pain during a migraine headache is the temporal artery; the one which runs up the side of your head, next to your ear. During the headache the pulsations of this artery increase.

Temporal artery amplitude control is meant to work by helping you gain some control over the size of these pulsations, making any changes a less extreme and less abrupt action, thereby stopping or at least decreasing the pain of the headache.

Of course, achieving this state will take time and practice, so try to spend at least fifteen minutes a day, every day in practice.

APPENDIX 1.7

PROGRESSIVE RELAXATION EXERCISES - WHY SHOULD THEY WORK?

In the human body there is a large system of arteries, veins and smaller blood vessels. Eight pints of blood are pumped around this system by the heart. These vessels are not rigid, arteries have muscular walls which can expand or contract slightly, regulating the amount of blood that flows through them to any particular part of the body. There is evidence to show that relaxation can affect bodily functions in many different ways. Migraine headaches are often associated with an increase in blood flow to the head and this can be caused in a number of ways. Stress and tension can often start a headache by causing an increase in blood flow to the head and at the first signs of the headache the tension may often increase in anticipation of the headache, thus causing more blood to rush to the head, more headache signs and consequently a headache will result. I am not saying that tension is the only cause of your headaches but it probably plays quite an important role in its appearance.

Progressive relaxation exercises can help you in two main ways:

Firstly, if you can teach yourself to feel mentally and physically tranquil during times of stress and these times include the periods either before or during a headache, then you will be able to prevent further tension mounting and eventually bring about relief from your headache.

Secondly, as stated above, relaxation is also associated with many bodily changes such as a decrease in heart rate and a redistribution of the blood supply in the body so that blood will be more evenly distributed around the body during relaxation instead of being concentrated in the arteries of the head causing the headache.

Of course, achieving this state will take time and practice, so try to spend at least fifteen minutes a day, every day in practice.

APPENDIX 1.7

CONTROLLING HEART RATE - WHY SHOULD IT WORK?

In the human body there is a large system of arteries, veins and smaller blood vessels. Eight pints of blood are pumped around this system by the heart. These vessels are not rigid, arteries have muscular walls which can expand or contract slightly, regulating the amount of blood that flows through them to any particular part of the body. There is a lot of evidence to show that migraine headaches are associated with disturbances in blood flow to the head and probably other parts of the body as well. It is reasonable to suppose that the rate at which your heart beats will affect the rate at which blood flows to the head, decreasing heart rate will in effect reduce the amount of blood flowing through the arteries in the head which are stretched and cause pain during a migraine attack.

Heart rate is also an index of how anxious you feel, lower heart rate is associated with a state of relaxation and this is probably something that you don't feel when experiencing a migraine headache. So, learning to control your heart rate will hopefully enable you to achieve two things:

Firstly, it will enable you to gain some control over how much blood is carried to those arteries of the head that cause migraine headaches.

Secondly, it will help you to relax and counter those feelings of anxiety that accompany the anticipation of a migraine headache.

Of course, achieving this state will take time and practice, so try to spend at least fifteen minutes a day, every day in practice.

APPENDIX 2.

PILOT STUDY, EXPERIMENT 1 (APPENDIX 2.1)

FINGER TEMPERATURE (Mean Change Per 30 Second Epoch)

CONDITION		1	2	3	4	5
Subject	1	-0.3	-1.5	-1.4	-2.1	-1.8
	2	+2.1	+3.5	+0.2	-4.0	-4.8
	3	+0.3	-0.1	-0.9	-0.8	-0.9
	4	-0.3	-3.2	-4.4	-4.7	-6.0
	5	-0.1	+0.6	-0.3	-1.7	-1.4
	6	-0.5	-0.9	-3.2	-5.6	-8.3
	7	-0.1	-3.7	-0.1	-3.6	-3.1
	8	-0.1	+0.9	-0.4	-1.6	-0.9
Means		+0.1	-0.5	-1.3	-3.0	-3.5

TEMPORAL ARTERY AMPLITUDE (Mean Per 30 Second Epoch)

CONDITION		1	2	3	4	5
Subject	1	6.6	8.6	9.4	8.6	9.4
	2	18.1	18.4	17.7	17.1	16.9
	3	3.0	3.5	2.3	3.6	3.0
	4	13.9	16.3	14.9	4.4	6.0
	5	18.1	18.7	18.6	18.9	19.3
	6	8.7	19.3	16.8	14.4	14.3
	7	8.7	7.9	7.0	6.8	6.9
	8	16.5	17.2	16.2	16.1	5.0
Means		11.7	13.7	12.9	11.2	10.1

RESPIRATION DEPTH (Mean Per 30 Second Epoch)

CONDITION		1	2	3	4	5
Subject	1	33.5	34.3	34.2	34.2	29.9
	2	21.2	20.2	16.7	13.9	10.4
	3	16.8	16.7	18.2	18.2	18.1
	4	30.8	21.8	19.8	22.3	19.9
	5	14.2	16.3	9.1	8.7	9.2
	6	34.7	27.4	29.4	20.8	27.5
	7	38.7	38.8	31.5	25.0	29.3
	8	28.0	34.8	32.0	33.7	32.6
Means		27.2	26.3	23.9	22.1	22.1

RESPIRATION RATE (Per Minute) (APPENDIX 2.1)

CONDITION		1	2	3	4	5
Subject	1	15.7	15.5	11.7	9.2	15.5
	2	18.2	18.3	16.9	15.8	17.9
	3	18.8	17.8	14.1	15.1	15.5
	4	16.0	16.3	15.7	16.1	16.0
	5	12.7	12.1	12.3	13.1	12.1
	6	10.3	12.6	9.9	16.1	8.8
	7	16.1	13.5	15.8	17.2	15.6
	8	17.3	17.3	17.5	18.0	18.0
Means		15.6	15.4	14.2	15.1	14.9

PEARSON PRODUCT MOMENT CORRELATION COEFFICIENTS (Per 30 Second Epoch)

Temp = Finger temperature changes
T.A. = Temporal artery amplitude
R.D. = Respiration depth

SUBJECT 1

Cond	Temp/TA	Temp/RD	RD/TA
A	-0.08	+0.18	+0.29
B	+0.31	-0.12	+0.04
A	+0.04	-0.04	+0.05
C	+0.45	-0.15	+0.01
A	-0.33	+0.12	-0.03

SUBJECT 2

Cond	Temp/TA	Temp/RD	RD/TA
A	-0.05	-0.55	+0.35
B	-	-	-
A	-0.26	+0.11	-0.20
C	-.0.08	-0.21	+0.15
A	-	-	-

SUBJECT 3

Cond	Temp/TA	Temp/RD	RD/TA
A	+0.03	-0.39	-0.23
B	-0.53	-0.38	+0.27
A	+0.08	+0.10	+0.14
C	-0.27	-0.12	-0.39
A	-0.01	+0.09	+0.35

SUBJECT 4

Cond	Temp/TA	Temp/RD	RD/TA
A	-0.31	+0.24	-0.16
B	+0.11	+0.62	-0.17
A	-0.58	-0.44	+0.15
C	+0.07	+0.35	-0.08
A	-0.23	+0.33	-0.29

SUBJECT 5

Cond	Temp/TA	Temp/RD	RD/TA
A	-0.03	-0.17	-0.37
C	-0.39	-0.41	+0.16
A	-0.37	-0.42	+0.09
B	-0.23	-0.17	+0.11
A	-0.13	+0.12	+0.02

SUBJECT 6

Cond	Temp/TA	Temp/RD	RD/TA
A	-0.27	-0.08	-0.28
C	-0.10	+0.25	-0.39
A	+0.24	-0.46	+0.22
B	+0.18	-0.10	-0.08
A	+0.04	-0.05	+0.16

SUBJECT 7

Cond	Temp/TA	Temp/RD	RD/TA
A	+0.04	-0.08	-0.12
C	+0.02	-0.01	-0.02
A	-0.12	-0.52	+0.29
B	-0.19	+0.06	-0.35
A	-0.08	+0.36	+0.08

SUBJECT 8

Cond	Temp/TA	Temp/RD	RD/TA
A	+0.34	+0.16	+0.04
C	-0.10	+0.30	+0.06
A	-0.61	+0.71	-0.60
B	+0.22	+0.04	+0.20
A	-0.52	+0.30	-0.16

PILOT STUDY, EXPERIMENT 2 (APPENDIX 2.1)

Finger Temperature Changes (Mean Per Session)

(A) Progressive Relaxation

Weeks		1	2	3	4	5	6
Subject	1	-0.15	+0.50	-0.11	-0.14	-0.10	+0.34
	2	-0.39	-0.65	-0.39	-0.10	+0.20	+0.87
	3	+0.24	+0.40	-0.02	+0.15	+0.22	-0.10
	4	+0.71	+1.12	+0.36	-0.82	+1.19	+0.20
	5	+0.25	-0.11	+0.14	+0.02	+0.11	-0.10
	6	+0.06	+0.52	+0.18	+0.60	+0.04	-0.12
		+0.12	+0.29	+0.03	-0.05	+0.31	+0.18

(B) Concentration task

Weeks		1	2	3	4	5	6
Subject	1	-0.23	-0.36	-0.26	-0.32	+0.20	-0.05
	2	+0.65	+0.43	0.00	-0.13	-0.46	-0.54
	3	+0.08	+0.02	+0.03	0.00	-0.08	+0.02
	4	+0.91	-1.48	-1.53	-1.85	-1.81	-0.46
	5	+0.80	-0.04	+0.20	-0.55	-0.16	-0.19
	6	-0.12	-1.41	-0.68	-0.41	-0.30	-0.38
		+0.35	-0.47	-0.37	-0.54	-0.44	-0.26

(C) Finger Temperature Biofeedback

Weeks		1	2	3	4	5	6
Subject	1	-0.30	+0.29	+0.08	+0.01	+0.17	+0.03
	2	+0.44	+2.04	+1.29	+0.53	-0.35	+0.42
	3	+0.07	-0.31	+0.15	+0.02	+0.04	+0.09
	4	+0.78	-0.56	-0.72	-0.35	+2.39	+0.59
	5	-0.12	-0.06	+0.25	+0.04	+0.68	+0.76
	6	-0.13	-0.10	-0.43	-0.46	0.00	-0.10
		+0.22	+0.22	+0.10	-0.04	+0.49	+0.29

FINGER TEMPERATURE CONDITION (APPENDIX 2.2)

Finger temperature changes within periods of each treatment session

Patient	Period	Sessions 1	2	3	4	5	6	7	8	9	10
1	1	-09.5	+02.8	-02.2	-04.2	+10.3	+03.7	+19.0	+01.7	+01.4	+07.2
1	2	-16.2	+13.2	+02.2	+07.5	+17.2	+07.6	+50.0	+08.4	+01.5	+12.5
1	3	-12.2	+10.8	+01.6	-06.4	+12.7	+05.5	+61.4	+10.8	-07.8	+09.4
2	1	+02.2	+13.1	+00.0	-02.9	-26.3	-00.7	+02.9	+08.2	-17.2	+04.2
2	2	+05.1	+18.4	+03.5	-03.4	-31.3	-01.2	+03.6	+17.4	-36.9	-03.1
2	3	+07.8	+04.7	+00.0	+08.3	+04.4	-02.9	+02.9	+12.9	-59.8	-15.5
3	1	-07.1	-00.7	+02.6	+02.7	+02.6	+00.1	+03.1	+02.1	-00.4	-07.7
3	2	+03.1	+05.6	+05.4	+05.7	+05.0	+00.2	+05.7	+03.9	-02.7	-06.9
3	3	-03.3	+13.3	+03.7	+04.0	+05.9	-01.1	+00.4	-00.9	-07.6	+05.2
4	1	-10.6	-07.1	-03.5	+02.2	-04.0	-01.8	+14.4	+09.2	+04.5	-04.4
4	2	-13.0	-05.4	-09.3	+06.9	+07.9	-02.2	+20.2	+23.3	+08.2	-09.0
4	3	-11.0	-17.3	-13.4	+07.0	-01.8	-03.3	+14.6	+13.8	+13.3	-22.8
5	1	-00.4	+00.3	+04.7	-09.2	+01.5	-01.5	-03.0	+01.8	-00.2	+01.6
5	2	+00.7	+01.0	+06.6	-02.6	+01.5	-01.8	-02.7	-00.9	-00.8	+01.7
5	3	+01.5	-02.0	+04.7	-04.1	+02.3	-02.4	-02.1	-01.0	+01.0	+00.6
6	1	-01.8	-00.4	+00.3	+00.6	-00.3	+03.2	+02.0	-01.3	+01.2	+00.3
6	2	-02.9	+00.6	+00.0	+00.9	-00.4	+04.6	+01.6	+01.5	+02.6	+04.5
6	3	-03.8	-01.1	-00.6	+01.3	+02.1	+05.2	+02.9	+00.4	+04.2	+03.3
7	1	+00.6	+01.3	+01.6	+03.5	+00.1	+04.2	+04.8	+00.0	+00.8	+03.5
7	2	-04.6	-00.7	+02.3	+04.1	-01.9	+05.7	+06.1	-00.6	+01.0	+03.8
7	3	-11.1	-04.1	+02.6	+01.5	-05.3	+08.7	+09.3	-00.8	+01.9	+03.0
8	1	+03.8	-12.7	-00.8	-01.6	-05.5	+00.9	-05.7	+13.9	+00.9	-01.3
8	2	+01.4	-12.2	+00.6	+08.1	+01.0	+01.4	-00.1	+27.4	+00.7	+02.1
8	3	-09.0	-26.6	+01.6	+08.1	-05.7	-11.6	-01.5	+17.0	+00.9	+02.8

TEMPORAL ARTERY AMPLITUDE CONDITION (APPENDIX 2.2)

Finger temperature changes within periods of each treatment session

Patient	Period	Sessions 1	2	3	4	5	6	7	8	9	10
1	1	+00.2	+00.3	+03.2	+03.1	+00.1	-03.0	+00.0	+03.1	+01.4	+04.7
1	2	+00.6	+05.2	+06.7	+03.0	+01.0	-03.9	+00.5	+03.6	+02.8	+07.5
1	3	-00.8	+07.6	+08.8	+05.0	-00.8	+02.5	-00.2	-00.3	+02.1	+07.5
2	1	-01.1	+01.4	+00.8	+00.8	+00.9	+02.9	+00.6	+00.6	+00.5	+01.0
2	2	-00.7	+02.0	+01.6	+00.9	+02.2	+04.2	+00.2	+00.2	-01.7	+01.0
2	3	+01.1	+01.1	+01.5	+00.5	+02.4	+03.0	-00.2	+00.1	-03.1	+00.3
3	1	+00.0	+01.1	-03.7	-00.5	-06.3	-01.5	+04.2	+02.0	-07.3	-20.7
3	2	+01.5	+01.3	-12.2	+10.6	+06.7	-01.3	+02.9	+02.9	-15.2	-29.8
3	3	+01.5	+00.4	-20.1	+11.0	+08.5	-03.2	-01.5	+02.9	-20.0	-27.8
4	1	+03.9	+01.1	+03.6	+02.3	-01.2	-00.8	+01.5	+02.4	+10.4	-01.4
4	2	+05.0	+01.3	+05.0	+03.7	+00.9	-02.1	+01.5	-04.2	+13.4	+00.7
4	3	+05.8	+04.4	+03.5	+00.7	-01.7	-01.9	+00.0	-03.4	+10.3	+03.2
5	1	-12.0	+01.6	-06.4	+01.8	+00.3	+07.7	-08.3	-07.1	-00.4	-18.9
5	2	-08.4	+01.3	-15.8	+06.6	-04.5	-00.7	-13.9	-09.2	-03.8	-13.4
5	3	-06.7	+02.4	-32.7	-00.6	-12.6	-08.2	-11.2	-13.8	-09.3	-14.6
6	1	-02.5	-03.7	-01.8	-10.8	-03.3	+01.7	+01.5	-00.4	+00.3	+10.9
6	2	+02.0	-14.2	+07.1	-15.1	+01.1	+02.7	+03.5	+00.8	-10.1	+09.3
6	3	+36.9	-15.8	+36.5	-10.7	-03.6	+02.7	+04.6	+01.9	-10.4	-01.2
7	1	-19.1	+02.9	+04.0	+07.9	-06.3	+04.1	+01.7	+02.8	+05.0	-00.6
7	2	-30.0	+03.8	+03.8	+03.2	-06.7	-02.2	+01.1	+03.5	+05.0	+00.1
7	3	-32.4	+01.7	+02.4	+03.9	+03.1	+01.0	-00.3	+03.5	+05.1	-03.2
8	1	+02.0	-04.7	+01.1	-05.5	-00.5	+00.3	-13.7	+01.0	-01.7	+01.8
8	2	+02.7	+00.3	+01.6	-08.9	+00.4	+00.4	-01.8	+06.7	+00.8	-00.7
8	3	+00.7	+04.0	+00.3	-04.5	+00.1	+00.1	-03.9	+10.9	-05.6	-07.0

PROGRESSIVE RELAXATION CONDITION (APPENDIX 2.2)

Finger temperature changes within periods of each treatment session

Patients	Periods	Sessions 1	2	3	4	5	6	7	8	9	10
1	1	-03.7	-04.0	-03.3	-01.5	+00.2	-03.4	-08.1	-22.5	-13.6	-06.2
1	2	-04.6	-03.7	+02.5	-04.7	-00.3	-03.4	-15.8	-54.7	-13.5	-05.8
1	3	-05.2	-03.4	+10.6	-01.3	-01.2	-05.0	-14.5	-71.7	-16.6	-01.8
2	1	-08.5	-08.8	+00.0	+04.8	-10.7	+07.4	-07.2	-03.1	+04.7	-06.3
2	2	-10.8	-08.0	-00.7	+06.8	-07.4	+04.5	-07.4	-11.5	+02.9	-07.4
2	3	-08.3	-08.0	-01.1	+05.9	-00.3	+00.5	-12.9	-12.5	-04.4	-04.8
3	1	-04.1	-01.1	+02.1	+10.0	-01.0	+03.7	+02.9	+00.3	-10.2	+03.2
3	2	-05.8	-03.5	+01.2	+11.7	+01.1	+04.7	+05.8	-09.6	-16.6	+01.8
3	3	-09.2	-06.4	-00.1	+11.2	+03.9	+00.9	+05.9	-10.8	-19.2	-00.3
4	1	-01.5	+03.1	+04.6	+00.8	+10.2	+04.8	+02.2	-01.6	+01.1	+02.8
4	2	-01.2	+04.4	+07.2	+02.8	+13.6	+05.4	+06.4	-01.0	-00.4	+04.0
4	3	-00.2	+04.8	+06.8	+04.0	+13.8	+05.7	+09.8	+03.3	-00.3	+02.4
5	1	+14.9	-21.8	+07.5	-01.5	-02.9	-01.1	+00.1	+16.1	+01.5	+10.4
5	2	+22.8	-00.8	+13.7	+07.3	-03.3	-04.7	+05.7	+21.8	+00.3	+22.0
5	3	+23.3	+02.1	+15.3	+15.0	-10.5	-26.1	+02.7	+15.7	-04.6	+06.0
6	1	-01.5	+02.2	+02.8	+02.9	+00.9	+02.5	-00.2	-00.1	-00.8	+02.0
6	2	+00.7	+01.8	+04.2	+02.7	-00.1	-04.2	-01.2	+00.3	-01.7	+01.6
6	3	+00.0	+01.6	+02.2	+00.4	-00.5	-00.1	-03.8	-00.4	-02.6	-00.1
7	1	+00.7	-52.9	-09.8	+00.9	-04.9	+29.8	-01.0	-25.2	-09.6	-05.2
7	2	-00.2	-68.2	-15.2	+00.9	-05.8	+26.9	-00.7	-32.4	-09.0	-07.5
7	3	-00.2	-78.2	-20.1	+01.2	-06.7	+26.9	+00.5	-33.8	-08.5	-10.3

HEART RATE CONDITION (APPENDIX 2.2)

Finger temperature changes within periods of each treatment session

Patient	Period	Sessions 1	2	3	4	5	6	7	8	9	10
1	1	-15.6	-01.0	-13.7	+00.0	-15.5	+02.2	+00.2	-03.8	+01.0	-13.1
1	2	-37.1	+02.2	-25.6	-02.7	-28.6	+40.3	-01.0	-10.1	+01.6	-23.3
1	3	-55.4	-03.2	-36.5	-06.9	-40.0	+78.9	-03.0	-20.1	-00.4	-32.4
2	1	-01.8	+00.2	-00.3	-01.3	+00.7	+00.4	-01.1	+01.3	+01.8	+01.7
2	2	-00.2	+04.7	+01.9	-01.7	+03.4	+03.3	+05.7	+03.2	+02.9	+03.4
2	3	-03.2	+07.6	+02.1	-03.7	+04.7	+03.8	+06.2	+03.8	+05.2	+04.3
3	1	+02.7	+02.5	+00.7	+00.7	+01.1	+01.3	+00.6	+00.9	-00.1	+01.7
3	2	+03.1	+03.8	+00.8	-01.1	-00.8	-00.4	+00.8	-03.6	+02.4	+02.9
3	3	+04.1	+04.0	+01.0	-04.2	-05.2	-02.6	-00.2	-10.6	+03.3	+03.7
4	1	-02.8	+02.6	+01.1	+03.6	+01.2	+02.0	+04.5	-00.2	+02.9	+13.8
4	2	+02.6	+07.8	+01.9	+04.4	+02.0	+01.6	+06.0	+00.9	+04.6	+12.1
4	3	+03.1	+08.6	+02.1	+06.7	+02.0	+03.1	+07.5	-00.8	+04.2	+16.9
5	1	+01.6	-00.1	-00.7	-07.7	+02.2	+00.8	+02.8	+00.9	+00.4	+01.5
5	2	+02.0	+00.4	-08.6	-03.7	+04.7	+02.1	+02.7	+02.1	-00.1	+01.7
5	3	+02.2	+00.0	-03.6	-04.4	+06.5	+03.2	+02.2	-02.3	+00.8	-01.6
6	1	+05.7	-08.3	+12.0	-06.6	-00.8	+06.7	+00.2	+03.5	+00.6	+05.5
6	2	+08.7	+03.7	+27.1	-06.2	+00.9	+22.7	+01.0	+03.8	-00.2	+31.0
6	3	+03.0	-07.0	+28.7	+00.1	-05.9	+25.1	-03.5	+05.9	+01.4	+25.6
7	1	-00.2	-00.6	-01.0	-02.6	+00.9	-00.4	+00.1	-00.2	+02.2	-02.1
7	2	+00.1	+00.3	+01.5	-00.2	+02.4	+00.3	+02.3	+00.8	+03.9	+00.5
7	3	+00.8	+00.3	+02.6	+00.4	+02.8	+01.1	+02.6	+00.8	+04.6	+04.7
8	1	-02.7	+00.5	+09.3	-02.9	-07.6	-01.2	-01.2	+00.7	+00.7	-00.2
8	2	-03.0	-02.7	+27.9	-05.8	-14.1	-01.6	-02.7	-01.8	+00.6	-00.1
8	3	-05.0	-06.7	+18.9	-08.2	-21.3	-02.2	-02.2	-01.3	+00.3	+01.0
9	1	-02.2	+01.6	+01.9	+03.4	-03.6	+00.2	+01.5	+02.9	-06.8	+03.0
9	2	+00.4	-04.9	-05.8	+04.5	-05.7	+02.5	+01.5	+03.6	-13.1	+02.0
9	3	-05.7	-08.2	-07.3	+02.5	-05.9	-02.0	+00.7	+03.8	-19.2	+02.4

FINGER TEMPERATURE (APPENDIX 2.2)

Cell Means Between Conditions, Across Sessions and Within Periods

		Conditions			
Session	Period	Finger Temp	Temp Art Amp	Prog Rel'n	Heart Rate
1	1	-2.85	-3.55	-0.53	-1.70
	2	-3.30	-3.45	+0.13	-2.60
	3	-5.14	+0.59	+0.03	-6.23
2	1	-0.43	0.00	-11.79	-0.29
	2	+2.56	+0.13	-11.14	+1.70
	3	-2.79	+0.73	-12.50	-0.51
3	1	+0.34	+0.10	+0.56	+1.03
	2	+1.41	-0.28	+1.84	+2.34
	3	+0.03	+0.03	+1.94	+0.89
4	1	-1.11	-0.11	+2.34	-1.49
	2	+3.40	+0.50	+3.93	-1.39
	3	+2.46	+0.66	+5.20	-1.97
5	1	-2.70	-2.04	-1.17	-2.38
	2	-0.13	+0.14	-0.31	-3.98
	3	+1.83	-0.58	-0.21	-6.90
6	1	+1.01	+1.43	+6.24	+1.33
	2	+1.79	-0.36	+4.17	+7.87
	3	-0.74	-0.50	+0.40	+12.04
7	1	+4.69	-1.56	-1.61	+0.84
	2	+10.55	-0.75	-1.03	+1.81
	3	+10.99	-1.59	-1.76	+1.14
8	1	+4.45	+0.55	-5.16	+0.67
	2	+10.05	+0.54	-3.19	-0.12
	3	+6.53	+0.23	-15.74	-2.31
9	1	-1.13	+1.03	-3.84	+0.30
	2	-3.30	-1.10	-5.43	+0.29
	3	-6.74	-3.86	-8.03	+0.02
10	1	+0.43	-2.90	+0.10	+1.31
	2	+0.70	-3.16	+1.24	+3.36
	3	-1.75	-5.35	-1.27	+2.73

FINGER TEMPERATURE (APPENDIX 2.2)

Cell Standard Deviations between Conditions, Across Sessions and Within Periods.

		Conditions			
Session	Period	Finger Temp	Temp Art Amp	Prog Rel'n	Heart Rate
1	1	5.49	7.87	7.39	5.94
	2	7.67	11.54	10.75	13.33
	3	7.08	18.87	10.98	18.80
2	1	7.48	2.71	19.99	3.27
	2	9.79	6.00	25.48	3.89
	3	13.53	7.06	29.35	6.29
3	1	2.63	3.76	5.69	7.21
	2	4.87	8.76	8.88	16.72
	3	5.70	20.37	11.37	17.99
4	1	4.28	5.71	4.06	3.95
	2	4.56	8.41	5.27	3.86
	3	5.51	6.48	5.99	4.77
5	1	10.66	2.92	6.37	5.80
	2	13.99	4.10	6.89	10.91
	3	6.12	6.09	7.77	14.98
6	1	2.39	3.45	10.99	2.29
	2	3.73	2.69	10.99	14.18
	3	5.80	3.83	15.61	26.46
7	1	8.27	6.12	4.35	1.84
	2	17.36	5.56	8.23	2.82
	3	21.14	4.54	9.20	3.89
8	1	5.33	3.31	14.29	2.09
	2	11.19	5.06	27.90	4.49
	3	7.79	7.02	29.09	8.22
9	1	6.67	5.12	7.12	2.83
	2	13.94	8.89	7.57	5.29
	3	22.46	9.66	7.22	7.49
10	1	4.83	11.12	6.26	7.06
	2	6.89	12.72	10.33	14.04
	3	11.21	11.24	5.21	15.76

FINGER TEMPERATURES (APPENDIX 2.3)

Stability point finger temperatures for all patients, between conditions and across sessions

Conditions	Patients	Sessions 1	2	3	4	5	6	7	8	9	10
Finger Temperature	1	34.0	32.5	35.6	34.7	33.4	35.1	24.9	22.1	35.0	24.5
	2	32.0	35.5	35.0	36.0	32.0	36.0	36.1	33.2	34.1	34.8
	3	35.0	35.5	35.1	36.4	35.8	35.5	34.0	33.2	34.0	34.6
	4	36.3	36.4	34.6	21.7	27.0	21.4	33.0	30.0	20.5	33.5
	5	35.2	34.8	34.6	32.8	34.4	34.4	34.1	34.2	34.1	34.6
	6	35.0	34.4	35.3	34.9	35.0	34.6	35.3	35.6	35.0	35.0
	7	32.8	32.4	35.2	34.0	34.7	32.4	35.1	33.0	34.6	35.7
	8	25.7	34.3	34.8	34.9	35.2	34.9	33.8	35.3	35.2	35.2
Temporal Artery Amplitude	1	36.5	36.5	35.3	33.0	34.5	33.5	33.9	34.8	35.2	34.9
	2	35.6	35.5	35.5	35.8	35.2	32.3	35.7	35.5	34.5	35.5
	3	36.0	36.5	36.5	36.7	37.3	36.6	36.0	32.5	33.4	35.2
	4	36.6	36.7	36.0	35.5	34.0	34.7	35.4	34.4	33.1	35.5
	5	35.2	32.8	32.8	32.8	33.3	32.2	33.0	33.0	35.1	34.8
	6	23.3	34.8	31.0	35.2	30.4	20.1	19.4	35.0	34.5	35.2
	7	28.9	34.3	35.4	34.0	33.8	34.2	34.9	34.8	35.2	35.6
	8	35.0	35.4	36.3	35.5	36.0	35.6	35.0	33.0	31.0	31.8
Progressive Relaxation	1	35.5	35.9	36.5	36.8	32.1	32.1	30.8	33.5	35.3	33.5
	2	35.8	35.2	35.7	34.5	35.5	34.0	32.7	33.8	32.4	34.5
	3	35.5	35.2	34.0	33.6	35.5	35.0	34.0	35.4	34.4	35.6
	4	36.4	32.0	32.5	32.9	32.5	34.8	34.0	34.0	35.3	35.0
	5	31.0	32.1	20.3	25.0	34.9	33.9	29.9	32.7	35.6	30.5
	6	33.4	34.4	33.7	34.7	35.2	33.9	34.8	23.1	34.0	33.7
	7	18.9	33.3	25.6	20.0	27.4	30.5	19.3	31.6	34.0	33.2
Heart Rate	1	35.4	35.6	29.5	36.0	29.7	26.3	35.9	35.4	35.7	29.7
	2	34.7	31.0	34.0	34.7	34.0	34.0	34.2	34.3	34.8	34.4
	3	33.0	33.3	30,1	35.0	35.4	34.9	35.8	33.5	35.4	35.4
	4	35.1	35.5	34.6	36.0	32.2	32.9	32.8	33.3	32.5	30.0
	5	35.4	34.9	34.6	33.9	18.8	35.4	34.4	35.0	35.5	35.9
	6	28.7	34.0	28.2	33.7	33.9	22.1	34.7	34.4	34.0	34.2
	7	35.8	36.0	35.8	34.8	35.0	35.5	35.7	35.8	35.5	35.5
	8	35.0	35.5	31.0	35.0	34.7	34.8	35.5	35.3	35.0	35.0
	9	34.0	34.6	35.0	34.3	33.5	34.4	35.4	35.0	33.0	35.3

FINGER TEMPERATURE (APPENDIX 2.3)

Stability point finger temperatures cell means and standard deviations between conditions, across sessions

CELL MEANS	Conditions			
Sessions	Finger Temperature	Temporal Artery	Progressive Relaxation	Heart Rate
1	34.44	33.86	34.33	33.24
2	35.22	32.21	31.16	33.49
3	33.25	33.39	32.36	34.12
4	34.47	35.31	34.01	34.49
5	35.02	34.85	31.19	32.53
6	33.18	34.81	31.07	34.82
7	33.44	34.31	33.30	31.91
8	33.04	32.39	33.46	32.26
9	33.29	32.91	30.79	34.93
10	32.08	34.13	32.01	34.67

STANDARD DEVIATIONS	Conditions			
Sessions	Finger Temperature	Temporal Artery	Progressive Relaxation	Heart Rate
1	1.62	1.59	1.10	5.19
2	0.58	5.07	5.66	3.29
3	3.35	4.77	6.22	2.20
4	1.42	1.33	1.57	1.56
5	0.35	1.93	5.98	2.82
6	4.77	1.39	6.15	0.80
7	2.86	2.05	2.96	5.21
8	4.82	5.19	1.60	4.75
9	3.53	5.55	5.37	1.01
10	4.38	1.12	4.10	0.86

FINGER TEMPERATURE CONDITION (APPENDIX 2.4)

Temporal artery amplitude changes (means) within periods of each treatment session

Patient	Period	Sessions 1	2	3	4	5	6	7	8	9	10
1	1	+0.13	-0.08	-0.01	-0.15	-0.07	-0.05	-0.02	+0.11	-0.02	+0.16
1	2	+0.04	-0.09	0.00	-0.02	+0.14	+0.13	+0.02	+0.23	+0.04	+0.15
1	3	+0.08	-0.07	+0.11	+0.09	+0.14	+0.18	+0.05	+0.18	+0.18	+0.15
2	1	+0.02	+0.39	-0.04	-0.01	+0.15	-0.07	-0.22	-0.09	+0.10	+0.07
2	2	-0.14	+0.15	+0.03	+0.08	+0.14	-0.11	-0.63	+0.22	-0.01	-0.07
2	3	-0.01	+0.30	+0.10	+0.02	+0.16	-0.14	-0.84	-0.08	+0.17	-0.01
3	1	-0.23	+0.01	-0.11	+0.30	-0.28	-0.03	+0.06	-0.23	+0.25	-0.14
3	2	-0.33	+0.15	+0.01	+0.17	-0.54	-0.10	-0.25	-0.21	+0.37	-0.05
3	3	-0.29	+0.05	+0.09	+0.16	-0.55	-0.02	-0.21	-0.11	+0.50	-0.02
4	1	-0.08	+0.05	+0.02	+0.04	0.00	-0.03	+0.01	+0.15	-0.14	+0.09
4	2	+0.12	+0.11	+0.10	+0.11	+0.07	0.00	+0.05	+0.10	-0.08	+0.04
4	3	+0.04	+0.31	+0.19	+0.14	+0.22	+0.12	+0.04	-0.06	-0.08	-0.06
5	1	-0.06	+0.18	-0.48	+0.09	0.00	+0.19	+0.22	-0.53	+0.10	+0.09
5	2	-0.21	-0.03	-0.68	0.00	+0.07	+0.26	+0.31	-0.66	+0.19	+0.10
5	3	-0.48	+0.15	-0.66	-0.21	+0.14	+0.34	+0.50	-0.43	+0.33	+0.23
6	1	-0.09	-0.08	+0.14	-0.12	-0.19	-0.30	+0.02	+0.22	+0.07	+0.30
6	2	-0.68	+0.10	+0.24	+0.43	+0.03	-0.29	+0.28	+0.29	-0.69	+0.46
6	3	-0.65	+0.18	+0.35	+0.66	+0.28	-0.55	+0.49	+0.29	-0.57	+0.45
7	1	+0.23	-0.28	-0.02	+0.31	-0.03	+0.09	+0.12	-0.04	+0.17	+0.25
7	2	+0.28	-0.14	-0.01	+0.24	+0.08	+0.15	+0.20	+0.15	+0.04	+0.23
7	3	+0.29	-0.03	+0.05	+0.33	+0.20	+0.27	+0.27	+0.11	+0.14	+0.27
8	1	+0.23	+0.48	+0.27	+0.11	+0.23	+0.16	-0.14	+0.55	+0.06	+0.10
8	2	+0.11	+0.50	+0.48	+0.18	+0.40	+0.14	+0.14	+0.67	+0.09	+0.09
8	3	+0.15	+0.42	+0.55	+0.25	+0.48	+0.14	+0.29	+0.52	+0.10	+0.08

TEMPORAL ARTERY AMPLITUDE CONDITION (APPENDIX 2.4)

Temporal artery amplitude changes (means) within periods of each treatment session

Patient	Period	Sessions 1	2	3	4	5	6	7	8	9	10
1	1	0.00	-0.27	+0.03	-0.11	-0.07	-0.09	-0.17	-0.11	-0.01	+0.15
1	2	+0.02	-0.23	+0.16	-0.22	-0.10	-0.04	-0.13	-0.12	+0.26	+0.22
1	3	+0.10	-0.28	+0.44	-0.11	-0.24	-0.02	-0.11	-0.12	+0.19	+0.13
2	1	+0.08	-0.01	-0.07	+0.03	-0.25	-0.05	-0.06	-0.01	-0.05	-0.17
2	2	0.00	-0.03	-0.15	+0.04	-0.13	-0.09	-0.09	+0.01	-0.07	-0.33
2	3	+0.16	-0.14	-0.37	+0.01	+0.07	-0.01	-0.08	+0.07	+0.01	-0.25
3	1	+0.12	-0.14	+0.10	+0.07	+0.02	+0.09	+0.01	+0.31	+0.01	+0.04
3	2	+0.23	0.00	+0.35	+0.41	+0.49	+0.14	+0.11	+0.57	+0.01	+0.09
3	3	+0.28	+0.03	+0.48	+0.53	+0.73	+0.31	+0.22	+0.74	+0.24	+0.02
4	1	-0.31	+0.11	+0.16	+0.14	-0.10	0.00	-0.09	+0.18	-0.03	-0.21
4	2	-0.42	+0.27	+0.24	+0.29	+0.14	+0.06	-0.05	+0.12	-0.16	-0.27
4	3	-0.38	+0.35	+0.34	+0.30	+0.30	+0.15	-0.03	0.00	-0.12	-0.12
5	1	-0.29	-0.09	+0.22	+0.13	-0.05	+0.14	-0.01	+0.19	-0.14	-0.05
5	2	+0.11	-0.08	+0.36	+0.15	0.00	+0.05	+0.07	+0.05	-0.15	+0.06
5	3	+0.32	+0.27	+0.26	+0.38	-0.01	+0.41	+0.32	+0.27	-0.10	+0.13
6	1	+0.23	+0.28	+0.19	+0.17	+0.17	+0.29	+0.19	+0.09	+0.05	+0.22
6	2	+0.31	+0.18	+0.39	+0.40	+0.26	+0.39	+0.20	+0.21	+0.22	+0.27
6	3	+0.37	+0.17	+0.48	+0.44	+0.23	+0.68	+0.31	+0.17	+0.30	+0.35
7	1	+0.12	-0.04	+0.07	+0.12	+0.06	+0.03	+0.08	-0.10	0.00	-0.06
7	2	+0.21	+0.33	+0.15	+0.14	+0.05	+0.04	+0.06	+0.06	+0.23	+0.12
7	3	+0.37	+0.38	+0.23	+0.32	+0.19	-0.03	+0.36	+0.20	+0.44	+0.21
8	1	+0.09	+0.22	+0.30	+0.22	+0.09	+0.35	-0.23	-0.14	+0.35	+0.14
8	2	+0.31	+0.59	+0.59	+0.48	+0.42	+0.33	+0.12	-0.01	+0.13	+0.27
8	3	+0.47	+0.77	+0.69	+0.55	+0.58	+0.54	+0.07	+0.23	+0.22	+0.42

PROGRESSIVE RELAXATION CONDITION (APPENDIX 2.4)

Temporal artery amplitude changes (means) within periods of each treatment session

Patient	Period	Sessions 1	2	3	4	5	6	7	8	9	10
1	1	+0.03	+0.23	+0.13	+0.39	+0.40	+0.16	+0.18	+0.18	+0.12	-0.13
1	2	+0.09	+0.29	+0.22	+0.48	+0.56	+0.28	+0.35	+0.19	+0.19	-0.16
1	3	+0.33	+0.35	+0.27	+0.59	+0.65	+0.32	+0.34	+0.12	+0.26	-0.26
2	1	+0.35	+0.36	+0.24	+0.09	-0.06	+0.22	-0.01	+0.25	-0.27	+0.13
2	2	+0.10	+0.12	+0.35	+0.20	+0.10	+0.51	+0.23	+0.45	-0.36	+0.46
2	3	+0.22	+0.22	+0.32	+0.03	+0.26	+0.38	+0.46	+0.56	-0.35	+0.68
3	1	+0.38	+0.13	+0.06	-0.06	-0.05	+0.03	+0.08	+0.17	+0.13	+0.05
3	2	+0.37	+0.33	+0.27	-0.12	+0.21	+0.16	+0.19	+0.29	+0.18	+0.17
3	3	+0.35	+0.36	+0.34	+0.01	+0.07	+0.18	+0.31	+0.40	+0.17	+0.24
4	1	-0.11	+0.10	-0.15	-0.21	+0.01	-0.75	+0.02	+0.03	0.00	+0.03
4	2	+0.02	+0.17	-0.04	-0.12	+0.07	-0.68	+0.06	-0.06	+0.02	+0.03
4	3	+0.20	+0.22	-0.02	+0.07	+0.21	-0.64	+0.14	-0.12	+0.18	+0.06
5	1	-0.10	-0.04	-0.05	+0.98	+0.11	+0.02	-0.13	0.00	+0.15	+0.24
5	2	-0.11	+0.07	-0.01	+1.03	+0.17	+0.10	-0.05	-0.03	+0.13	+0.27
5	3	-0.09	+0.20	+0.08	+1.16	+0.11	-0.02	-0.26	+0.09	+0.08	+0.10
6	1	-0.10	-0.18	+0.67	+0.27	-0.33	-0.11	-0.03	+0.16	+0.51	-0.23
6	2	-0.10	-0.42	+0.77	+0.12	-0.07	-0.04	-0.07	+0.07	+0.76	-0.29
6	3	-0.18	-0.45	+0.67	+0.19	-0.09	+0.18	+0.12	+0.16	+0.96	-0.39
7	1	-0.21	+0.24	+0.18	+0.09	+0.33	+0.20	+0.25	+0.37	+0.10	+0.16
7	2	-0.07	+0.47	+0.33	+0.22	+0.45	+0.16	+0.54	+0.65	+0.23	+0.37
7	3	+0.06	+0.59	+0.51	+0.47	+0.77	+0.37	+0.71	+0.81	+0.45	+0.41

HEART RATE CONDITION (APPENDIX 2.4)

Temporal artery amplitude changes (means) within periods of each treatment session

Patient	Period	1	2	3	4	5	6	7	8	9	10
1	1	+0.13	-0.12	+0.05	+0.22	+0.18	+0.02	-0.05	-0.13	+0.17	-0.19
1	2	+0.25	-0.18	+0.21	+0.45	+0.33	+0.05	+0.20	-0.23	+0.14	+0.06
1	3	+0.41	-0.03	+0.30	+0.55	+0.37	+0.20	+0.13	-0.25	+0.19	+0.09
2	1	+0.05	+0.15	+0.02	-0.02	+0.07	+0.12	+0.07	-0.07	+0.23	+0.01
2	2	+0.12	+0.18	+0.02	+0.19	+0.24	+0.17	+0.14	+0.11	+0.36	+0.17
2	3	+0.21	+0.38	+0.09	+0.36	+0.13	+0.15	+0.13	+0.26	+0.49	+0.20
3	1	-0.18	-0.12	+0.25	+0.03	-0.04	+0.17	0.00	-0.09	0.00	+0.08
3	2	-0.03	-0.05	+0.38	+0.24	-0.04	+0.15	+0.08	-0.19	-0.15	+0.06
3	3	+0.12	+0.02	+0.36	+0.42	-0.02	+0.34	+0.04	-0.07	+0.26	+0.07
4	1	+0.15	+0.27	+0.08	+0.27	+0.32	+0.06	+0.06	+0.01	-0.04	+0.16
4	2	+0.24	+0.08	+0.19	+0.24	+0.41	-0.05	+0.28	+0.05	+0.10	+0.18
4	3	+0.25	+0.36	+0.26	+0.39	+0.54	-0.05	+0.36	+0.09	+0.20	+0.30
5	1	+0.19	+0.18	-0.03	+0.09	+0.01	+0.02	+0.06	+0.05	0.00	+0.16
5	2	+0.23	+0.90	+0.03	+0.21	+0.06	+0.04	+0.25	+0.16	-0.13	+0.32
5	3	+0.27	+0.22	+0.07	+0.26	+0.12	+0.23	+0.29	+0.24	+0.05	+0.40
6	1	+0.20	+0.29	+0.34	+0.22	+0.31	+0.19	-0.06	-0.03	-0.09	+0.06
6	2	+0.24	+0.25	+0.14	+0.18	+0.40	+0.22	-0.16	+0.04	-0.06	+0.01
6	3	+0.27	+0.40	+0.13	+0.14	+0.36	+0.26	-0.08	+0.11	-0.07	+0.03
7	1	+0.13	+0.20	+0.34	+0.26	+0.10	+0.03	+0.04	+0.11	+0.19	+0.27
7	2	+0.16	+0.22	+0.32	+0.38	+0.31	+0.17	+0.15	+0.09	+0.31	+0.28
7	3	+0.30	+0.16	+0.35	+0.29	+0.40	+0.06	+0.41	+0.20	+0.16	+0.21
8	1	+0.09	+0.08	+0.10	+0.04	+0.03	+0.10	+0.13	+0.12	+0.02	+0.05
8	2	+0.02	+0.07	+0.09	+0.05	+0.06	+0.11	+0.13	+0.13	+0.07	+0.16
8	3	+0.02	+0.19	+0.11	+0.11	+0.27	+0.27	+0.13	+0.32	+0.07	+0.22
9	1	+0.10	-0.06	-0.03	-0.03	+0.04	+0.01	+0.46	+0.09	+0.08	-0.07
9	2	+0.16	+0.10	-0.24	-0.12	+0.17	+0.05	+0.41	+0.16	+0.27	-0.08
9	3	+0.28	+0.06	-0.13	+0.16	+0.43	-0.03	+0.38	+0.08	+0.18	+0.11

TEMPORAL ARTERY AMPLITUDE (MEANS) (APPENDIX 2.4)

Cell means between conditions, across sessions and within periods.

Session	Period	Conditions: Finger Temp	Temporal Artery Amplitude	Progress Relaxation	Heart Rate
1	1	0.02	0.01	0.03	0.09
1	2	0.08	0.01	0.12	0.09
1	3	-0.03	0.13	0.15	0.12
2	1	0.07	0.09	0.22	0.12
2	2	-0.02	-0.02	0.06	0.11
2	3	-0.01	0.09	-0.03	0.08
3	1	0.01	-0.04	0.05	0.08
3	2	0.02	0.05	0.17	0.01
3	3	0.07	0.02	0.10	0.06
4	1	0.12	0.01	0.04	0.05
4	2	-0.10	0.09	0.04	0.15
4	3	0.09	0.13	0.15	0.51
5	1	0.02	0.26	0.27	0.13
5	2	0.15	0.21	0.26	0.20
5	3	0.05	0.14	0.21	0.26
6	1	0.02	0.11	0.07	0.10
6	2	0.02	0.04	0.18	0.16
6	3	0.09	0.11	0.22	0.04
7	1	-0.01	0.06	0.16	0.10
7	2	0.12	0.05	0.12	0.13
7	3	-0.11	0.21	0.13	0.24
8	1	0.16	0.19	0.21	0.19
8	2	0.09	0.32	0.31	0.17
8	3	0.18	0.30	0.36	0.29
9	1	0.13	0.23	0.28	0.29
9	2	0.04	0.25	0.11	0.16
9	3	0.07	0.13	0.26	0.19
10	1	0.05	0.19	0.29	0.11
10	2	0.09	0.15	0.25	0.17
10	3	0.13	0.11	0.12	0.18

TEMPORAL ARTERY AMPLITUDE (MEANS) (APPENDIX 2.4)

Cell standard deviations between conditions, across sessions and within periods.

Session	Period	Conditions: Finger Temp	Temporal Artery Amplitude	Progress Relaxation	Heart Rate
1	1	0.17	0.19	0.24	0.11
1	2	0.25	0.19	0.18	0.16
1	3	0.22	0.12	0.26	0.15
2	1	0.17	0.10	0.39	0.12
2	2	0.17	0.13	0.25	0.13
2	3	0.16	0.16	0.34	0.07
3	1	0.14	0.13	0.13	0.16
3	2	0.32	0.17	0.13	0.09
3	3	0.12	0.14	0.23	0.11
4	1	0.13	0.16	0.17	0.14
4	2	0.31	0.24	0.17	0.10
4	3	0.19	0.26	0.28	1.28
5	1	0.33	0.22	0.27	0.18
5	2	0.14	0.23	0.39	0.17
5	3	0.26	0.23	0.22	0.16
6	1	0.18	0.17	0.37	0.09
6	2	0.32	0.11	0.22	0.16
6	3	0.39	0.21	0.26	0.15
7	1	0.31	0.17	0.33	0.19
7	2	0.17	0.23	0.28	0.13
7	3	0.33	0.27	0.20	0.11
8	1	0.17	0.33	0.32	0.16
8	2	0.35	0.31	0.24	0.16
8	3	0.25	0.24	0.42	0.15
9	1	0.29	0.31	0.31	0.18
9	2	0.28	0.27	0.36	0.14
9	3	0.44	0.19	0.30	0.17
10	1	0.29	0.26	0.32	0.18
10	2	0.32	0.19	0.39	0.16
10	3	0.17	0.23	0.36	0.12

FINGER TEMPERATURE CONDITION (APPENDIX 2.5)

Temporal artery amplitude changes (standard deviations) within periods of each treatment session

Patient	Period	Sessions 1	2	3	4	5	6	7	8	9	10
1	1	+0.03	-0.57	+0.05	-0.48	-0.37	-0.26	-0.16	-0.28	-0.01	+0.02
1	2	+0.03	-0.42	-0.25	+0.22	+0.51	-0.58	+0.12	-0.43	+0.06	+0.28
1	3	+0.12	-0.48	-0.14	+0.35	+0.07	-0.34	-0.10	+0.04	+0.16	+0.28
2	1	+0.32	+0.72	-0.11	-0.52	-0.46	-0.28	-0.09	-0.07	+0.31	-0.55
2	2	-0.25	+0.01	+0.03	-0.27	-0.29	-0.43	-0.31	+0.35	+0.13	-0.69
2	3	-0.40	+0.19	-0.22	-0.10	-0.22	-0.07	-0.71	+0.09	+0.21	-0.37
3	1	+1.28	+0.02	+0.24	+0.39	+0.22	+0.27	-0.10	-0.13	+0.10	+0.36
3	2	+1.31	-0.01	+0.15	+0.18	-0.10	+0.14	-0.65	-0.31	+0.27	+0.26
3	3	+1.27	+0.23	+0.06	+0.08	-0.56	+0.11	-0.41	+0.05	+0.04	+0.00
4	1	+0.08	-0.04	+0.03	-0.56	+0.03	-0.20	-0.17	+0.13	-0.13	+0.08
4	2	+0.33	+0.30	+0.08	-0.10	+0.13	-0.03	-0.22	+0.57	-0.41	-0.04
4	3	+0.46	+0.46	+0.20	-0.19	+0.39	+0.07	-0.26	+0.22	-0.28	-0.09
5	1	+0.08	+0.01	-1.06	-0.13	-0.19	+0.07	+0.59	-0.33	+0.13	-0.05
5	2	-0.24	-0.03	-1.02	-0.17	-0.40	+0.07	+0.37	-0.12	+0.15	+0.05
5	3	-0.09	+0.35	-1.16	-0.24	-0.54	-0.02	+0.52	-0.31	+0.50	+0.01
6	1	-0.93	+0.10	+0.35	-0.37	+0.17	-0.82	+0.29	+0.17	+0.34	+0.10
6	2	-0.84	+0.30	+0.94	+0.45	+0.32	-0.46	+0.37	-0.03	-0.67	+0.05
6	3	-0.66	+0.62	+0.79	-0.43	+0.24	-0.28	+0.49	+0.28	-0.07	+0.59
7	1	+0.14	+0.04	+0.16	+0.22	-0.35	-0.01	-0.39	-0.11	+0.39	+0.03
7	2	-0.08	+0.30	+0.02	+0.40	+0.24	+0.41	+0.08	-0.08	+0.24	-0.52
7	3	+0.22	-0.31	+0.44	+0.53	+0.11	+0.42	-0.14	+0.25	+0.30	+0.02
8	1	+0.34	+0.81	+0.48	-0.08	+0.58	+0.56	+0.02	+0.47	+0.34	+0.25
8	2	+0.20	+0.07	+0.79	-0.11	+0.49	+0.22	+0.14	+0.45	+0.53	+0.31
8	3	-0.40	+0.35	+0.96	-0.13	+0.64	+0.42	+0.35	+0.25	+0.13	+0.40

TEMPORAL ARTERY AMPLITUDE CONDITION (APPENDIX 2.5)

Temporal artery amplitude changes (standard deviations) within periods of each treatment session.

Patient	Period	Sessions 1	2	3	4	5	6	7	8	9	10
1	1	+0.06	+0.19	+0.23	-0.07	-0.52	+0.12	-0.16	+0.08	+0.09	+0.29
1	2	-0.03	+0.19	-0.03	-0.51	-0.28	+0.22	-0.21	-0.08	+0.84	+0.14
1	3	+0.01	+0.10	+1.07	-0.56	-0.23	+0.16	-0.32	-0.15	+0.38	-0.06
2	1	+0.36	+0.10	-0.27	-0.04	-0.04	-0.59	-0.34	-0.10	+0.25	+0.09
2	2	-0.12	-0.17	-0.33	-0.29	-0.02	-0.44	-0.12	+0.15	+0.06	+0.04
2	3	+0.45	-0.06	+0.02	+0.11	+0.21	-0.19	-0.51	-0.05	-0.28	+0.11
3	1	+0.17	-0.25	-0.18	+0.39	+0.08	+0.35	-0.22	+0.64	+0.40	+0.02
3	2	+0.39	+0.08	+0.56	+0.36	+0.55	-0.06	+0.44	+0.62	+0.43	-0.04
3	3	+0.40	-0.33	+0.08	-0.70	+0.56	+0.49	+0.40	+0.61	+1.10	+0.14
4	1	-0.64	+0.18	-0.32	-0.06	-0.18	-0.42	-0.01	-0.21	+0.03	-0.11
4	2	-0.97	-0.05	-0.20	+0.08	-0.37	-0.34	+0.01	+0.09	-0.17	-0.11
4	3	-0.95	-0.01	-0.47	+0.38	-1.18	+0.10	+0.16	-0.08	-0.16	+0.01
5	1	-0.50	-0.37	+0.17	+0.49	-0.32	-0.07	-0.14	-0.11	-0.39	-0.37
5	2	+0.26	+0.07	+0.40	+0.22	-0.37	+0.09	+0.14	-0.03	-0.03	+0.12
5	3	+0.42	+0.35	+0.40	+0.74	-0.13	+0.05	+0.19	+0.36	-0.27	-0.21
6	1	+0.15	-0.24	+0.21	+0.25	+0.64	-0.20	+0.02	-0.03	-0.15	-0.49
6	2	-0.22	+0.28	+0.07	-0.21	+0.37	-0.05	-0.23	-0.56	+0.20	-0.26
6	3	-0.26	-0.19	-0.35	-0.16	+0.52	-0.51	+0.38	+0.09	+0.18	-0.13
7	1	-0.36	+0.07	-0.04	+0.19	+0.26	-0.17	+0.01	-0.21	+0.03	+0.09
7	2	+0.02	+0.79	-0.33	+0.85	+0.00	-0.46	+0.17	+0.25	+0.11	+0.16
7	3	+0.26	+0.74	-0.24	+0.29	+0.14	+0.24	+0.64	+0.31	+0.11	+0.19
8	1	-0.10	+0.38	+0.19	+0.35	+0.32	+0.26	+0.10	+0.46	+0.12	-0.18
8	2	+0.46	+0.41	+0.40	+0.23	+0.57	+0.30	+0.35	+0.74	-0.07	+0.07
8	3	+0.29	+0.55	+0.29	+0.05	+0.54	+0.56	+0.30	+0.76	-0.02	+0.24

PROGRESSIVE RELAXATION CONDITION (APPENDIX 2.5)

Temporal artery amplitude changes (standard deviations) within periods of each treatment session

Patient	Period	Sessions 1	2	3	4	5	6	7	8	9	10
1	1	-0.03	+0.30	+0.18	+0.09	+0.11	-0.09	+0.41	-0.20	+0.35	-0.02
1	2	-0.12	+0.51	+0.08	+0.35	+0.36	+0.30	+0.36	-0.06	+0.55	-0.04
1	3	+0.26	+0.37	+0.15	+0.40	+0.43	-0.03	+0.40	-0.07	+0.48	+0.14
2	1	+0.32	+0.33	-0.41	+0.24	-0.34	+0.79	+0.25	-0.10	+0.13	+0.54
2	2	-0.29	-0.34	-0.37	+0.06	+0.02	+0.99	+0.31	+0.15	+0.18	+0.67
2	3	-0.30	-0.39	-0.42	+0.06	+0.11	+1.01	+0.39	-0.04	+0.10	+0.89
3	1	+0.00	-0.04	+0.33	-0.15	+0.17	+0.34	+0.35	+0.05	-0.35	+0.25
3	2	-0.29	+0.26	+0.65	-0.10	+0.53	+0.28	+0.73	+0.06	-0.24	+0.47
3	3	-0.72	-0.39	+0.45	+0.10	+0.55	+0.53	+0.71	+0.11	-0.30	-0.54
4	1	+0.09	+0.41	+0.13	-0.18	-0.04	+0.09	+0.33	-0.10	+0.52	+0.19
4	2	+0.52	+0.30	+0.25	-0.07	-0.06	-0.10	+0.51	-0.07	+0.40	+0.36
4	3	+0.47	+0.80	+0.27	+0.16	+0.07	+0.20	+0.29	-0.11	+0.52	+0.67
5	1	+0.03	-0.25	-0.24	+0.77	+0.02	+0.32	-0.06	+0.30	+0.11	-0.07
5	2	+0.00	-0.03	+0.44	+0.61	-0.47	+0.70	+0.75	+0.40	+0.38	+0.23
5	3	-0.21	-0.28	+0.93	+0.91	-0.52	+0.30	+0.37	+0.28	+0.03	-0.40
6	1	-0.52	-1.65	+0.69	+0.78	+0.31	+0.80	-0.34	+0.22	+0.33	-0.25
6	2	+0.02	-1.21	+0.28	+0.59	+0.70	+0.51	+0.53	-0.11	+0.66	-0.41
6	3	-0.28	-0.90	+0.45	+0.35	+0.06	+0.38	+0.68	+0.02	+1.02	-0.26
7	1	-0.52	+0.17	+0.39	-0.27	+0.01	+0.03	+0.11	+0.61	-0.23	-0.18
7	2	-0.35	+0.58	+0.49	-0.33	-0.07	-0.01	+0.49	+0.90	+0.05	-0.18
7	3	-0.26	+0.49	+0.39	-0.01	+0.58	+0.33	+0.50	+0.97	+0.07	+0.18

HEART RATE CONDITION (APPENDIX 2.5)

Temporal artery amplitude changes (standard deviations) within periods of each treatment session.

		Sessions									
Patient	Period	1	2	3	4	5	6	7	8	9	10
1	1	+0.20	-0.13	-0.41	+0.32	-0.14	-0.28	-0.23	-0.51	+0.12	-0.17
1	2	+0.41	-0.29	-0.21	+0.43	-0.26	+0.22	-0.02	-0.59	-0.03	-0.06
1	3	-0.41	-0.45	-0.48	+0.43	-0.40	-0.23	-0.03	-0.82	+0.14	+0.35
2	1	+0.44	-0.01	+0.27	-0.09	-0.13	+0.07	-0.22	-0.02	+0.29	+0.07
2	2	+0.22	+0.04	+0.46	-0.12	-0.39	+0.37	-0.15	-0.05	+0.06	+0.27
2	3	-0.01	+0.50	-0.08	+0.02	+0.01	+0.46	+0.16	+0.02	+0.06	+0.42
3	1	-0.58	+0.32	+0.51	-0.11	+0.06	+0.16	+0.22	+0.42	-0.04	+0.11
3	2	-0.23	+0.47	+0.57	+0.29	-0.10	+0.12	+0.15	+0.23	-0.43	+0.07
3	3	-0.30	+0.33	+0.42	+0.17	+0.31	+0.44	-0.07	+0.77	+0.75	-0.11
4	1	+0.24	+0.15	+0.37	+0.72	+0.86	-0.27	-0.28	-0.56	-0.28	+0.65
4	2	+0.23	-0.78	+0.37	+0.44	+0.15	-0.44	+0.05	-0.03	-0.05	+0.34
4	3	+0.14	-0.69	+0.21	+0.30	+0.06	-0.82	+0.17	-0.08	-0.25	+0.36
5	1	+0.38	+0.25	+0.09	-0.18	-0.27	+0.63	+0.10	-0.04	+0.23	+0.26
5	2	+0.37	+0.47	+0.24	+0.05	-0.47	+0.46	+0.07	-0.13	+0.05	+0.66
5	3	+0.27	+0.38	-0.03	-0.09	+0.19	+0.57	+0.11	+0.25	+0.25	+0.39
6	1	-0.08	+0.42	+0.55	+0.54	+0.03	+0.26	-0.04	-0.25	-0.39	+0.03
6	2	+0.32	+0.38	+0.03	+0.15	+0.23	+0.11	-0.27	-0.70	-0.21	+0.12
6	3	+0.12	+0.48	-0.11	+0.20	+0.18	+0.30	-0.04	-0.28	-0.41	-0.40
7	1	+0.10	-0.13	+0.41	+0.28	+0.06	-0.09	-0.39	+0.08	+0.26	-0.18
7	2	-0.22	-0.07	+0.25	-0.82	+0.07	+0.13	-0.10	+0.05	+0.26	-0.21
7	3	-0.05	-0.54	+0.28	+0.25	+0.31	-0.01	-0.38	+0.09	-0.07	-0.57
8	1	-0.03	-0.04	+0.50	+0.04	-0.11	+0.08	+0.32	+0.13	+0.65	+0.31
8	2	+0.19	-0.05	+0.28	-0.07	-0.04	+0.47	+0.08	+0.03	+0.43	+0.43
8	3	+0.46	+0.02	+0.27	+0.30	+0.17	+0.54	+0.10	+0.23	+0.28	+0.34
9	1	+0.05	+0.45	-0.14	-0.39	+0.27	-0.21	+0.11	-0.10	+0.06	-0.14
9	2	+0.10	+0.82	-0.38	-0.26	+0.01	-0.02	+0.15	+0.11	+0.02	+0.05
9	3	+0.01	+0.51	+0.09	-0.02	+0.43	-0.68	+0.23	-0.10	+0.00	-0.08

TEMPORAL ARTERY AMPLITUDE (STANDARD DEVIATIONS) (APPENDIX 2.5)

Cell Means Between Conditions, Across Sessions and Within Periods

		Conditions			
Session	Period	Finger Temp	Temp Art Amp	Prog Rel'n	Heart Rate
1	1	0.17	-0.11	-0.09	0.16
	2	0.06	-0.03	-0.07	0.04
	3	0.11	0.08	-0.08	0.07
2	1	0.14	0.01	-0.10	0.02
	2	0.07	0.20	0.01	0.00
	3	0.18	0.14	-0.13	0.18
3	1	0.12	0.00	0.15	0.25
	2	0.09	0.07	0.26	0.06
	3	0.12	0.10	0.20	0.32
4	1	-0.19	0.19	0.18	0.04
	2	0.08	0.09	0.16	0.19
	3	-0.02	-0.23	0.33	0.10
5	1	-0.05	0.03	0.03	-0.10
	2	0.11	0.06	0.14	0.10
	3	0.02	0.05	0.08	0.09
6	1	-0.08	-0.09	0.33	0.18
	2	-0.08	-0.09	0.38	0.16
	3	0.04	0.11	0.30	0.08
7	1	0.00	-0.09	0.15	-0.02
	2	-0.01	0.07	0.53	0.00
	3	-0.03	0.16	0.37	-0.02
8	1	-0.02	0.07	0.11	-0.15
	2	0.05	0.15	0.18	0.02
	3	0.11	0.23	-0.05	-0.04
9	1	0.18	0.05	0.12	0.01
	2	0.04	0.17	0.28	0.09
	3	0.12	0.13	0.28	0.09
10	1	0.03	-0.08	0.07	0.20
	2	-0.04	0.02	0.16	0.09
	3	0.11	0.04	0.05	0.14

TEMPORAL ARTERY AMPLITUDE (STANDARD DEVIATIONS) (APPENDIX 2.5)

Cell Standard Deviations between Conditions, Across Sessions and Within Periods

Session	Period	Conditions: Finger Temp	Temp Art Amp	Prog Rel'n	Heart Rate
1	1	0.60	0.36	0.32	0.25
	2	0.61	0.45	0.29	0.28
	3	0.59	0.48	0.41	0.32
2	1	0.44	0.26	0.72	0.43
	2	0.24	0.29	0.62	0.49
	3	0.38	0.37	0.56	0.22
3	1	0.47	0.23	0.38	0.25
	2	0.61	0.35	0.33	0.29
	3	0.66	0.49	0.49	0.24
4	1	0.36	0.22	0.44	0.41
	2	0.27	0.43	0.36	0.17
	3	0.32	1.07	0.29	0.38
5	1	0.36	0.37	0.20	0.25
	2	0.34	0.39	0.41	0.23
	3	0.43	0.58	0.35	0.35
6	1	0.41	0.32	0.36	0.29
	2	0.36	0.29	0.39	0.48
	3	0.28	0.35	0.41	0.29
7	1	0.31	0.15	0.27	0.14
	2	0.35	0.25	0.17	0.18
	3	0.45	0.38	0.31	0.25
8	1	0.26	0.32	0.28	0.32
	2	0.37	0.41	0.36	0.46
	3	0.19	0.34	0.24	0.29
9	1	0.19	0.24	0.32	0.26
	2	0.39	0.33	0.31	0.36
	3	0.24	0.45	0.43	0.34
10	1	0.27	0.28	0.28	0.28
	2	0.37	0.14	0.38	0.39
	3	0.30	0.16	0.55	0.27

FINGER TEMPERATURE CONDITION (APPENDIX 2.6)

Heart rate changes within periods of each treatment session

Patients	Periods	1	2	3	4	5	6	7	8	9	10
1	1	+31	+00	-06	+01	+14	+10	+02	-05	-01	+08
1	2	-04	+20	+00	+16	+10	+04	-07	-23	-15	+04
1	3	-13	+16	-22	-01	+08	+00	-26	-32	-28	+04
2	1	-08	+26	-11	+04	-10	-13	-03	-02	-03	-14
2	2	-08	+02	-16	-03	-05	-21	-04	+08	-03	-23
2	3	-08	-08	+01	+04	+09	-21	-04	-13	-05	-34
3	1	-05	-05	-06	-02	-06	-09	+00	+10	-14	-08
3	2	+16	+16	+02	+17	+05	+04	-17	+04	-10	+00
3	3	+12	+12	+12	-03	+07	-11	-16	+00	-04	+02
4	1	+01	-10	-02	-08	-01	-04	-10	-13	-03	-09
4	2	-09	-04	-01	-05	-05	-01	-17	-11	-05	-09
4	3	-07	-16	-01	+02	-19	-01	-19	-05	-13	-09
5	1	+10	+08	+00	+26	+07	+14	-01	+08	+05	+05
5	2	+08	+04	-02	+23	+07	+10	-11	-14	-08	-14
5	3	+08	+04	-05	+20	+06	+02	-15	-23	-15	-16
6	1	+05	+04	+05	+01	-12	-02	-05	+05	+03	+04
6	2	+03	+04	-02	+01	-16	-11	+00	+00	-09	+06
6	3	+01	-06	+00	-03	-20	-12	+01	+03	-11	+12
7	1	+00	+05	+17	-17	+10	+09	-08	-08	-14	-01
7	2	-02	+02	+18	-14	+00	-08	-13	-03	-01	+01
7	3	-08	+04	+14	-13	-12	-11	-27	-14	-07	-05
8	1	+08	+52	+10	+03	-05	+41	+51	+26	+40	+44
8	2	+01	+39	+13	+03	-07	+31	+46	+42	+54	+19
8	3	+01	+22	+10	-02	-15	+10	+33	+39	+25	+15

TEMPORAL ARTERY AMPLITUDE CONDITION (APPENDIX 2.6)

Heart rate changes within periods of each treatment session

Patients	Period	1	2	3	4	5	6	7	8	9	10
1	1	-86	-18	-20	-07	-08	-11	-09	+12	-05	-06
1	2	-85	-24	-30	-05	-15	-18	-08	+02	+04	-04
1	3	-89	-22	-18	+48	-17	-13	-05	-05	-05	-04
2	1	-09	-11	-09	+03	+07	-08	-02	-08	+07	-18
2	2	-03	-09	-03	+09	+17	-08	-02	-08	+03	-23
2	3	-08	-19	-08	+07	+24	-08	-04	-08	-09	-23
3	1	-02	-02	-15	-11	-09	-07	-02	-01	-11	-19
3	2	-03	-06	-27	-13	+01	-07	-21	-03	-16	-22
3	3	+07	-01	-27	-19	-01	-11	-28	+08	-13	-22
4	1	-05	-09	+07	+12	-04	-02	-03	-03	-03	+00
4	2	+01	-09	+00	+09	-17	-02	+02	-06	-09	+02
4	3	+03	-06	-05	+09	-16	+00	-09	-07	-06	-02
5	1	+02	+05	-02	-06	-03	-04	-07	+00	-04	-04
5	2	+13	+09	-09	-03	+04	-03	-02	+12	+01	-02
5	3	+15	+06	-05	+00	+10	+02	-07	+14	-01	+06
6	1	-08	-04	+02	-14	-15	-03	-11	-19	-10	-12
6	2	-13	-08	-03	-16	-14	-05	-10	-14	-03	-13
6	3	-18	-11	+00	-19	-15	-04	-12	-18	-15	-15
7	1	-06	-07	-17	-02	+03	-01	+10	-11	-02	-09
7	2	-18	-07	-12	-05	+00	-01	-05	-14	-18	-15
7	3	-24	-10	-08	-14	+04	-08	-07	-18	-26	-19
8	1	-09	+05	-16	-23	-15	-18	-27	-11	-03	+04
8	2	-13	-04	-47	-32	-32	-23	-16	-30	-26	+03
8	3	-14	-04	-17	-51	-40	-13	-25	-62	-22	-04

PROGRESSIVE RELAXATION CONDITION (APPENDIX 2.6)

Heart rate changes within periods of each treatment session

Patients	Period	1	2	3	4	5	6	7	8	9	10
1	1	+30	-14	-28	-63	-50	+00	-20	-30	-25	-35
1	2	+40	-28	-05	-73	-56	-52	-25	-19	-27	-33
1	3	+40	-18	+00	-33	-60	+27	+03	-19	-07	-49
2	1	+08	-08	-45	-13	-16	-26	-15	-26	+00	-40
2	2	+20	+02	-09	-11	-18	-28	-16	-27	+00	-33
2	3	+20	-04	-34	+02	-24	-34	-29	-27	-03	-15
3	1	+04	-26	-07	-13	-28	+03	-10	-18	-06	-29
3	2	-07	-30	-15	-04	-18	+02	-08	-13	-02	-21
3	3	+02	-26	-13	-04	-18	-15	-16	-20	+01	-31
4	1	+08	-20	+03	-25	-13	-04	-20	-29	-13	-03
4	2	+06	-06	+08	-21	-09	-21	-16	-20	-12	-10
4	3	+12	-23	+15	-23	-21	-17	-22	-34	-17	-17
5	1	+04	-04	+05	-08	-05	-21	+21	-08	+05	-50
5	2	-07	+03	-12	-48	-09	-07	+25	-08	+04	-37
5	3	+04	-37	-19	-49	-16	-01	+23	-04	+01	-36
6	1	+04	+00	+13	+00	+48	+55	+06	-02	-12	-09
6	2	+18	+01	+12	+00	+00	+01	+25	-10	+01	-18
6	3	+17	-14	+13	-02	-06	-03	+25	-02	+01	-16
7	1	-04	+01	-08	-13	-09	+06	-14	+01	-06	-03
7	2	-08	+05	-08	-09	-07	+05	-12	+04	-06	-09
7	3	+00	+04	-08	-01	-08	+02	-12	+00	-03	-12

HEART RATE CONDITION (APPENDIX 2.6)

Heart rate changes within periods at each treatment session.

Patients	Periods	Sessions 1	2	3	4	5	6	7	8	9	10
1	1	-08	-09	-03	-16	-06	-19	-05	-30	-32	-47
1	2	-23	-21	-16	-24	-10	+04	-29	-13	-35	-39
1	3	-40	-38	-15	-32	-19	-20	-24	-25	-61	-53
2	1	-04	-03	-07	+00	+03	-02	-05	-01	-05	+00
2	2	-12	+00	-10	+05	-05	-01	-06	-01	+00	-01
2	3	+02	+02	-20	-01	-02	-04	-09	+08	-08	+04
3	1	-09	-12	-15	-19	-03	-02	-05	+07	-07	+04
3	2	-15	-15	-24	-21	-01	+01	+01	+09	-11	-04
3	3	-07	+08	-29	-23	-11	+06	-08	-05	-14	-07
4	1	-19	-09	-14	-02	-02	+00	-05	-12	-03	+05
4	2	-03	-11	-12	-29	+03	-10	-09	-30	-07	+00
4	3	-02	-32	-13	-34	-06	-22	-21	-36	-08	+00
5	1	-06	-21	-33	-01	-13	-30	-05	-08	+15	-19
5	2	-16	-23	-43	-09	-26	-38	-23	+00	-06	-39
5	3	-06	-27	-42	-13	-19	-48	-16	-20	-01	-50
6	1	-01	-11	-06	-08	-09	+05	-11	-02	-07	-10
6	2	-12	-18	-09	-04	-16	-09	-09	-15	-02	-11
6	3	-14	-33	-20	-16	-25	-14	-19	-08	-05	-17
7	1	+01	+09	+05	+10	+01	+02	+04	+06	+00	+02
7	2	-03	+09	+02	+13	+01	+04	+05	+07	-01	+05
7	3	+02	+19	+05	+12	+01	+04	+10	+03	+01	+00
8	1	-14	+00	+14	-15	-10	-11	-08	-17	-15	-12
8	2	-30	-20	+04	-18	-22	-14	-23	-30	-35	-15
8	3	-28	-24	-13	-20	-20	-27	-22	-29	-36	-20
9	1	-11	-08	-04	+04	+18	-09	-24	-06	-01	-17
9	2	+04	-20	-27	-05	+27	+00	-34	-12	+06	-13
9	3	-07	+02	-14	+13	+17	+09	-24	-13	-08	-01

HEART RATE (APPENDIX 2.6)

Cell Means between Conditions, Across Sessions and Within Periods

		Conditions			
Session	Period	Finger temp	Temp Art Amp	Prog Rel'n	Heart Rate
1	1	5.25	-15.38	7.71	-7.89
	2	0.63	-15.13	8.86	-12.22
	3	-1.75	-16.00	13.57	-11.11
2	1	10.00	-5.13	-10.14	-7.11
	2	10.38	-7.25	-7.57	-13.22
	3	3.50	-8.38	-16.86	-13.67
3	1	0.88	-8.75	-9.57	-7.00
	2	1.50	-16.38	-4.14	-15.00
	3	1.23	-11.00	-6.57	-17.89
4	1	1.00	-6.00	-19.29	-5.22
	2	4.75	-7.00	-23.71	-10.22
	3	0.50	-4.88	-15.71	-12.67
5	1	-0.38	-5.50	-10.43	-2.33
	2	-1.38	-7.00	-16.71	-5.44
	3	-4.50	-6.38	-21.86	-9.33
6	1	-5.75	-6.75	1.86	-7.33
	2	1.00	-8.38	-14.29	-7.00
	3	-5.50	-6.88	-5.86	-12.89
7	1	3.25	-6.38	-7.43	-7.11
	2	-2.88	-7.75	-3.86	-14.11
	3	-9.13	-12.13	-4.00	-14.78
8	1	2.63	-5.13	-16.00	-7.00
	2	0.38	-7.63	-13.29	-9.44
	3	-5.63	-12.00	-15.14	-13.89
9	1	1.63	-3.88	-8.14	-6.11
	2	0.38	-8.00	-6.00	-10.11
	3	-7.25	-12.13	-3.86	-15.56
10	1	3.63	-8.00	-24.14	-10.44
	2	-2.00	-9.25	-23.00	-13.00
	3	-3.88	-10.38	-25.14	-16.00

HEART RATE (APPENDIX 2.6)

Cell Standard Deviations between Conditions, Across Sessions and Within Periods

		Conditions			
Session	Period	Finger Temp	Temp Art Amp	Prog Rel'n	Heart Rate
1	1	12.07	28.78	10.61	6.29
	2	8.38	29.87	18.13	10.53
	3	8.71	32.33	13.93	14.21
2	1	20.04	7.88	10.27	8.42
	2	13.99	8.97	15.04	10.88
	3	12.95	9.21	13.77	21.25
3	1	9.29	9.97	20.35	13.23
	2	10.31	16.63	10.22	14.71
	3	11.59	8.88	17.52	12.79
4	1	12.26	10.69	20.66	9.88
	2	12.73	13.51	26.94	13.95
	3	9.34	28.69	19.64	17.33
5	1	9.64	7.89	29.88	9.25
	2	8.57	15.34	18.44	15.87
	3	13.08	19.77	18.04	13.29
6	1	17.17	5.65	26.39	11.27
	2	15.64	7.96	20.73	13.29
	3	9.93	5.72	19.03	18.59
7	1	19.71	10.50	15.34	7.47
	2	20.64	7.72	20.38	13.63
	3	19.59	9.23	21.51	11.03
8	1	12.37	9.34	13.03	11.63
	2	19.63	12.44	10.03	14.43
	3	21.45	23.11	13.29	14.82
9	1	17.00	5.51	9.75	12.60
	2	22.10	11.08	10.63	14.90
	3	15.09	8.63	6.52	20.17
10	1	18.04	8.16	19.08	16.45
	2	13.03	10.39	11.47	16.12
	3	15.98	10.76	13.83	21.68

TEMPORAL ARTERY AMPLITUDE: ACCURACY OF FEEDBACK (APPENDIX 2.7)

(Correlation Coefficients)

		Low Intensity Gain		Medium Intensity Gain		High Intensity Gain	
		Display	Amp(mms)	Display	Amp(mms)	Display	Amp(mms)
Subject 1	1	1691	7	3461	21	5592	32
	2	1917	8	3169	19	4523	27
	3	1784	7	3487	21	4828	30
	4	1734	7	3456	21	5112	31
	5	2488	9	3244	20	4753	30
	6	1938	7	2709	17	5263	30
	7	2202	8	3528	22	5579	32
	8	2033	8	3198	19	5052	29
	9	2206	8	3621	22	4707	28
	10	2066	8	2957	18	4757	30
		r = +0.904		r = 0.982		r = +0.835	
Subject 2	1	1711	7	3388	19	7924	46
	2	1784	8	3215	20	6779	39
	3	1595	6	3495	20	3271	20
	4	1674	6	3348	20	3595	22
	5	1618	6	3809	23	3462	21
	6	1764	7	4094	25	3601	22
	7	1624	7	3847	24	3551	20
	8	1606	6	3478	22	3710	22
	9	1523	6	3937	24	3318	19
	10	1668	7	3715	23	3293	20
		r = 0.782		r = +0.932		r = +0.998	
Subject 3	1	2510	13	3024	17	4925	30
	2	2516	13	2782	17	3766	25
	3	2028	10	3394	10	4144	27
	4	1970	9	5605	33	6433	36
	5	2350	12	2943	20	4035	25
	6	2987	18	2758	17	5488	31
	7	2109	10	3716	22	4188	26
	8	1.2801	50	3091	20	3979	25
	9	2990	18	3651	22	5033	30
	10	2948	18	2924	19	5205	30
		r = +0.985		r = 0.978		r = +0.987	

Correlation across all readings

r = +0.887

APPENDIX 3.

FREQUENCY OF HEADACHES (BASELINE MONTHS) (APPENDIX 3.1)

Between conditions

Conditions	Fing Temp			Temp Art			Rel'n			Heart R.		
Months	1	2	3	1	2	3	1	2	3	1	2	3
	05	00	02	06	02	11	02	01	00	07	05	04
	03	01	01	08	08	06	03	00	02	01	02	01
	03	02	04	08	02	07	02	00	08	04	02	00
	00	00	16	07	02	18	04	06	02	06	07	06
	08	07	04	05	01	04	02	02	00	03	00	04
	05	02	02	03	01	01	00	00	00	05	03	02
	00	00	03	13	04	16	00	00	00	04	02	05
	16	08	07	04	00	00				01	03	03

FREQUENCY OF HEADACHES (BASELINE MONTHS)

Means for each condition

Cell Means

Month	Treatment	Fing Temp C.	Temp Art	Prog Rel'n	Heart Rate
1	1	5.00	6.75	1.86	3.88
2	2	2.50	2.50	1.29	3.00
3	3	4.88	7.75	1.71	2.75

FREQUENCY OF HEADACHES (BASELINE MONTHS)

Standard deviations for each condition

Standard Deviations

Month	Treatment	Fing Temp C.	Temp Art	Prog Rel'n	Heart Rate
1	1	5.18	3.11	1.46	2.17
2	2	3.21	2.51	2.21	2.14
3	3	4.85	6.71	2.93	2.31

Between treatment conditions across the baseline and follow-up months

CONDITION	FING TEMP				TEMP ART AMP				PROG REL				HEART RATE				WAITING LIST			
MONTHS	1	2	3	4	1	2	3	4	1	2	3	4	1	2	3	4	1	2	3	4
	02	02	00	00	11	05	01	04	00	07	00	01	04	02	00	00	03	10	04	00
	01	00	01	00	06	08	08	05	02	02	02	04	01	00	00	00	12	08	07	05
	04	02	00	00	07	04	02	03	08	07	00	00	06	00	04	00	00	05	00	01
	16	00	00	00	18	05	04	04	02	02	03	02	04	00	00	00				
	04	01	06	03	03	00	00	03	00	00	01	00	03	00	00	01				
	02	00	00	00	01	03	01	02												
	03	00	00	00	16	07	14	05												
	07	05	02	03	00	00	02	00												

Frequency of headaches (Relative difference scores)across follow-up months

	FING TEMP			TEMP ART AMP			PROG REL			HEART RATE			WAITING LIST		
MONTHS	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3
	+0.0	+2.0	+2.0	+0.8	+1.7	+0.9	-2.0	0.0	-2.0	+0.7	+2.0	+2.0	-1.1	-0.3	+2.0
	+2.0	+0.0	+2.0	-0.3	-0.3	+0.2	0.0	0.0	-0.7	+2.0	+2.0	+2.0	+0.4	+0.5	+0.8
	+0.4	+2.0	+2.0	+0.5	+1.1	+0.8	+0.1	+2.0	+2.0	+2.0	+0.4	+2.0	-2.0	0.0	-2.0
	+2.0	+2.0	+2.0	+1.1	+1.3	+1.3	0.0	-0.4	0.0	+2.0	+2.0	+1.0			
	+1.2	-0.4	+0.3	+2.0	+2.0	0.0	0.0	-0.2	0.0						
	+2.0	+2.0	+2.0	-1.0	0.0	-0.7									
	+2.0	+2.0	+2.0	+0.8	+0.1	+1.1									
	+0.3	+1.4	+0.8	0.0	-2.0	0.0									

(APPENDIX 3.2)

FREQUENCY OF HEADACHES (APPENDIX 3.2)

Mean Changes (RD Scores) across Follow-up Months.

Cell Means

	Fing Temp	Temp A.A.	Prog Rel'n	Heart Rate	Waiting List
Month 1	1.38	0.25	-0.40	1.60	-1.00
Month 2	1.13	0.38	0.00	1.60	0.00
Month 3	1.25	0.25	0.00	1.80	0.00

Standard Deviation Changes (RD Scores) across Follow-up Months.

Standard Deviations

	Fing Temp	Temp A.A.	Prog Rel'n	Heart Rate	Waiting List
Month 1	0.92	0.89	0.89	0.89	1.00
Month 2	1.46	1.19	1.41	0.89	0.00
Month 3	1.04	0.46	1.41	0.45	2.00

INTENSITY OF HEADACHES

Between Treatment conditions across the Baseline and Follow-up Months

Conditions	FING TEMP				TEMP ART AMP				PROG REL'N				HEART RATE				WAITING LIST			
Months	1	2	3	4	1	2	3	4	1	2	3	4	1	2	3	4	1	2	3	4
	4.0	4.0	0.0	0.0	2.1	1.8	2.0	2.8	0.0	3.3	0.0	4.0	3.0	3.0	0.0	0.0	2.3	2.1	3.8	0.0
	3.0	0.0	1.0	0.0	4.0	4.3	4.3	4.2	4.5	3.0	3.5	3.3	4.0	0.0	0.0	0.0	3.3	3.3	3.0	3.2
	3.3	3.5	0.0	0.0	4.3	3.8	3.5	3.0	3.0	3.0	0.0	0.0	3.0	0.0	3.0	0.0	0.0	4.4	0.0	5.0
	3.1	0.0	0.0	0.0	2.8	3.0	2.3	2.3	3.0	3.0	3.0	3.5	3.8	0.0	0.0	0.0				
	2.5	2.0	2.3	3.0	4.3	0.0	0.0	5.0	0.0	0.0	3.0	0.0	3.3	0.0	0.0	3.0				
	4.7	0.0	0.0	4.7	3.0	4.3	4.0	3.5												
	3.0	0.0	0.0	0.0	3.6	4.0	3.9	3.2												
	3.1	3.0	3.0	3.0	0.0	0.0	3.5	0.0												

Frequency of Headaches(Relative Difference Scores)Across Follow-up Months

Conditions	FING TEMP			TEMP ART AMP			PROG REL'N			HEART RATE			WAITING LIST		
Months	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3
	+0.0	+2.0	+2.0	+0.2	+0.1	-0.3	-2.0	0.0	-2.0	0.0	+2.0	+2.0	+0.1	-0.5	+2.0
	+2.0	+1.0	+2.0	-0.1	-0.1	-0.1	+0.4	+0.3	+0.3	+2.0	+2.0	+2.0	0.0	+0.1	0.0
	-0.1	+2.0	+2.0	+0.1	+0.2	+0.4	0.0	+2.0	+2.0	+2.0	0.0	+2.0	-2.0	0.0	-2.0
	+2.0	+2.0	+2.0	-0.1	+0.2	+0.2	0.0	0.0	-0.2	+2.0	+2.0	+2.0			
	+0.2	+0.1	-0.2	+2.0	+2.0	-0.3	0.0	-2.0	0.0	+2.0	+2.0	+0.1			
	+2.0	+2.0	0.0	-0.4	-0.3	-0.2									
	+2.0	+2.0	+2.0	-0.1	-0.1	+0.1									
	0.0	0.0	0.0	0.0	-2.0	0.0									

(APPENDIX 3.3)

INTENSITY OF HEADACHES (APPENDIX 3.3)

Mean Changes (R.D. Scores) across Follow-up Months

Cell Means

	Fing Temp	Temp A.A.	Prog Rel'n	Heart Rate	Waiting List
Month 1	1.00	0.25	0.40	1.60	-0.67
Month 2	1.50	0.00	0.00	1.60	0.00
Month 3	1.25	0.00	0.80	1.60	0.00

Standard Deviation Changes (R.D. Scores) across Follow-up Months

Standard Deviations

	Fing Temp	Temp A.A.	Prog Rel'n	Heart Rate	Waiting List
Month 1	1.07	0.71	0.89	0.89	1.15
Month 2	0.93	1.07	1.41	0.89	0.00
Month 3	1.04	0.00	1.09	0.89	2.00

DURATION OF HEADACHES

Between Treatment Conditions Across the Baseline and Follow-up Months

Condition	FING TEMP				TEMP ART AMP				PROG REL'N				HEART RATE				WAITING LIST			
Months	1	2	3	4	1	2	3	4	1	2	3	4	1	2	3	4	1	2	3	4
	11.0	15.0	00.0	00.0	08.6	07.8	07.8	09.5	00.0	13.9	00.0	13.0	06.0	10.0	00.0	00.0	08.7	02.7	01.8	00.0
	05.0	00.0	01.0	00.0	15.0	12.5	17.3	14.6	20.0	21.0	22.0	17.5	24.0	00.0	00.0	00.0	04.8	04.3	03.0	02.0
	18.3	15.5	00.0	00.0	16.3	14.0	15.0	14.0	09.4	17.3	00.0	18.0	17.5	00.0	10.0	00.0	00.0	15.4	00.0	24.0
	21.7	00.0	00.0	00.0	05.2	04.2	02.0	02.8	19.0	15.5	16.0	00.0	09.3	00.0	00.0	00.0				
	07.5	06.0	04.6	05.7	07.0	00.0	00.0	17.0	00.0	00.0	24.0	00.0	11.7	00.0	00.0	09.0				
	22.0	00.0	00.0	24.0	13.0	15.7	24.0	15.5												
	10.0	00.0	00.0	00.0	13.1	22.1	17.6	15.8												
	17.9	06.0	15.5	14.7	00.0	00.0	16.5	00.0												

Duration of Headaches (Relative Difference Scores) Across Follow-up Months

Conditions	FING TEMP			TEMP ART AMP			PROG REL'N			HEART RATE			WAITING LIST		
Months	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3
	-0.3	+2.0	+2.0	+0.2	+0.2	-0.1	-2.0	0.0	-2.0	-0.8	+2.0	+2.0	+1.1	+1.3	+2.0
	+2.0	+1.3	+2.0	+0.3	-0.1	0.0	-0.1	-0.1	+0.1	+2.0	+2.0	+2.0	+0.1	+0.5	+0.8
	+0.2	+2.0	+2.0	+0.2	0.0	+0.2	-0.6	+2.0	-6.0	+2.0	+0.6	+2.0	-2.0	0.0	-2.0
	+2.0	+2.0	+2.0	+0.2	+0.9	+0.9	+0.2	+0.2	+2.0	+2.0	+2.0	+2.0			
	+0.2	+0.5	+0.3	+2.0	+2.0	-0.6	0.0	-2.0	0.0	+2.0	+2.0	+0.1			
	+2.0	+2.0	-0.1	-0.2	-0.6	-0.1									
	+2.0	+2.0	+2.0	-0.5	-0.3	-0.2									
	+1.0	+0.1	+0.2	0.0	-2.0	0.0									

(APPENDIX 3.4)

DURATION OF HEADACHES (APPENDIX 3.4)

Mean Changes (R.D. Scores) across Follow-up Months

Cell Means

	Fing Temp	Temp A.A.	Prog Rel'n	Heart Rate	Waiting List
Month 1	1.13	0.25	-0.40	1.60	0.33
Month 2	1.38	0.25	0.00	1.60	0.33
Month 3	1.25	0.00	0.00	1.60	0.00

Standard Deviation Changes (R.D. Scores) across Follow-up Months

Standard Deviations

	Fing Temp	Temp A.A.	Prog Rel'n	Heart Rate	Waiting List
Month 1	0.99	0.71	0.89	0.89	0.58
Month 2	0.92	0.71	1.41	0.89	0.58
Month 3	1.04	0.00	1.41	0.89	2.00

Between treatment conditions across baseline and follow-up months				Relative difference scores across follow-up month		
		FING TEMP COND				
MONTH 1	MONTH 2	MONTH 3	MONTH 4	1	2	3
0099.0	0124.0	0000.0	0000.0	-0.4	+2.0	+2.0
0015.0	0000.0	0001.0	0000.0	+2.0	+1.8	+2.0
0241.6	0108.5	0000.0	0000.0	+0.8	+2.0	+2.0
1076.3	0000.0	0000.0	0000.0	+2.0	+2.0	+2.0
0075.0	0012.0	0063.5	0051.3	+1.5	+0.2	+0.4
0206.8	0000.0	0000.0	0338.4	+2.0	+2.0	-0.5
0090.0	0000.0	0000.0	0000.0	+2.0	+2.0	+2.0
0388.4	0090.0	0093.0	0132.3	+1.3	+1.3	+0.9
		TEMP ART AMP CONT				
0179.7	0070.2	0014.0	0106.3	+0.9	+0.9	+0.5
0360.0	0430.0	0595.1	0306.6	-0.2	-0.5	+0.2
0490.6	0212.8	0105.0	0126.0	+0.8	+1.3	+1.2
0262.8	0063.0	0018.0	0025.0	+1.2	+1.7	+1.6
0090.3	0000.0	0000.0	0255.0	+2.0	+2.0	-0.9
0039.0	0202.5	0096.0	0108.5	-1.4	-0.8	-0.9
0754.6	0618.8	0960.9	0252.8	+0.2	-0.2	+1.0
0000.0	0000.0	0115.5	0000.0	0.0	-2.0	0.0
		PROG REL COND				
0000.0	0321.1	0000.0	0052.0	-2.0	0.0	-2.0
0180.0	0126.0	0154.0	0231.0	+0.2	+0.2	-0.3
0225.6	0363.3	0000.0	0000.0	-0.5	+2.0	+2.0
0114.0	0093.0	0114.0	0126.0	+0.2	0.0	-0.1
000.0	0000.0	0072.0	0000.0	0.0	2.0	0.0
		HEART RATE COND				
0072.0	0060.0	0000.0	0000.0	+0.2	+2.0	+2.0
0096.0	0000.0	0000.0	0000.0	+2.0	+2.0	+2.0
0315.0	0000.0	0120.0	0000.0	+2.0	+0.9	+2.0
0141.4	0000.0	0000.0	0000.0	+2.0	+2.0	+2.0
0115.8	0000.0	0000.0	0027.0	+2.0	+2.0	+1.2
		WAITING LIST COND				
0060.0	0056.7	0027.4	0000.0	+0.1	+1.1	+2.0
0190.1	0113.5	0063.0	0032.0	+0.5	+1.0	+1.5
0000.0	0338.8	0000.0	0120.0	-2.0	0.0	-2.0

INDEX OF HEADACHE ACTIVITY (APPENDIX 3.5)

Mean Changes (R.D. Scores) across Follow-up Months

Cell Means

	Fing Temp	Temp A.A.	Prog Rel'n	Heart Rate	Waiting List
Month 1	1.25	0.25	-0.40	1.60	-0.67
Month 2	1.50	0.25	0.40	1.60	0.67
Month 3	1.25	0.38	0.00	1.80	0.33

Standard Deviation Changes (R.D. Scores) across Follow-up Months

Standard Deviations

	Fing Temp	Temp A.A.	Prog Rel'n	Heart Rate	Waiting List
Month 1	0.87	0.89	0.89	0.89	1.15
Month 2	0.76	1.16	1.67	0.89	0.58
Month 3	1.04	0.52	1.41	0.45	2.08

ANALGESIC INDEX (APPENDIX 3.6)

Between treatment conditions across baseline and follow-up months				Relative difference scores across follow-up months		
	FING TEMP COND					
MONTH 1	MONTH 2	MONTH 3	MONTH 4	1	2	3
024	006	000	000	+1.2	+2.0	+2.0
002	000	000	000	+2.0	+2.0	+2.0
000	000	000	000	0.0	0.0	0.0
180	000	000	000	+2.0	+2.0	+2.0
019	006	027	021	+1.0	-0.4	-0.2
000	000	000	000	0.0	0.0	0.0
024	000	000	000	+2.0	+2.0	+2.0
015	101	004	012	+0.3	+1.2	+0.2
	TEMP ART AMP CONT					
094	026	014	034	+1.1	+1.4	+0.9
032	044	064	040	-0.3	-0.7	-0.2
003	003	003	006	0.0	0.0	-0.7
036	008	000	000	+1.1	+2.0	+2.0
000	000	000	000	0.0	0.0	0.0
000	006	000	000	-2.0	0.0	0.0
068	078	010	000	-0.1	+1.5	+2.0
000	000	012	000	0.0	-2.0	0.0
	PROG REL COND					
000	040	000	004	-2.0	0.0	-2.0
024	015	015	033	+0.5	+0.5	-0.3
102	000	000	000	+2.0	+2.0	+2.0
012	012	018	012	0.0	-0.4	0.0
000	000	000	000	0.0	0.0	0.0
	HEART RATE COND					
030	012	000	000	+0.9	+2.0	+2.0
000	000	000	000	0.0	0.0	0.0
041	000	012	000	+2.0	+1.1	+2.0
034	000	000	000	+2.0	+2.0	+2.0
058	000	000	030	+2.0	+2.0	+0.6
	WAITING LIST COND					
028	072	008	000	-0.9	+1.1	+2.0
026	000	000	000	+2.0	+2.0	+2.0
000	015	000	003	-2.0	0.0	-2.0

ANALGESIC POTENCY (APPENDIX 3.6)

Mean Changes (R.D. Scores) across Follow-up Months

Cell Means

	Fing Temp	Temp A.A.	Prog Rel'n	Heart Rate	Waiting List
Month 1	1.00	0.00	0.00	1.20	0.00
Month 2	1.13	0.25	0.40	1.40	1.00
Month 3	1.00	0.50	0.00	1.20	0.67

Standard Deviation Changes (R.D. Scores) across Follow-up Months

Standard Deviations

	Fing Temp	Temp A.A.	Prog Rel'n	Heart Rate	Waiting List
Month 1	0.93	0.93	1.41	1.09	2.00
Month 2	0.99	1.16	0.89	0.89	1.00
Month 3	1.07	0.93	1.41	1.09	2.31

VASOCONSTRICTOR MEDICATION

Between Treatment Conditions, Across Baseline and Follow-up Months

Condition	FING TEMP				TEMP ART AMP				PROG REL'N				HEART RATE				WAITING LIST			
Months	1	2	3	4	1	2	3	4	1	2	3	4	1	2	3	4	1	2	3	4
	00	00	00	00	06	02-	00	00	00	00	00	00	08	04	00	00	28	00	00	00
	00	00	00	00	00	00	00	00	00	00	00	00	01	00	00	00	26	20	05	08
	00	01	00	00	07	03	00	00	00	04	00	00	01	00	02	00	00	01	00	00
	00	00	00	00	00	00	00	00	00	00	00	00	00	00	00	00				
	00	00	00	00	00	00	00	00	00	00	00	00	00	00	00	00				
	00	00	00	00	01	02	02	00												
	00	00	00	00	00	00	00	00												
	16	18	14	17	00	00	00	00												

Relative Difference Scores Across Follow-up Months

Condition	FING TEMP			TEMP ART AMP			PROG REL'N			HEART RATE			WAITING LIST		
Months	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3
	0.0	0.0	0.0	+1.3	+2.0	+2.0	0.0	0.0	0.0	+0.7	+2.0	+2.0	+2.0	+2.0	+2.0
	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	+2.0	+2.0	+2.0	+0.3	+1.4	+1.1
	0.0	+2.0	0.0	+0.7	+2.0	+2.0	0.0	-2.0	0.0	+2.0	-0.7	+2.0	-2.0	0.0	0.0
	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0			
	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0			
	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0			
	0.0	0.0	0.0	-0.7	-0.7	+2.0									
	0.0	0.0	0.0	0.0	0.0	0.0									
	+0.7	+1.2	+0.8	0.0	0.0	0.0									

VASOCONSTRICTOR MEDICATION (APPENDIX 3.7)

Mean Changes (R.D. Scores) across Follow-up Months

Cell Means

	Fing Temp	Temp A.A.	Prog Rel'n	Heart Rate	Waiting List
Month 1	0.25	0.13	-0.40	0.80	0.00
Month 2	0.13	0.25	0.00	0.80	1.00
Month 3	0.00	0.63	0.00	1.20	1.00

Standard Deviation Changes (R.D. Scores) across Follow-up Months

Standard Deviations

	Fing Temp	Temp A.A.	Prog Rel'n	Heart Rate	Waiting List
Month 1	0.71	0.34	0.89	1.09	2.00
Month 2	0.35	0.71	0.00	1.09	1.00
Month 3	0.00	0.92	0.00	1.09	1.00

PROPHYLACTIC MEDICATION

Between Treatment Conditions, Across Baseline and Follow-up Months

Condition	FING TEMP				TEMP ART AMP				PROG REL'N				HEART RATE				WAITING LIST			
Months	1	2	3	4	1	2	3	4	1	2	3	4	1	2	3	4	1	2	3	4
	000	000	000	000	000	000	000	000	000	000	000	000	093	093	093	093	000	000	000	000
	000	000	000	000	186	186	000	000	000	000	000	000	000	000	000	000	000	000	084	000
	000	000	000	000	000	000	000	000	000	000	000	000	000	000	000	000	000	000	000	000
	000	000	000	000	000	000	000	000	036	000	000	060	000	000	000	000				
	000	000	000	000	000	000	000	000	000	000	000	000	000	000	000	000				
	000	000	000	000	000	000	093	000												
	000	000	000	000	000	000	000	000												

Relative Difference Scores Across Follow-up Months

Condition	FING TEMP			TEMP ART AMP			PROG REL'N			HEART RATE			WAITING LIST		
Months	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3
	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.0	0.0	+2.0	+2.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	-2.0	0.0
	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.0	0.0	0.0	0.0	0.0	+2.0	+2.0	-0.5	0.0	0.0			
	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0			
	0.0	0.0	0.0	0.0	0.0	0.0									
	0.0	0.0	0.0	0.0	-2.0	0.0									
	0.0	0.0	0.0	0.0	0.0	0.0									

(APPENDIX 3.8)

PROPHYLACTIC MEDICATION (APPENDIX 3.8)

Mean changes (R.D. scores) across follow-up months

Cell Means

	Fing Temp	Temp A.A.	Prog Rel'n	Heart Rate	Waiting List
Month 1	0.00	0.00	0.40	0.00	0.00
Month 2	0.00	0.00	0.40	0.00	-0.67
Month 3	0.00	0.25	0.00	0.00	0.00

Standard Deviation Changes (R.D. Scores) across Follow-up Months

Standard Deviations

	Fing Temp	Temp A.A.	Prog Rel'n	Heart Rate	Waiting List
Month 1	0.00	0.00	0.89	0.00	0.00
Month 2	0.00	0.07	0.89	0.00	1.15
Month 3	0.00	0.71	0.00	0.00	0.00

MULTIPLE REGRESSION ANALYSIS (APPENDIX 3.9)

Column 1. Finger temperature : mean change, period 1.

2. Finger temperature : mean change, period 2.

3. Finger temperature : mean change, period 3.

4. Temporal artery amplitude (RD means) mean change, period 1.

5. Temporal artery amplitude (RD means) mean change, period 2.

6. Temporal artery amplitude (RD means) mean change, period 3.

7. Temporal artery amplitude (RD standard deviations) mean change, period 1.

8. Temporal artery amplitude (RD standard deviations) mean change, period 2.

9. Temporal artery amplitude (RD standard deviations) mean change, period 3.

10. Heart rate, mean change, period 1.

11. Heart rate, mean change, period 2.

12. Heart rate, mean change, period 3.

13. Mean 'Index of Headache Activity' score.

14. Age of patient.

15. Duration of illness.

FINGER TEMPERATURE CONDITION

Column	PATIENT 1	PATIENT 2	PATIENT 3	PATIENT 4
1	+03.02	-01.65	-00.27	-00.11
2	+10.40	-02.79	+02.50	+02.76
3	+08.58	-03.72	+01.96	-02.09
4	+00.00	+00.03	-00.04	+00.01
5	+00.06	-00.03	-00.08	+00.06
6	+00.11	-00.02	-00.04	+00.09
7	-00.20	-00.07	+00.27	-00.08
8	-00.05	-00.17	+00.12	+00.06
9	+00.00	-00.16	+00.09	+00.11
10	+05.30	-03.40	-04.50	-05.90
11	+00.50	-07.30	+03.70	-06.70
12	-09.40	-07.90	+01.10	-08.80
13	+01.20	+01.90	+01.60	+02.00
14	+23.70	+54.60	+55.80	+27.00
15	+19.00	999999	999999	+15.00

Column	PATIENT 1	PATIENT 2	PATIENT 3	PATIENT 4
1	-00.44	+00.38	+02.04	-00.81
2	+00.27	+01.30	+01.52	+03.04
3	-00.15	+01.39	+00.17	-02.40
4	-00.02	-00.01	+00.08	+00.21
5	-00.07	+00.02	+00.12	+00.28
6	-00.01	+00.09	+00.19	+00.30
7	-00.09	-00.06	+00.01	+00.31
8	-00.13	+00.04	+00.10	+00.31
9	-00.10	+00.16	+00.18	+00.30
10	+08.20	+00.80	-00.70	+27.00
11	+00.30	-20.40	-02.00	+24.10
12	-03.40	-03.50	-07.90	+13.80
13	+00.70	+01.50	+02.00	+01.20
14	+52.30	+49.90	+43.90	+33.30
15	+28.00	+25.00	+06.00	+15.00

TEMPORAL ARTERY AMPLITUDE CONDITION

Column	PATIENT 1	PATIENT 2	PATIENT 3	PATIENT 4
1	+0.131	+00.84	-03.27	+02.18
2	+02.70	+00.99	-03.26	+02.52
3	+03.14	+00.67	-04.83	+02.09
4	-00.07	-00.06	+00.06	-00.02
5	-00.02	-00.08	+00.24	+00.02
6	+00.00	-00.05	+00.36	+00.08
7	+00.03	-00.06	+00.14	-00.17
8	+00.03	-00.12	+00.33	-00.20
9	+00.04	-00.02	+00.28	-00.22
10	-15.80	-04.80	-07.90	-01.00
11	-18.30	-02.70	-11.70	-02.90
12	-13.00	-05.60	-10.70	-03.90
13	+00.80	+04.00	+01.10	+01.50
14	+51.80	+42.00	+47.80	+16.90
15	+20.00	+31.00	+34.00	+08.00

	PATIENT 5	PATIENT 6	PATIENT 7	PATIENT 8
1	-40.17	-00.81	+00.24	-01.99
2	-06.18	-01.29	-01.87	+00.15
3	-10.73	+04.09	-01.52	-00.63
4	+00.01	+00.19	+00.03	+00.14
5	+00.06	+00.28	+00.14	+00.32
6	+00.23	+00.35	+00.27	+00.45
7	-00.16	+00.02	-00.02	+00.19
8	+00.05	-00.06	+00.16	+00.35
9	+00.19	-00.04	+00.27	+00.36
10	-02.30	-09.40	-04.20	-11.30
11	+02.00	-09.90	-09.50	-22.00
12	+04.00	-12.70	-13.00	-24.00
13	+01.60	-01.00	+00.50	-07.00
14	+20.20	+50.30	+21.08	+21.92
15	999999	+20.00	999999	999999

(APPENDIX 3.9)

Column	PATIENT 1	PATIENT 2	PATIENT 3	PATIENT 4	PATIENT 5
1	-06.61	+02.69	+00.58	+02.65	+02.32
2	-10.40	-03.90	-00.92	+04.12	+08.48
3	-11.01	-04.59	-02.41	+05.01	+03.89
4	+00.17	+00.13	+00.09	-00.10	+00.12
5	+00.25	+00.22	+00.21	-00.05	+00.16
6	+00.30	+00.28	+00.24	+00.03	+00.15
7	+00.11	+00.18	+00.10	+00.14	+00.19
8	+00.23	+00.14	+00.24	+00.20	+00.30
9	+00.25	+00.24	+00.05	+00.33	+00.14
10	-23.50	-18.10	-13.00	-11.60	-01.60
11	-27.80	-12.00	-11.60	-10.10	-09.60
12	-11.60	-14.80	-14.00	-14.70	-13.40
13	-01.30	+00.00	+01.20	+00.00	+00.70
14	+30.30	+36.80	+15.60	+40.10	+22.70
15	+18.00	+14.00	999999	999999	+06.00

Heart Rate Condition

Column	PATIENT 1	PATIENT 2	PATIENT 3	PATIENT 4	PATIENT 5
1	-05.93	+01.21	+00.17	+01.85	+00.19
2	-08.43	+00.79	+00.33	+09.25	-01.50
3	-11.90	-00.67	+00.30	+07.34	-03.89
4	+00.03	+00.01	+00.07	+00.14	+00.06
5	+00.13	+00.05	+00.21	+00.13	+00.09
6	+00.20	+00.15	+00.18	+00.16	+00.15
7	-00.12	+00.11	+00.15	+00.11	+00.00
8	-00.04	+00.11	+00.18	+00.02	+00.06
9	-00.19	+00.27	+00.23	+00.00	+00.04
10	-17.50	-06.10	-12.10	+06.00	-05.80
11	-19.60	-08.00	-22.30	-10.50	-07.40
12	-32.70	-09.00	-24.20	-17.10	-03.60
13	+01.40	+02.00	+01.60	+02.00	+01.70
14	+60.08	+52.20	+51.20	+42.00	+49.60
15	+02.00	999999	+16.00	+10.00	+45.00

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Abbreviated Progressive Muscular Relaxation Exercises

"I want you to sit quietly - rest your hands on the arms of the chair and sit with feet slightly apart. Do not fold your arms or cross your legs.

Close your eyes and concentrate on your toes: Curl them down toward the soles of your feet and hold the tension - then slowly let the tension go. Notice the feeling of relief. As you relax, repeat the word "relax" to yourself.

With your eyes closed, concentrate on your calves, the lower part of your legs: Point your toes up towards your face and tighten your calves. Hold the tension - then slowly let the tension go. Repeat the word 'relax' to yourself.

Now concentrate on your thighs, the upper part of your legs: Push your feet on the floor and feel the tension in your thighs and hold the tension - slowly let the tension go. Notice the feelings of relief and repeat the word 'relax' to yourself. Notice the difference now between the feelings you are getting from your legs as compared to the feelings from your upper body. Your legs feel relaxed and heavy, and your toes may be tingling. Concentrate on the differences you feel.

Concentrate on your stomach area: Tense your stomach muscles and imagine you are protecting yourself from a punch - hold the tension - then slowly let the tension go. Feel the relaxation.

Now I want you to concentrate on your upper body and shoulders: Keep breathing steadily, regularly and calmly. Tighten your shoulders by shrugging them, bringing your shoulders up around your ears.

Lift your shoulders and hold the tension - then slowly let it go. Repeat the word 'relax' to yourself.

Notice what is happening to your body now. It is feeling more relaxed, heavier and perhaps you are feeling a tingling sensation over your body. Concentrate on your forehead, your eyes and your mouth. Let the muscles relax, feel comfortable and warm. Breath steadily and stay calm and quiet.

The patients receiving the progressive muscular relaxation exercises reported them to be effective in producing feelings of relaxation. However, no record of the patients' responses were kept and no systematic attempt was made to determine the degree of relaxation achieved by the patients. In fact no attempt was made to assess any patients perceived success of their treatment task. In this way all self report data related to clinical information only, in an attempt to minimize "performance" related anxieties and thus maximize patient compliance.

Results

Finger Temperature Biofeedback

Graph I shows the mean change in finger temperature for each subject (per 30 second epoch). Although subject I in the finger temperature feedback condition achieved a mean temperature elevation in slight excess of $0.7^{o}c$, the other subjects failed to demonstrate changes of a similar magnitude. In fact six subjects in the Heart Rate feedback group displayed above baseline changes compared with four subjects in the Finger Temperature feedback group.

A visual inspection of the results would suggest that finger temperature feedback did not facilitate increases in temperature. However, the number of finger temperature feedback subjects showing only small decreases in temperature may suggest that feedback could militate against the effects of downward 'drift'. This, however, is speculative, and a point of departure for future research.

Temporal Artery Amplitude Biofeedback

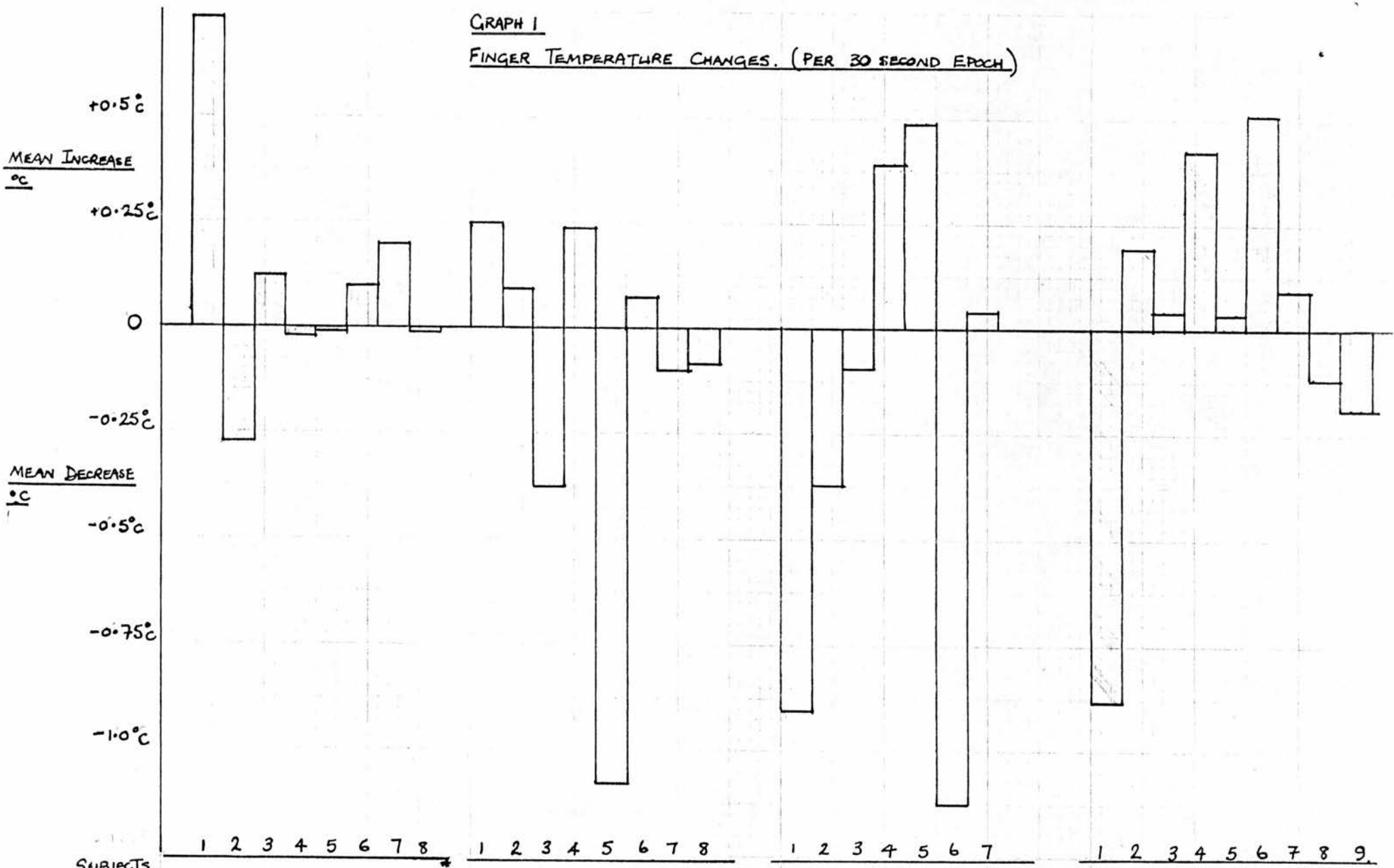
Graph II shows the mean change in temporal artery amplitude, for each subject across treatment periods. Those subjects receiving information about temporal artery amplitude changes, were expected to show decreases in amplitude, compared with the other groups. A visual inspection of the results would indicate that temporal artery amplitude feedback did not enable subjects to reduce the mean size of artery pulses. The finger temperature feedback group has a greater number of subjects displaying a reduction in artery amplitude. than any other treatment group.

Heart Rate Biofeedback

Graph III shows the mean change in heart rate for each subject across treatment periods. It was anticipated that the Heart Rate feedback group would show greater decreases in heart rate than the other treatment groups. A visual examination of the results would suggest that the heart rate changes displayed by the Progressive Relaxation and Heart Rate Groups are similar. The similarities are in both, number of subjects achieving a decrease and the magnitude of decrease. The finger temperature group responses are conspicuous by their general tendency to display reduced heart rate change. They are also the only group with three subjects showing increases in heart rate. The reason for these differences in responses are still to be elucidated.

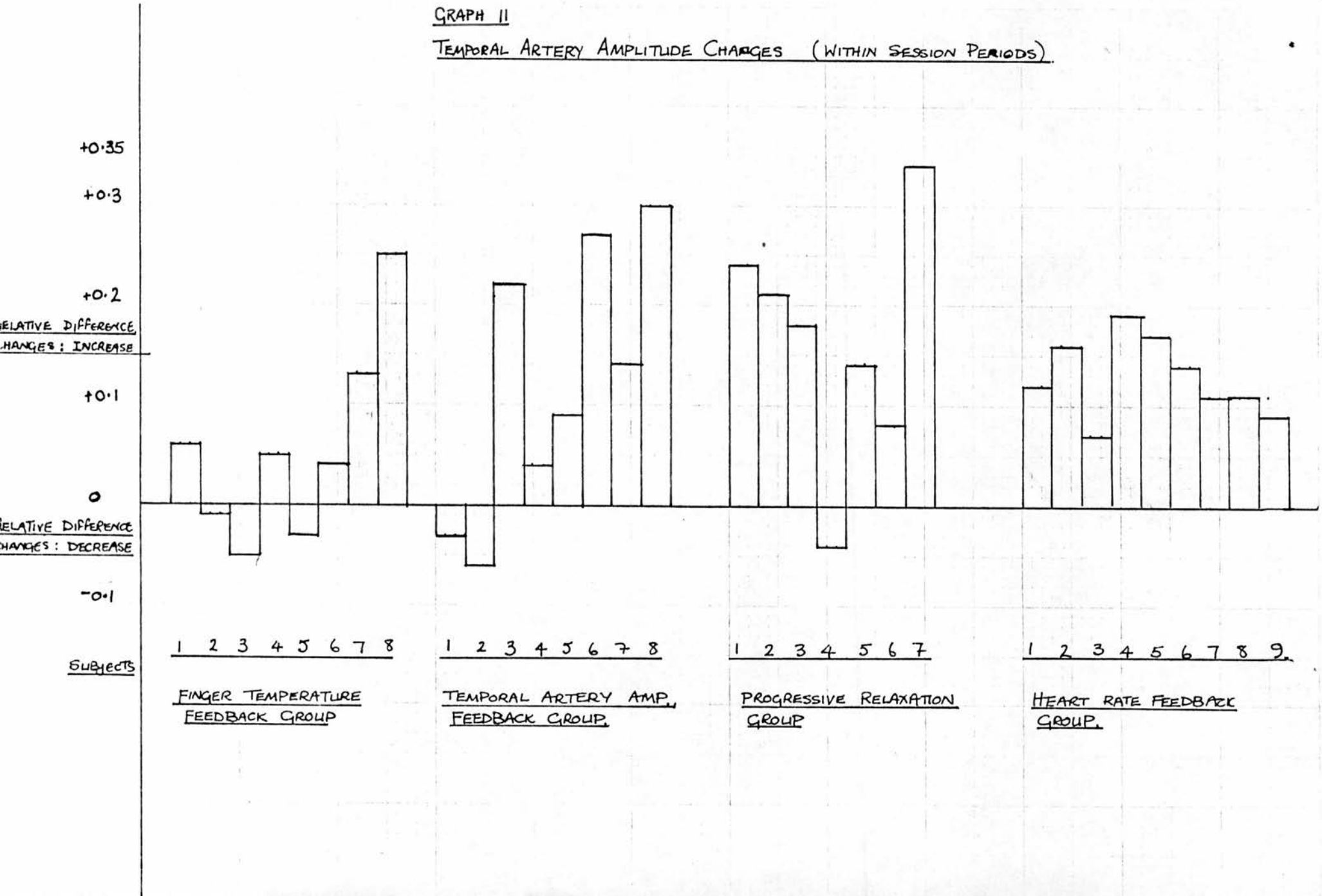
GRAPH 1

FINGER TEMPERATURE CHANGES. (PER 30 SECOND EPOCH)



GRAPH II

TEMPORAL ARTERY AMPLITUDE CHANGES (WITHIN SESSION PERIODS)



GRAPH III

HEART RATE CHANGES (WITHIN SESSION PERIODS)