THE ROLE OF COGNITIVE PROCESSES

IN RECURRENT HEADACHE

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A thesis submitted for the degree of Doctor of Philosophy from the Australian National University.

September, 1989.
I declare that this thesis reports my original work; that no part has been previously accepted or presented for the award of any degree or diploma from any university; and that, to the best of my knowledge, no material previously published or written by any other person is included, except where due acknowledgement is given.

Nicholas Ian Francis-Jones.
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ABSTRACT

The studies reported in this thesis sought to identify the cognitive processes mediating the hypothesised relationship between stressful events and recurrent headache. The prospects for linking cognitive processes to headache, through the mechanism of sensory modulation, were also explored.

A treatment study (reported in Chapter 6) compared rational-emotive therapy with progressive relaxation training and correlated changes in headache activity with alterations in rationality. The results did not support the hypothesis that rationality mediates improvements in headache. Rational-emotive therapy was no more effective in reducing headache activity (headache frequency, duration, intensity and medication consumption) than progressive relaxation training. Changes in headache activity occurring in each treatment were not correlated significantly with changes in scores on the Rationality Scale developed for the purposes of the study. It was suggested that the reduction in headache activity, observed in both treatments, may have been mediated by increases in self-efficacy which may have flowed from the acquisition of skills for the management of stressful events and headache.

In a second study (reported in Chapter 7) self-efficacy, concerning control over behavioural, cognitive and affective responses (self-control-efficacy), was observed to buffer the relationship between stressful events and headache frequency. Stressful events were found to precede headache attacks more often than periods of headache freedom. Higher levels of emotional upset in response to stressful events were found to precede periods of headache freedom, whilst lower levels preceded headache episodes. It was suggested that stronger emotional reactions to stress may inhibit the processes underlying headache onset.

Stressful events occurring during headache were followed by increases in the intensity of the attack. In these instances, avoidance coping was associated with higher headache intensity ratings following the event and direct coping with lower post-event ratings. The appraisal of stressful events as amenable to change and higher levels of coping behaviour were found to reduce the frequency of ensuing headache episodes. There was no differential effect of strategy use (avoidance coping, direct coping or affective regulation) on the occurrence of headache in the face of stressful events. The results were considered to support the thesis that cognitive processes may contribute to the onset of headache attacks.

The final study (reported in Chapter 8) explored the prospects for linking the cognitive constructs studied in Chapter 7 to headache, through the mechanism of sensory modulation.
Headache subjects were found to have lower pain threshold and tolerance levels for electrical stimulation of the finger than control subjects. Headache subjects were also observed to have lower pain tolerance levels when ice was applied to the temporal region but, no significant difference between groups was found for temporal ice pain threshold. When threshold and tolerance levels for electrical finger pain were assessed in headache subjects, during and between attacks, no significant difference between conditions was observed.

The $P_1-N_1$ amplitude of the somatosensory average evoked potential (SSEP) was found to be greater, and to increase more rapidly with stimulus intensity, for headache sufferers than for control subjects. The $N_1-P_2$ amplitude was also found to be larger for headache subjects than for controls, although there was no significant difference between groups on the rate at which this amplitude increased with stimulus intensity. When the $P_1-N_1$ and $N_1-P_2$ SSEP components were assessed in headache subjects, during and between attacks, no significant differences between conditions were observed. No significant differences between pain sensitive and pain insensitive headache subjects were observed on $P_1-N_1$ or $N_1-P_2$ amplitudes. For the control subjects, the $N_1-P_2$ component increased more rapidly with stimulus intensity for pain sensitive than for pain insensitive subjects, but this result was not replicated for the headache sufferers.

The results of the study reported in Chapter 8 were interpreted as being consistent with the view that headache sufferers may be deficient in their capacity to modulate sensory input. It was suggested that this might represent one avenue through which cognitive processes could contribute to headache.

It was concluded that cognitive processes may play a significant role in the relationship between stressful events and headache and, may be related to headache through the mechanism of sensory modulation. Lines of inquiry from which further research might profit are discussed.
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CHAPTER 1

INTRODUCTION.

1.1. Background, Epidemiology and Demography.

Headache has been a common complaint of the human race for many centuries. The problem is mentioned in the writings of the ancient Babylonians, dating from about 3000 B.C. (Alvarez, 1945; McHenry, 1969), with the earliest clinical descriptions being recorded by the ancient Greek physician, Hippocrates (460-370 B.C.) (Critchley, 1967). Lance (1977) has drawn attention to the practice of trepanning (drilling holes in the skull), which, until recently, had been employed by the Melanesian Islanders as a method for relieving epilepsy, insanity and chronic headache. Evidence of trepanning has been found in skulls dating back to the Neolithic and Bronze ages, leading Lance (1977) to venture the suggestion that headache may have been troubling the human race for over ten thousand years.

In modern times, headache has become one of the most commonly presented complaints in outpatient medical care (De Lozier and Gagnon, 1975; Leviton, 1978). In the twelve months from 1973 to 1974, Americans made 12,314,000 visits to medical practitioners on account of headache (De Lozier and Gagnon, 1975). A survey conducted in Britain (Dunnell and Cartright, 1972) indicated that 38% of adults consulting general practitioners complained of headache. No other health problem was mentioned to doctors so frequently.

Surveys of the general population have confirmed the ubiquity of headache (e.g., Kaganov, Bakal and Dunn, 1981; Ogden, 1952; Waters and O’Connor, 1975). A study of the prevalence of headache in Calgary, Canada (Kaganov et al., 1981) revealed that 90% of females and 78% of males reported experiencing a headache in the last year. In a survey of 4,634 Americans, Ogden (1952) found that 48% of the sample suffered with headache more than once per month; 30.8% suffered in excess of two headaches per month and more than 1% suffered headache every day. Nikiforow and Hokkanen (1978) surveyed a Finnish population and found that 37% (40% of females and 33% of males) suffered at least one mild headache per week. Furthermore, 15% of the sample (15% of females and 16% of males) experienced severe headaches at least once per week.

Females tend to be afflicted by headache more often than males with about 75% of headache sufferers being female (Friedman et al, 1954; Lance, Curran and Anthony, 1965;
Martin and Nathan (1987) observed the prevalence of headache amongst undergraduate psychology students to be 2.6 to 3.6 times greater for Americans than for Australians, suggesting that there may be some cultural influence on the prevalence of the complaint. However, headache has been reported to occur independently of such factors as social class, education or intelligence (e.g., Friedman et al., 1954; Markush, Karp, Heyman and O'Fallon, 1975; Waters, 1975).

Passchier and Orlebeke (1985) studied the prevalence of headache among 602 elementary school children and 1579 secondary school children living in Amsterdam. Of the elementary school children (aged 7 to 10 years), 16% (12% of males and 20% of females) reported suffering with headache several times per month and 8% (6% of males and 11% of females) several times per week. For the secondary school children (aged 12 to 17 years), 17% (12% of males and 22% of females) experienced headache several times per month and 10% (5% of males and 15% of females) several times per week. Thus, headache is a common problem for children as well as adults.

1.2. The Classification of Headache.

The first attempt at a formal classification of headache was made by the Ad Hoc Committee on Classification of Headache (1962). The Ad Hoc Committee sought to classify headache on the assumption that differential features of the attack represent the involvement of differential pain mechanisms. In 1962, when the Ad Hoc Committee designed their classificatory system, opinion on the pathophysiology of headache was influenced heavily by the work of Wolff and his colleagues. These researchers suggested that tension headache was occasioned by contraction of the muscles of the neck and cranium (Ostfeld, Reis and Wolff, 1957) and that migraine arose from vasomotor instability of the cranial arteries (Tunis and Wolff, 1954). Thus, the Ad Hoc Committee's (1962) classification reflects the view that tension and migraine headache are qualitatively distinct disorders, each with a specific pathophysiology.

The Ad Hoc Committee (1962, p. 378) identified 15 varieties of headache:

"1. Vascular headache of migraine type.
   a. Classic migraine.
   b. Common migraine.
   c. Cluster headache.
   d. Hemiplegic and ophthalmoplegic migraine.
   e. Lower-half headache."
2. Muscle-contraction (tension) headache.


4. Headache of nasal vasomotor reaction.

5. Headache of delusional, conversion or hypochondriacal states (psychogenic headache).


7. Traction headache.

8. Headache due to overt cranial inflammation.

9-13. Headache due to disease of ocular, aural, nasal and sinus, dental or other cranial or neck structures.


15. Cranial neuralgias."

The vast majority of headache sufferers tend to be classified into one of the first three categories. For example, in a study of 1152 headache clinic patients, 94% were classified into the migraine, tension or combined categories (Lance et al, 1965). This thesis is concerned with these headache types.

Vascular headaches of the migraine type were defined as follows:

"Recurrent attacks of headache, widely varied in intensity, frequency and duration. The attacks are commonly unilateral in onset, are usually associated with anorexia and sometimes with nausea and vomiting, in some are preceded by or associated with, conspicuous sensory, motor and mood disturbances, and are often familial...cranial arterial disention and dilatation are important in the pain phase but cause no permanent changes in the involved vessel (Ad Hoc Committee, 1962, p. 378).

The subtypes of this first category were defined as follows:

a) Classic (or classical) migraine, where headache is preceded or accompanied by transient sensory disturbances such as visual or motor prodromes.
b) Common migraine, where the vascular basis to the headache remains in the absence of sensory prodromes.

c) Cluster headache, where headache is predominantly unilateral, consistently affecting the same side of the head, of brief duration and usually occurring in bouts separated by lengthy remissions. It is usually associated with flushing, sweating, rhinorrhea and increased lacrimation.

d) Hemiplegic and ophthalmoplegic migraine, where the sensory and motor prodromes persist during and for a period following the headache.

e) Lower-half headache, where the painful area is the lower region of the face rather than the head.

Bakal (1975) has noted that the distinction between the classical and common migraine types is not typically observed in the literature, with researchers often content to subsume both subtypes into the single category of migraine. In a revision of the Ad Hoc Committee's (1962) classification, the Headache Classification Committee of the International Headache Society (1988) classified cluster and lower-half headache in categories separate from migraine. Thus, in this thesis, the term migraine does not encompass the cluster and lower-half headache types.

The Ad hoc Committee (1962, p. 379) offered the following definition of tension headache:

"...ache or sensation of tightness, pressure or constriction, widely varied in intensity, frequency and duration, sometimes long lasting and commonly suboccipital. It is associated with sustained contraction of skeletal muscles in the absence of permanent structural change, usually as part of the individual's reaction during life stress."

The combined headache category, often referred to as tension-vascular (e.g., Lance and Curran, 1964) or mixed (e.g., Feurstein, Bush and Corbisiero, 1982) headache, was formulated to encompass those headaches for which the features of both the migraine and tension headache types co-exist in the one attack (Ad Hoc Committee, 1962). This classification has also been applied to persons who suffer migraine and tension headaches on separate occasions (e.g., Olesen, 1978).

As Martin (1985) has noted, the Ad Hoc Committee's classificatory system is weakened considerably by the body of evidence suggesting that migraine sufferers cannot be reliably distinguished from tension headache sufferers on the basis of cranial or neck muscle tension levels (e.g., Andrasik, Blanchard, Arena, Saunders and Barron, 1982; Arena, Blanchard,
Andrasik, Applebaum and Meyers, 1985; Philips and Hunter, 1982). Indeed, resting electromyographic (EMG) levels have been observed to be higher for migraineurs than for tension headache sufferers (e.g., Bakal and Kaganov, 1977; Pozniak-Patewicz, 1976) and it has been reported that increases in extracranial (Drummond and Lance, 1983) or cerebral (Oleson, Lauritzen, Tfelt-Hansen, Henriksen and Larsen, 1982) blood flow are not necessary for migraine headache. The pathophysiology of migraine and tension headache is discussed in detail in Chapter 2.

The results of factor analytic studies involving the frequency with which migrainous features accompany or precede headache, have also questioned the Ad Hoc Committee's (1962) grounds for the distinction between the tension and migraine types (e.g., Drummond and Lance, 1984a; Peck and Attfield, 1981; Ziegler, Hassanein and Couch, 1982; Ziegler, Hassanein and Hassanein, 1972). These researchers reasoned that if migraine does exist as a qualitatively distinct headache condition, the factor structure emerging from an analysis of symptoms should reveal a cluster of migrainous features on a single factor representing the migraine syndrome. In each instance this expectation was not confirmed, thereby, undermining the view of migraine as a separate clinical entity.

Drummond and Lance (1984a) found that, although the features of cluster headache loaded on a clearly defined factor, subjects classified as suffering from classical migraine, common migraine, tension-vascular or tension headache could not be identified with particular clusters of symptoms. That is separate factors, corresponding to each of the four headache types, did not emerge from the analysis. Instead, these groups of subjects tended to differ from each other in terms of the number, rather than the nature of migrainous features reported, with classical migraineurs reporting most features, followed by common migraineurs, tension-vascular headache sufferers and tension headache sufferers who reported the fewest migrainous features. Drummond and Lance (1984a) concluded that, with the exception of cluster headache, clinically defined headache categories represent different points on a continuum rather than discrete entities.

Other researchers have found that the frequency with which all types of features occur, be these musculoskeletal (e.g., dull aches or sensations of tightness), vascular (e.g., throbbing or pulsating pain) focal neurological (e.g., visual disturbances or paraesthesiae) or gastrointestinal (e.g., nausea or vomiting), increases with the rated severity of headache, but that no particular set of features differentiates tension from migraine headache sufferers (e.g., Bakal and Kaganov, 1979; Thompson, Haber, Figueroa and Adams, 1980; Waters, 1974). Accordingly, a continuum model of headache has been proposed such that any given headache sufferer may be located on a continuum of severity, between the extremities of tension and migraine headache, at a point corresponding to his or her usual headache, but may suffer
headaches at quite disparate points within that headache spectrum (e.g., Bakal and Kaganov, 1977, 1979; Holroyd and Andrasik, 1982a; Thompson et al, 1982). According to this view, common psychobiological processes underlie both tension and migraine headache, with the frequency of associated features increasing with headache severity.

Against the continuum model of headache severity, Peck and Attfield (1981) found that the frequency of migrainous features was not associated with the rated severity of headache. All subjects in this study, however, were migraineurs and the range of severity ratings was very narrow. These characteristics of the sample may have obscured any correlation between severity ratings and the frequency of migrainous features that might have emerged over a broader range of severity ratings.

Hunter and Philips (1981) compared tension headache and migraine sufferers on pain descriptors chosen from the McGill Pain Questionnaire (Melzack, 1975). Although diary recordings of headache activity indicated no differences in severity between migraineurs and tension headache subjects, the former chose significantly more adjectives describing vascular features (e.g., sickening, nauseating, blinding, sharp) than did the tension headache cases, who preferred the word "tight" as descriptive of their pain. No such difference between groups in the quality of pain experience is predicted by the continuum model. However, many of the subjects studied by Hunter and Philips (1981) were psychiatric patients. Thus, the generality of the findings may be limited. Indeed, in a similar study, involving a non-psychiatric sample, Hunter (1983) was unable to replicate these results and interpreted the data as being in line with the continuum model.

Although the weight of the evidence favours the continuum model, researchers persist with the distinction between migraine and tension headache and this seems unlikely to change in the near future.

Martin (1985) has pointed out that a multiaxial system of classification could be applied to headache. Within such a system, headache could be classified along a number of dimensions including, for example, a classification in terms of antecedents such as stress, certain foods, visual or auditory stimulation. Such a classification may prove useful to those planning interventions for headache sufferers.

The Headache Classification Committee of the International Headache Society (1988) recently revised the Ad Hoc Committee's (1962) system and listed operational criteria for the classification of headache into particular categories. This may reduce the level of heterogeneity within subject groups classified as "tension" or "migraine" sufferers. However, the evidence suggests that, although this distinction may provide a convenient method for
describing subjects' headaches, it does not imply the existence of two separate disorders distinguishable on the basis of clinical features (e.g., Bakal and Kaganov, 1979; Drummond and Lance, 1984a; Waters, 1974).

1.3. Genetic Factors.

Evidence relating to the genetic transmission of a predisposition to headache comes from studies of the family history of the disorder (e.g., Dalsgaard-Nielsen, 1965; Green, 1977; Lance and Anthony, 1966) and from investigations into the concordance rates for monozygotic and dizygotic twin pairs (Harvald and Hauge, 1956; Ziegler, Hassanein, Harris and Stewart, 1975).

In an extensive inquiry into the family backgrounds of 100 female migraineurs, Dalsgaard-Nielsen (1965) found 90% to report a positive family history. In 57% of these cases the afflicted relative was the mother. Studies involving parents, grandparents and siblings (e.g., Green, 1977; Selby and Lance, 1960) report a family history of migraine in about 50% to 60% of migrainous cases. A family history of migraine is found in about 16% of headache-free controls (Green, 1977). Once again, amongst migraineurs, the mother has been found to be the most commonly afflicted family member, accounting for 53% of positive case histories in Green's (1977) study. Lance and Anthony (1966) included the parents and siblings of tension headache and migraine sufferers. These researchers found a positive family history of migraine in 46% of the migraineurs, as compared with only 18% of the tension headache subjects.

Although the above studies employed interview methods to establish positive and negative family histories, Waters (1971) used a survey method. He observed the prevalence of migraine amongst the immediate relatives of migraineurs to be no greater than for those of headache-free control subjects. Survey methods may be less probing than interview methods and perhaps less likely to uncover positive family histories.

The evidence is generally supportive of the notion that familial factors of some kind may be involved in the aetiology of migraine. Far less data have been collected on the family history of tension headache. Although Friedman et al (1954) observed 40% of tension headache sufferers to report a family history of some kind of headache, this figure is difficult to evaluate in the absence of an estimate of the proportion of headache-free persons reporting a similar family history. Currently, the data is such that no conclusion can be drawn regarding the role of familial influences in tension headache.
Very few studies of concordance rates for headache in pairs of twins have been conducted, making it difficult to draw any firm conclusion regarding the question of genetic transmission. Harvald and Hauge (1956) identified 84 migraine sufferers from a sample of 1900 pairs of twins. Twenty four migraineurs were members of monozygotic twin pairs. For this group the concordance rate for migraine was 33%. Fifty seven migraine subjects belonged to dizygotic twin pairs, for whom the concordance rate was 5%. Such a result is consistent with the hypothesis that genetic factors contribute to migraine. However, Ziegler et al (1975) studied headache sufferers across the tension-migraine spectrum and observed concordance rates of about 20% for both monozygotic and dizygotic twin pairs. These concordance rates were not significantly greater for subjects reporting migrainous features.

The evidence for a genetic component in migraine draws most heavily upon analyses of the family history of the disorder (e.g., Dalsgaard-Nielsen, 1965; Green, 1977). However, conclusions drawn from this evidence are tempered by the possibility that environmental factors within the family, instead of, or as well as genetic influences, could be contributing to the onset and / or severity of the condition. In the case of tension headache, there exists no convincing body of evidence to support a role for genetic factors.

1.4. Factors Precipitating Headache.

Clinical interviews with headache sufferers have suggested that psychological stress is the most common precipitant of headache attacks (e.g., Friedman, 1979; Friedman et al., 1954; Howarth, 1965; Selby and Lance, 1960). It is an examination of the nature and strength of the relationship between psychological stress and headache which is a principal focus of this thesis. A discussion of the evidence pertaining to this relationship is presented in Chapter 3.

Investigations into other precipitating factors have been confined almost exclusively to studies involving migraine sufferers. This may be because tension headache is often present every day (Friedman et al., 1954; Lance and Curran, 1964), making it difficult to identify specific trigger factors. Among the factors which have been reported to precipitate migraine are oral contraceptives (e.g., Carrol, 1971; Kudrow, 1975), menstruation (e.g., Sacks, 1971; Selby and Lance, 1960), glare (e.g., Selby and Lance, 1960; Vijayan, Gould and Watson, 1980) and foods such as alcohol, chocolate, fruit and dairy products (e.g., Selby and Lance, 1960). However, several studies have found that, when the diets of migrainous subjects are manipulated, little effect of these foods on headache is evident (e.g., Medina and Diamond, 1978; Olesen, 1984).

This thesis is concerned with an examination of the relationship between stress and headache. Before considering the nature of this relationship, however, it is important to
consider the research that has been conducted into the pathophysiology of headache, as this will have implications for the mechanisms through which psychological processes might contribute to the condition. Accordingly, the next chapter is concerned with the pathophysiology of migraine and tension headache.
CHAPTER 2

THE PATHOPHYSIOLOGY OF MIGRAINE AND TENSION HEADACHE.

2.1. Introduction.

The literature on the pathophysiology of headache has been dominated by the view of tension headache as the result of sustained contraction of the muscles in the neck and cranium (Ad Hoc Committee, 1962; Ostfeld, Reis and Wolff, 1957) and by the view of migraine headache as arising from vasomotor instability of the cranial arteries (Tunis and Wolff, 1954; Wolff, 1963).

In this chapter, these two theories are critically reviewed and shown to be inadequate explanations for the occurrence of headache. The view of headache as being the result of some deficiency in central pain inhibitory processes (e.g., Sicuteri, 1982) is also considered, but is judged to have difficulty in accounting for the specific location of head pain. It is suggested that a complete account of headache is most likely to follow from a consideration of both central and peripheral factors (e.g., Lance, 1982). The recognition of the involvement of central pain regulatory processes in headache (Sicuteri, 1982) is regarded as an important development for researchers concerned with the role of psychological factors in this condition.

Given that the literature on the pathophysiology of headache is organised around the distinction between tension and migraine headache, for the sake of consistency and clarity, this review will consider research within each headache type separately.

2.2. The Role of Muscle Contraction in Tension Headache.

Investigations into the view that tension headache is produced by over-contraction of the neck and / or cranial muscles (Ad Hoc Committee, 1962) have focused on comparisons of headache sufferers with headache-free control subjects on muscle tension levels at a variety of sites under conditions of rest (e.g., Anderson and Franks, 1981; Andrasik, Blanchard, Arena, Saunders and Barron, 1982) and laboratory induced stress (e.g., Pritchard and Wood, 1983;
Thompson and Adams, 1984). These variables have also been examined for headache sufferers in headache and headache-free states (e.g., Haynes, Cuevas and Garron, 1982; Martin and Mathews, 1978).

Comparisons of electromyographic (EMG) levels between tension headache subjects and headache-free controls have not revealed consistent differences between groups. Although EMG levels have been reported to be higher for tension headache sufferers in the frontalis muscle (e.g., Cohen et al., 1982; Philips, 1977; Van Boxtel and Van der Ven, 1978) many other researchers have been unable to replicate these results (e.g., Anderson and Franks, 1981; Andrasik et al., 1982; Bakal and Kaganov, 1977; Martin and Mathews, 1978; Pearce, 1977; Pritchard and Wood, 1983; Sutton and Belar, 1982; Traue, Gottwald, Henderson and Bakal, 1985). Bakal and Kaganov (1977) and Traue et al (1985) reported neck EMG levels to be higher for tension headache sufferers than controls, but numerous studies have not confirmed this observation (e.g., Feuerstein et al., 1982; Martin and Mathews, 1978; Philips, 1977; Pozniak-Patewicz, 1976). No differences between tension headache sufferers and controls have been observed on the trapezius (Philips, 1977) or temporalis muscle (Philips, 1977; Van Boxtel and Van der Ven, 1978).

It has been suggested that the muscles of the shoulders, neck or cranium may be more reactive to stress for tension headache sufferers than for controls (Philips, 1977). However, studies comparing EMG responses to laboratory induced stress for tension headache and control subjects have provided equivocal results. Some researchers have reported increased EMG levels under stress for tension headache sufferers relative to controls on the frontalis muscle (e.g., Cohen et al., 1982; Philips and Hunter, 1982; Traue et al., 1985; Thompson and Adams, 1984), the neck muscles (e.g., Traue et al., 1985), the temporalis muscles (e.g., Thompson and Adams, 1984) and occipital muscles (e.g., Pritchard and Wood, 1983). However, other studies have found no differences between groups in their muscular reactions to stress on the frontalis (e.g., Anderson and Franks, 1981; Andrasik, Blanchard, Arena, Saunders and Barron, 1982; Bakal and Kaganov, 1977; Feurstein et al., 1982; Gannon, Haynes, Safranek and Hamilton, 1981; Martin and Mathews, 1978; Sutton and Belar, 1982; Vaughn, Pall and Haynes, 1977), neck (e.g., Bakal and Kaganov, 1977; Martin and Mathews, 1978) or temporalis (e.g., Philips and Hunter, 1982) muscles. Thus, there exists a sizeable body of evidence undermining the notion that the muscles of the neck or scalp are more reactive to stress in tension headache sufferers than in headache-free controls.

Some researchers have reported tension headache sufferers to have higher resting frontalis EMG levels than migraineurs (e.g., Cohen et al., 1982), whilst others have observed lower resting EMG levels for tension headache sufferers than for migraineurs (e.g., Anderson and Franks, 1981; Bakal and Kaganov, 1977). Many Other studies have reported no
differences between groups on resting EMG levels at the frontalis (e.g., Andrasik, Blanchard, Arena, Saunders and Barron, 1982; Arena et al., 1985; Philips and Hunter, 1982), temporalis (e.g., Philips and Hunter, 1982), occipital (e.g., Pritchard and Wood, 1983) or neck (e.g., Arena et al., 1985; Bakal and Kaganov, 1977) muscle sites. Similarly, although some studies have found tension headache sufferers to have higher frontalis and temporalis EMG levels than migraineurs under conditions of laboratory stress (e.g., Cohen et al., 1982; Thompson and Adams, 1984), other studies have found no differences during stress between these headache types on the frontalis (e.g., Anderson and Franks, 1981; Andrasik, Blanchard, Arena, Saunders and Barron, 1982; Arena et al., 1985), temporalis (e.g., Philips and Hunter, 1982) or neck (e.g., Arena et al., 1985) muscles. These results argue against the notion that elevated neck or scalp muscle tension distinguishes tension from migraine headache (Ad Hoc Committee, 1962).

Studies of EMG levels in the same tension headache sufferers in headache and headache-free states have found no consistent evidence for increased tension in the frontalis muscle during headache (e.g., Arena et al., 1985; Haynes et al., 1983; Philips, 1977; Philips and Hunter, 1981; Thompson and Adams, 1984). In direct contradiction to the muscle-contraction theory of tension headache, Martin and Mathews (1978) found frontalis tension levels to be lower in the headache than in the headache-free state. Haynes et al (1983) reported neck EMG levels to be higher during the headache state, but other studies have reported no such differences at this site (e.g., Arena et al., 1985; Martin and Mathews, 1978; Philips, 1977). With respect to other muscle sites, no differences in EMG levels between headache and headache-free states for tension headache sufferers have been observed on the temporalis (e.g., Philips, 1977; Philips and Hunter, 1982; Thompson and Adams, 1984) or trapezius (e.g., Philips, 1977) muscles. Furthermore, it has been reported that frontalis and temporalis muscle tension levels, for tension headache sufferers in the headache state, are no greater than for headache-free controls or for migraineurs in headache or headache-free states (Thompson and Adams, 1984). These findings indicate that neck or cranial muscle tension may not represent the primary source of pain in tension headache.

Several researchers have observed no significant relationship between EMG levels and locus (e.g., Bakal and Kaganov, 1979) or intensity (e.g., Haynes et al., 1983; Thompson and Adams, 1984) of reported headache pain. Moreover, Anderson and Franks (1981) observed a significant negative correlation between frontalis EMG levels and ratings of tension headache pain.

It has been suggested that it may be the duration for which the musculature is tense, rather than the actual magnitude of that tension, that may be critical to the mechanism of tension headache (Pearce, 1977; Philips, 1978). Pearce (1977) required tension, migraine and headache-free controls to contract the frontalis muscle at a constant level for one to three
minutes and to rate the intensity of any ensuing pain. In each of these studies no significant differences between groups on the threshold for pain and ratings of pain intensity were observed. In a replication of this study, Philips (1978) noted that pain ratings remained very low in tension headache sufferers despite the fact that, during the experiment, frontalis tension levels increased well above the levels observed during headache attacks.

Finally, the muscle contraction theory of tension headache is called further into question by the now substantial body of evidence indicating that in the EMG biofeedback treatment of tension headache, where the aim is to provide relief by reducing muscle tension levels, there is often no association between headache improvement and changes in EMG levels at the frontalis (e.g., Abramowitz and Bell, 1985; Blanchard et al., 1982; Epstein and Abel, 1977; Holroyd, Andrasik and Westbrook, 1977; Martin and Mathews, 1978; Philips and Hunter, 1981b), temporalis (e.g., Philips and Hunter, 1981b) or neck (e.g., Martin and Mathews, 1978) muscle sites.

It would appear that the bulk of the evidence on the psychophysiology of tension headache does not support the view that muscle contraction is essential to the aetiology of the disorder. Neither does this evidence indicate that tension headache can be differentiated from migraine by the degree of muscle contraction.

2.3. The Vascular Theory of Migraine.

Since the pioneering work of Wolff and his colleagues (e.g., Graham and Wolff, 1938; Wolff, 1963; Tunis and Wolff, 1953), it has been widely believed that a two-phase process of intracranial vasoconstriction, followed by extracranial vasodilatation, underlies migraine headache attacks (e.g., Ad Hoc Committee, 1962; Dalessio, 1980). Pain was considered to be associated with distension of the temporal artery on the painful side (Graham and Wolff, 1938; Tunis and Wolff, 1952). Thus, dilatation of the extracranial vasculature was claimed to constitute the source of pain in migraine. However, it was recognised that vasodilatation itself was not sufficient for a complete account of the pain of migraine. For example, distension of the superficial temporal artery was observed in migraineurs outside headache attacks (Wolff, 1963) and the locus of pain during attacks was often unilateral despite dilatation of the temporal arteries on both sides of the head (Graham and Wolff, 1938). Wolff (1963) sampled the periarterial fluid at the site of migraine attacks and identified an inflammatory polypeptide which he named neurokinin. Wolff (1963) proposed that the pain of migraine arises from the distension of extracranial blood vessels rendered pain sensitive by inflammatory substances.
In the present review the evidence pertaining to vasomotor instability in migraine will be considered, before moving on to a discussion of the role of local chemical action at the site of pain.

Investigations of the vascular theory of migraine have focused on comparisons of blood flow in migraineurs during and between headache attacks (e.g., Drummond and Lance, 1983; Tunis and Wolff, 1953) and on the vascular reactivity of migraineurs in comparison with control groups (e.g., Drummond and Lance, 1981; Feurstein et al., 1982). These two lines of research are reviewed separately in the following sections.

2.3.1. Vascular Processes During and Between Migraine Attacks.

Tunis and Wolff (1953) studied ten migraineurs and observed a relationship between temporal pulse amplitude and the intensity of headache. Other researchers, however, have reported difficulties in obtaining reliable measures of scalp artery pulse amplitudes and did not identify any significant relationship between these measures and headache intensity (e.g., Brazil and Friedman, 1956; Heyck, 1969). Furthermore, it has been pointed out that the magnitude of the temporal artery pulses, observed during migraine attacks by Tunis and Wolff (1953), were no greater than those observed in the same subjects when they were headache-free (Blau, 1978).

By monitoring the flow of the radio-active substance Xenon-133 in the extracranial vasculature, Sakai and Meyer (1978) demonstrated that extracranial blood flow, throughout the entire scalp, was about 50% greater for subjects with a migraine headache than for headache-free controls. However, Thompson and Adams (1984) monitored the temporal artery pulse amplitude on both sides of the head, and found no differences between migraineurs with headache and non-headache controls. Similar results were reported by Arena et al (1985).

Drummond and Lance (1983) studied pulse amplitudes in migraine sufferers experiencing attacks of unilateral headache. Recordings, made from the main trunk of the temporal artery on the headache side and from the frontal branch of the temporal artery at the site of pain, were compared with those made from the corresponding regions on the headache-free side. The pulse amplitude in the frontal branch of the temporal artery was significantly greater on the headache than on the headache-free side. No significant difference was observed for the main trunk of the temporal artery. Using thermography to measure temperature asymmetry in the face and scalp, Drummond and Lance (1983) observed asymmetries of 0.5°C or more (i.e., warmer temperatures on the headache than on the headache-free side) at temporal sites for 37 of 57 migraine subjects compared with 22 of 50 headache-free control subjects. In the region of the orbit, temperature asymmetry was evident in 25 migraineurs and
10 of the controls. In each of these instances, the frequency of asymmetries was significantly greater for the migraineurs than for the controls. The thermographic readings were validated as measures of extracranial blood flow by showing them to be correlated positively and significantly with the size of the amplitude pulse measured at the frontal branch of the temporal artery (Drummond and Lance, 1983).

Drummond and Lance (1983) identified a subgroup of their migraineurs (N = 22) for whom the intensity of headache could be reduced by at least 50% through compression of the ipsilateral carotid and/or superficial temporal artery. These subjects were considered to have an extracranial vascular basis to their headache. For this subgroup there was once again no difference in temporal artery pulse amplitude between the headache and headache-free sides. However, in 14 of these subjects, for whom pain was specific to the frontotemporal region, a significantly greater pulse amplitude was observed over the frontal branch of the temporal artery ipsilateral to headache. Thermographic readings for the extracranial group indicated that the affected side was warmer than the headache-free side in 11 out of 18 subjects, this proportion being significantly greater than that observed in the remaining subjects.

The results obtained by Drummond and Lance (1983) indicate that there may be an increase in extracranial blood flow on the headache side in about two thirds of persons experiencing unilateral migraine (as assessed by thermographic changes), but that the temporal artery and its branches are involved in only about one third of individuals suffering this kind of headache attack. The results suggest that there are likely to be many migraine sufferers for whom extracranial vascular blood flow has little to do with their headaches.

Studies of cerebral blood flow using the Xenon-133 method have yielded consistent results suggesting that cerebral blood flow is reduced during the prodromal phase of classical migraine (e.g., Edmeads, 1977; Olesen, Larsen and Lauritzen, 1981; Olesen, Lauritzen, Tfelt-Hansen, Henriksen and Larsen, 1982; Sakai and Meyer, 1978). Although some studies report increased cerebral blood flow during the painful phase of the attack (e.g., Edmeads, 1977; Sakai and Meyer, 1978), Olesen et al (1981) observed an increase relative to baseline in only four of eight subjects studied and in a later investigation (Olesen et al., 1982), a reduction in cerebral blood flow was observed during the headache phase.

Studies of cerebral and extracranial blood flow during and between episodes of migraine, have failed to provide unequivocal support for the vascular theory first proposed by Wolff and his co-workers (Graham and Wolff, 1938; Tunis and Wolff, 1953; Wolff, 1963). It would appear that although increases in cerebral (e.g., Sakai and Meyer, 1978) and extracranial (e.g., Tunis and Wolff, 1953) blood flow do sometimes accompany the occurrence
of migraine headache, these increments are not necessary to the pain phase of the attack (e.g., Drummond and Lance, 1983; Olesen et al., 1982).

2.3.2. Vascular Reactivity in Migraine Sufferers.

Tunis and Wolff (1953) considered that, even when migraine sufferers are free from headache, the pulsation of their superficial temporal arteries would be stronger than that observed for persons who are rarely troubled by headache. Wolff (1953) observed the scalp arteries of migraineurs to dilate readily during discussion of threatening issues. He considered that a hyper-reactivity of the extra-cranial vascular system, particularly in the face of stress, represents a predisposition to migraine (Wolff, 1963). However, investigations into the vascular functioning of migraine subjects (e.g., Andrasik et al., 1982; Arena et al., 1985; Drummond, 1985) have not supported Wolff's (1953, 1963) hypothesis that stress contributes to migraine through the mechanism of vasodilatation.

Studies of headache-free migraineurs, under standard baseline conditions, have shown consistently that their temporal pulse amplitudes are no different from those observed for control subjects or tension headache sufferers (e.g., Andrasik, Blanchard, Arena, Saunders and Bron, 1982; Bakal and Kaganov, 1977; Drummond, 1985; Drummond and Lance, 1981; Feurstein et al., 1982; Thompson and Adams, 1984). Baseline measures of general vascular functioning such as blood pressure, digital finger temperature and digital pulse amplitude, have also been reported not to differ across these groups (e.g., Anderson and Franks, 1981; Andrasik, Blanchard, Arena, Saunders and Barron, 1982; Arena et al., 1985; Feurstein et al., 1982; Morley, 1985), although Drummond has reported greater baseline digital pulse amplitudes (Drummond, 1984) and heart rates (Drummond, 1984, 1985) for migraineurs than controls.

Under conditions of laboratory stress, such as stressful imagery, pain, mental arithmetic or loud noise, no consistent pattern of temporal artery amplitude response has characterised migraineurs as distinct from headache-free controls or tension headache subjects, with many researchers reporting no differences between groups (e.g., Andrasik, Blanchard, Arena, Saunders and Barron, 1982; Cohen et al., 1982; Drummond, 1985; Feurstein et al., 1982; Thompson and Adams, 1984). Drummond (1982) observed a greater increase in temporal pulse amplitude on the habitually affected side for migraineurs than for headache-free controls in response to mental arithmetic, but he observed no such differences between groups in response to such noxious stimuli as cold pressor pain and radiant heat. Furthermore, in a subsequent study (Drummond, 1985), no differences in temporal pulse amplitudes were observed between migraine, tension and control groups in response to a reaction time task or
mental arithmetic. Similarly, inconsistent findings have been reported by Morley (1985), who observed migraineurs to demonstrate greater temporal pulse amplitudes, on the habitually affected side of the head, than headache-free controls during a reaction time task, no difference in response during stressful imagery, and a reduction in temporal pulse amplitude relative to controls during exposure to noise. This latter finding was also observed by Bakal and Kaganov (1977).

With respect to the behaviour of the peripheral vasculature under laboratory stress, Morley (1985) reported the digital pulse amplitudes of migraineurs to be greater than those of headache-free control subjects under conditions of stressful imagery, but no differences between groups were found during a reaction time task or during exposure to noise. In other studies of the peripheral vasculature under stress, Feurstein et al (1982) were unable to distinguish between migraine, tension headache and headache-free controls on digital pulse amplitude, as were Gannon et al (1981) on ear lobe pulse amplitude. The same negative findings have been obtained for finger temperature (e.g., Anderson and Franks, 1981; Andrasik, Blanchard, Arena, Saunders and Barron, 1982). Arena et al (1985) were unable to distinguish between tension, migraine and tension-vascular headache sufferers on this measure in response to mental arithmetic, stressful imagery or cold pressor pain.

Drummond and Lance (1981) examined the effect of structured exercise on temporal artery pulsation in migraineurs, tension headache sufferers and headache-free controls. No differences between groups were identified, although migraineurs did demonstrate greater amplitudes on the habitually affected side than on the side that was usually headache-free.

Drummond (1984) selected 30 unilateral migraine sufferers for study. In a previous investigation using thermography (Drummond and Lance, 1983), 11 of these subjects were found to demonstrate warmer temperatures over the affected region during headache and/or to rate their headache intensity as being reduced by at least 50% after compression of the superficial temporal artery ipsilateral to headache. In the remaining 19 subjects, this pattern of response during unilateral headache had not been evident. Drummond (1984) studied these 30 subjects whilst they were headache-free and recorded temporal artery pulse amplitudes on the habitually affected side during periods of exercise and mental arithmetic. The 11 subjects who had been identified previously as having an extracranial vascular basis to their headaches, were found to demonstrate significantly larger temporal artery pulse amplitudes than the remaining migraineurs, in response to both the exercise and mental arithmetic tasks. These results suggest that scalp arteries dilate readily during stress and exercise in only a subgroup of migraineurs and that, therefore, this phenomenon cannot be regarded as crucial to the pathophysiology of migraine.
It is clear that the majority of the studies conducted on the behaviour of the extracranial vasculature of migraneurs during and between headaches, as well as under conditions of laboratory stress or exercise, do not support the thesis that the pain of migraine is always accompanied by vasodilatation of the scalp arteries (Graham and Wolff, 1938; Tunis and Wolff, 1953). Neither do the findings support the claim that the extracranial vasculature of all migraneurs can be characterised by a tendency towards vasodilatation (Wolff, 1953, 1963). Rather, it would seem that vasodilatation may contribute to pain in only a minority of migraneurs (Drummond and Lance, 1983) for whom there may be a heightened tendency for scalp arteries, at the usual site of pain, to dilate during periods of exercise or stress (Drummond, 1984). For many migraine sufferers, however, these aspects would appear to have no place in explanations of their pain.

Attempts have been made to treat migraine through biofeedback procedures aimed at assisting subjects to learn a vasoconstrictive response which can then, supposedly, be used to help combat the pain that was presumed to arise from vasodilatation of the scalp arteries. Feedback is received from the fingers (e.g., Attfield and Peck, 1979; Daly, Donn, Galliher and Zimmerman, 1983) or from one of the temporal arteries (e.g., Feurstein and Adams, 1977; Friar and Beaty, 1976). The status of these procedures will be considered in a Chapter 4. It is sufficient to note at this point that, within each of these treatments, it has been common place to observe no significant correlation between learned control over the peripheral or extracranial vasculature and improvements in the migraine condition (e.g., Cohen, McArthur and Rickles, 1980; Gauthier, Lacroix, Cote, Doyon and Drolet, 1985). Hence, these findings are also at odds with the extracranial vascular theory of migraine.

2.4. Humoral and Metabolic Factors in Migraine.

The view that local chemical action at the site of pain may contribute to migraine was first raised by Wolff's research group (Chapman, Ramos, Goodell, Silverman and Wolff, 1960), who identified an inflammatory polypeptide, which they called neurokinin, in the periarterial fluid at the site of headache. Since this time there have been many investigations into the blood chemistry of migraine.

Blood levels of serotonin have been found to be lowered during migraine attacks (Anthony, Hinterberger and Lance, 1967; Curran, Hinterberger and Lance, 1965; Hilton and Cummings, 1972). This substance has been found to constrict extracranial arteries both in the human (Lance, Anthony and Gonski, 1967) and in the monkey (Spira, Mylecharane and Lance, 1976). Thus, it has been suggested that the sudden withdrawal of serotonin from the circulation could contribute to headache by giving rise to extracranial vasodilatation, and by producing
inflammation of the vasculature as it is absorbed into the vessel wall (Anthony and Lance, 1975). Indeed, Sicuteri (1967) has shown that the injection of serotonin into veins on the hand potentiates the pain-producing effect of bradykinin. Enzymes which release bradykinin into the blood stream have been observed to increase during migraine (Sicuteri, Fanciullacci and Anselmi, 1963).

Blood levels of histamine have been shown to be higher in migraineurs, during and between headaches, relative to controls (Heatley, Denburg, Bayer and Bienenstock, 1982). The injection of histamine into the external carotid artery has been reported to produce pain (Sicuteri, 1967). Thus, substances such as serotonin, bradykinin and histamine, in various combinations, could increase the sensitivity of the extracranial vasculature and thereby contribute to the pain of migraine. It has been suggested that these substances combine with vascular dilatation to produce pain at the site of migraine headache (e.g., Anthony and Lance, 1975; Fanchamps, 1974). However, the fact that migraine often occurs in the absence of extracranial vasodilatation (Drummond and Lance, 1983) tends to weaken this position.

Fluctuating blood levels of serotonin have also been implicated in the occurrence of the nausea and vomiting which often accompany migraine attacks, as this substance could stimulate the vomiting centre in the medulla (Anthony and Lance, 1975).

The extent to which serotonin might contribute to headache through central nervous system mechanisms is considered in the next section.

2.5. The Central Theory of Headache.

Sicuteri (1976, 1978, 1982) conjectured that headache is a disorder of central nociception. That is, that headache sufferers are rendered pain sensitive by a depletion of pain inhibitory substances, such as serotonin, noradrenaline, endorphins and enkephalin in the endogenous pain control system (Basbaum and Fields, 1978, 1984), and that this constitutes the basis of tension and migraine headache. Sicuteri, Fanciullacci and Michelacci (1978) proposed that the depletion of central pain inhibitory substances occurs episodically in migraine, thereby, increasing the pain sensitivity of the intra and extracranial vasculature such that, normally unperceived vascular pulsations are experienced as painful. In the case of tension headache, alterations in muscle tension are assumed to interact with hyperaesthesia to produce pain.

In order to account for pain being experienced in the head rather than elsewhere in the body, a point which is generally underemphasised in the central theory, Sicuteri (1982)
suggested that the disruption of pain modulation that occurs during headache, may be specific to the rostral section of the endogenous analgesic system which subserves the shoulders, neck and head. However, this is hardly an adequate explanation for the pain of migraine which is often unilateral and specific to a particular region of the scalp (Lance, 1982). As Lance (1982) has pointed out, the fact that headache is often localised suggests that a conceptualisation in terms of a failure of sensory modulation needs to be supplemented by some hypothesis regarding peripheral processes, such as local chemical action (Chapman et al., 1960) or extracranial vasodilatation (Tunis and Wolff, 1953). It may be that it is because peripheral processes interact with varying levels of sensory modulation efficacy that the study of muscular and vascular processes per se has not revealed consistent relationships with headache.

Unfortunately, Sicuteri and his colleagues (Sicuteri, 1982; Sicuteri, Fanciullacci and Michelacci, 1978) are rather vague about the nature of the deficiency of central pain control mechanisms thought to characterise headache sufferers. It is not clear whether this deficiency is considered to be present at all times or only during episodes of headache. Furthermore, it has been suggested by this research group, on one occasion, that the deficiency is specific to the shoulders, neck and head (Sicuteri, 1982), and on other occasions, that it is a general condition affecting all regions of the body (Sicuteri, Anselmi and Del Bianco, 1978; Sicuteri, Fanciullacci and Michelacci, 1978). Obviously, the theory is in need of some clarification.

The central theory of headache reflects the influence of the gate control theory of pain (Melzack and Wall, 1965), and represents the first attempt to integrate the concept of a pain gate into an account of headache. The gate control theory is elaborated in Appendix A. It is sufficient to note at this point that the essential feature of the theory is the postulation of a pain gating mechanism within the central nervous system (CNS) which modulates afferent impulses before they reach the cortical structures subserving the experience of pain. Both the endogenous opioid system and brain stem monoamines have been implicated in the regulation of this mechanism (e.g., Basbaum and Fields, 1978, 1984; Lance and Bogduk, 1982), which is thought to be located within the substantia gelatinosa (Basbaum and Fields, 1978, 1984; Bogduk and Lance, 1981; Melzack and Wall, 1982). Investigations into the central theory of headache have focused on the role of serotonin, endogenous opiates and pain sensitivity.

2.5.1, Serotonin and Endogenous Opioids.

Sicuteri and his colleagues (Sicuteri, 1976; Sicuteri, Anselmi and Del Bianco, 1978) have argued that the lowered blood levels of serotonin observed to accompany migraine headaches (Anthony et al., 1967; Curran et al., 1965; Hilton and Cummings, 1972) might be
concurrent with a depletion of this monoamine within the CNS, thus causing the pain gate to be opened. The assertion that brain levels of serotonin may be chronically low in cases of daily tension headache (Sicuteri, 1982), derives some support from the observation that blood levels of serotonin are lower in persons suffering daily tension headaches than in headache-free controls (Anthony and Lance, 1985; Rolf, Wiele and Bruner, 1981). However, the extent to which one can extrapolate from blood levels to brain levels of amines is uncertain (Fozard, 1982).

In support of their theory, Sicuteri and his co-workers (e.g., Sicuteri, 1976; Sicuteri, Anselmi and Del Bianco, 1978) often cite their observation that the administration of para-chlorophenylalanine (an inhibitor of the enzyme tryptophan hydroxylase, essential to the body's manufacture of serotonin) produces pain in the skin and muscles of some migraineurs (Sicuteri, Anselmi and Del Bianco, 1973). However, since the drug produced pain in only 4 of 16 migraine subjects and given that, even in these 4, pain was experienced elsewhere in body as well as in the head, this evidence can be discounted.

Studies of cerebrospinal fluid (CSF) levels of endogenous opioids have been supportive of the involvement of pain inhibitory substances in headache. For example, migraineurs between headache attacks have been shown to have lower CSF levels of beta-endorphin (Genazzani et al., 1984; Nappi, Facchinetti, Martignoni, Petraglia, Bono et al., 1985; Nappi, Facchinetti, Martignoni, Petraglia, Manzoni et al., 1985). CSF beta-endorphin levels between attacks have also been found to be negatively correlated with clinicians' ratings of migraine headache severity (Nappi, Facchinetti, Martignoni, Petraglia, Bono et al., 1985; Nappi, Facchinetti, Martignoni, Petraglia, Manzoni et al., 1985). Furthermore, CSF levels of enkephalin have been observed to be lower during migraine attacks than between attacks (Anselmi, Baldi, Casacci and Salmon, 1980). To date, no studies of CSF morphine-like substances have been conducted with tension headache sufferers.

Investigations of blood plasma levels of endogenous opioids in headache sufferers have been less consistent. In the case of daily tension headache, some studies report lower plasma levels of beta-endorphin (e.g., Baldi et al., 1982; Facchinetti, Nappi, Savoldi and Genazzani, 1981; Nappi, Facchinetti, Martignoni, Petraglia, Bono et al., 1985; et al., 1985a) for these subjects than for headache-free controls. However, Fettes, Gawel, Kuzniak and Edmeads (1983) observed no difference between these groups. Furthermore, subjects with episodic common migraine studied between attacks, have been found to have plasma beta-endorphin levels no different from those of headache-free controls (e.g., Fettes et al., 1983; Nappi, Facchinetti, Martignoni, Petraglia, Bono et al., 1985; Nappi, Facchinetti, Martignoni, Petraglia, Manzoni et al., 1985), although Fettes et al (1983) did observe diminished plasma beta-endorphin levels in classical migraine sufferers relative to controls. Finally, in subjects
suffering migraine with interparoxysmal headache, plasma levels of beta-endorphin have been found to be lower than in headache-free controls (Nappi, Facchinetti, Martignoni, Petraglia, Bono et al., 1985; Nappi, Facchinetti, Martignoni, Petraglia, Manzoni et al., 1985). Thus, it would appear that plasma levels of beta-endorphin are most likely to be lowered when headache is constant or when subjects are assessed during headache attacks.

2.5.2. Pain Sensitivity.

An alternative to the biochemical line of inquiry into the central theory is offered by studies of the pain sensitivity of headache sufferers (e.g., Gannon et al., 1981; Martin and Mathews, 1978). Surprisingly, very few of these investigations have been conducted.

Sicuteri, Anselmi and Del Bianco (1978) reported on a study of pain sensitivity in headache sufferers and headache-free controls employing an electrical tourniquet device relying upon muscular work to induce pain. For headache sufferers, the pain threshold was found to be lower and the painful after-effects of the procedure were found to persist for longer, than for control subjects. Unfortunately, Sicuteri, Anselmi and Del Bianco (1978) provided little in the way of methodological detail making their results difficult to interpret. For example, it is not stated whether or not the headache sufferers were experiencing headache during the assessment of pain sensitivity.

Gannon et al (1981) employed an occlusion cuff inflated to 220 mm Hg to induce pain in the arm. Comparing a mixed group of headache sufferers with control subjects, there was a trend for the proportion of headache sufferers requesting termination of the cuff to be greater than the proportion of controls; the size of the difference fell just short of significance (p = 0.06).

Feurstein et al (1982) employed the Forgione-Barber Pain Stimulator (Forgione and Barber, 1971), a method of applying pressure pain to the fingers, but found no differences between tension, tension-vascular, migraine headache and control subjects on pain threshold, tolerance or intensity ratings. All headache sufferers were headache-free. Unfortunately, the number of subjects within each group was very small, there being only eight subjects in the control group.

Drummond (1987) also employed the Forgione-Barber Pain Stimulator. He assessed the rate of increase in pressure pain over time and found no differences between migraine, tension headache and headache-free controls regardless of the presence or absence of headache. However, for a sub-group of the tension headache sufferers with episodic headaches, pain
increased more rapidly than for the remaining subjects. For both the migraine and tension headache sufferers the pain threshold was greater during than between headaches, but did not differ from the threshold for control subjects.

Langemark and Olesen (1987) reported that the pericranial muscles of tension headache sufferers, during headache, are more sensitive to palpation than those of headache-free controls. Unfortunately, these researchers did not assess headache sufferers between attacks. The increased sensitivity they observed could have arisen simply from the pain of headache, rather than from any deficiency of sensory modulation.

Martin and Mathews (1978) employed thermal stimuli focused on the forehead and found no significant difference between the pain thresholds of tension headache sufferers and headache-free controls. Many of the headache subjects were experiencing a headache at the time of the assessment. Painful stimulation of one part of the body is known to elevate the pain threshold elsewhere in the body (e.g., Chen, Treede and Bromm, 1985; Le Bars, Roby and Willer, 1983). Thus, the presence of headache in some subjects could have obscured any differences between groups that may have otherwise emerged.

Consuming cold drinks or ice cream can evoke mild headache in some individuals, this phenomenon being termed "ice cream" headache (Lance, 1982; Mumford, 1979). Raskin and Knittle (1976) found that 93% of 59 migraine subjects admitted to having experienced ice cream headache compared with 31% of 49 headache-free subjects. Furthermore, Drummond and Lance (1984b) found that the prevalence of ice cream headache increased with the number of migrainous features associated with the subject's usual headache and that, in 30 of 90 headache subjects reporting ice cream headache, the site of pain corresponded to the site of their usual headache. Drummond and Lance (1984b) also found the reported location of sharp, jabbing "icepick-like" pains corresponded to the usual site of headache in 37 of 92 subjects studied. These findings suggest that, for some headache sufferers, a heightened sensitivity to pain may exist at the site of headache during periods of headache freedom. Indeed, employing the pressure algometer (Keele, 1954), Drummond (1987) observed scalp tenderness between headache attacks to be greater at the usual site of headache than at other locations for both tension and migraine headache sufferers. Scalp tenderness was also greater for headache subjects assessed between attacks than for headache-free controls. Thus, it is possible that some localised hypersensitivity to pain contributes to the location of headache during any failure of the central pain control system.

The assumption that there exists some weakness in the pain inhibitory systems of headache sufferers, rendering them sensitive to pain, has yet to be thoroughly explored. It would appear that headache sufferers between attacks may be more sensitive than controls to
pressure pain in the head (Drummond, 1987). However, there is very little evidence to support the hypothesis that headache sufferers are hypersensitive to pain in a variety of regions in the body (Sicuteri, Anselmi and Del Bianco, 1978). More research, carefully matching subjects on factors such as age and sex, is required on this point.

Sicuteri (1982, p. 72) has suggested that psychological factors may contribute to headache through their action on a weakened pain inhibitory system:

"if...(the endogenous antinociceptive system)...is efficient, stable and adaptable emotions leave it unimpaired or reinforce it. If the pain suppressor mechanism is phenotypically or genotypically fragile, repetitive emotional stress can provoke a weakening, a deterioration and ultimately a failure of the analgesizing apparatus resulting in hyperalgesia and spontaneous pain."

There is some evidence to support Sicuteri's (1982) view that stress could contribute to headache through an effect on pain inhibitory processes. The effect of stress on the opioid and aminergic systems is considered in the following sections.

2.5.3. Stress and the Opioid System.

Exposing animals to painful stimulation has been observed to activate endogenous opioid activity, stress-induced analgesia being attenuated by the administration of naloxone (an opiate antagonist) (e.g., Kelly, 1986). Providing animals with the capability to switch off electric shocks has been observed to attenuate opioid activation (e.g., Maier, 1986). Thus, it may be the stress arising from an absence of control that activates the opioids rather than mere exposure to pain (Maier, 1986).

Akil, Madden, Patrick and Barchas (1976) exposed rats to inescapable foot-shock for periods of up to an hour in length and then tested their sensitivity to heat (tail flick test). They found these "stressed" rats to be less reactive to the heat test relative to baseline and controls. These analgesic effects were accompanied by increased brain levels of morphine-like substances and could be diminished by naloxone. However, Akil et al (1976) also noted that when rats were repeatedly exposed to shock (every day for 12 days), there was an habituation of both responses, with the reaction latencies and brain opiate levels returning to their baseline levels.

Thus, while acute stress may precipitate an initial increase in morphine-like substances within the brain and concomitant analgesia, these effects tend to be absent under conditions of chronic stress. Hence, Rossier, Bloom and Guilleman (1980) have reported a series of studies
demonstrating lowered levels of beta-endorphin and leu-enkephalin in the hypothalamus of the rat brain, following prolonged foot-shock.

Although it is very difficult to generalize the results of these studies to situations of human stress, they raise the possibility that exposure to repeated stress could contribute to headache by reducing the level of brain opiates.

2.5.4. Stress and the Aminergic System.

It has been demonstrated that when rats are exposed to repeated ether stress, noradrenaline and serotonin levels are reduced in most brain regions (Telegdy, Fekete and Varszegi, 1980). Telegdy and Vermes (1976) have shown that exposing rats to electric shock reduces the level of serotonin in the limbic system and decreases the rate of synthesis of the amine. Although the above results are to an extent dependent on the intensity and type of stressor employed (Telegdy and Vermes, 1976) other researchers have observed similar effects for immobilization stress (Palkovits, Brownstein, Kiszer, Saavedra and Kopin, 1976) and activity-wheel stress (Hellhammer, Rea, Bell and Belkien, 1984).

The release of catecholamines into the blood stream which typically occurs in humans experiencing laboratory stress (Cox, Cox and Thirlaway, 1983; Forsman and Lindblad, 1983), has been shown to increase platelet monoamine oxidase (MAO) activity (Owen, Acker, Bourne, Firth and Riley, 1977). Hence, increased monoamine oxidase activity secondary to stress, may provide an additional route by which brain levels of serotonin could be reduced (Mathew and Ho, 1981).

Finally, catecholamine release has been shown to lead to the accumulation of free fatty acids (Carlson, Levi and Oro, 1968), which in turn release serotonin from platelets and occasion its rapid removal from the blood stream (Anthony, 1978). If such a process of serotonin depletion were to occur in the brain stem, this would constitute yet another avenue through which stress could conceivably contribute to headache through an action on brain levels of serotonin.

2.6. General Comments.

The central theory of headache suggests that stress contributes to headache by acting upon a fragile pain inhibitory system. Although there seems to be some support for this position, the main problem confronting the theory is one of explaining why pain is specific to
the cranium and, moreover, why headache is often confined to a particular region of the scalp. It would seem that in the last analysis, one has to postulate an interaction between central and peripheral factors in order to account for the clinical features of tension and migraine headache (e.g., Lance, 1982). The central theory of headache, however, has the potential to facilitate research on psychological aspects of headache, insofar as psychologists need not be required to articulate theories of the relationship between psychological factors and headache to peripheral physiological processes. For instance, as discussed previously, seeking relationships between stress, muscle contraction or the process of vasoconstriction-vasodilation has not proved to be a particularly successful line of inquiry into the relationship between stress and headache. Research might profit from the assumption that psychological factors may contribute to headache through central pain inhibitory processes.

This thesis is concerned with a description of the role of cognitive processes in the relationship between stress and headache, and with the prospects for linking these processes to the central mechanisms involved in sensory modulation. As a first step towards these goals, the next chapter deals with the literature on the role of psychological variables in headache. The concept of a cognitive process is discussed in Chapter 5.
CHAPTER 3

PSYCHOLOGICAL VARIABLES AND HEADACHE.

3.1. The Concept of Stress.

Despite the fact that the concept of stress has generated a wealth of research in psychology and medicine over recent decades, theoreticians and researchers have had little success in defining the term.

Stress has generally been conceptualised in one of three ways: as a state of the organism characterised by increases in sympathetic nervous system activity (e.g., Selye, 1956; Wolff, 1953) which may occur in response to some threat or demand; in terms of the stimulus properties of events that may compromise the well-being of the individual (e.g., Holmes and Rahe, 1967) or as a transaction between the environment and the individual, mediated by the process of cognitive appraisal (Lazarus and Folkman, 1984a, b; Lazarus, De Longis, Folkman and Gruen, 1985).

Each of the above definitions of stress may be criticised on the grounds that they are vague and question-begging. Selye defined stress as "the body's non-specific response to any demand placed on it, whether that demand is pleasant or not." (Selye and Cherry, 1978, p. 60). This confines the term stress to a description of a certain constellation of biological processes. As Hinkle (1973) has pointed out, such usage renders the term stress superfluous. One might just as well describe the biological processes without recourse to the confusing concept of stress.

To define stress in terms of the stimulus properties of events is clearly inadequate for it does not take into account the meaning or significance attached to the event by the individual on whom it impinges. For example, divorce is ranked second on the Social Readjustment Rating Scale (Holmes and Rahe, 1967). For the person who is very dependent on their spouse, this could indeed be very distressing. However, for the person who cares little for their spouse, or who is in love with someone else and wishes to marry them, the life event "divorce" takes
on a completely different meaning. As those favouring an interactional approach to the understanding of stress have argued:

"Without some cognitive mediational concept, we could never account for individual differences in the levels of stress response displayed to common environmental conditions." (Lazarus and Folkman, 1984a, pp. 289-290).

The interactionists have defined stress in terms of the balance between the person's appraisal of how much is at stake and their evaluation of their capacity to manage or escape from the perceived threat (Cox and Mackay, 1978; Lazarus and Folkman, 1984a, b). This definition has the advantage of incorporating both the stimulus characteristics of the event and the response of the organism. However, although the definition provides us with some idea of the conditions under which stress might be said to arise, it makes no statement about the nature of stress itself.

The demand for a complete definition of stress seems unlikely to be met. Popper (1966) has argued that demands for a definition inevitably lead to an infinite regress: the terms used to define the concept in question must themselves be defined, and so on ad infinitum. Popper suggests that one reason why the physical sciences have outstripped the social sciences in terms of progress, may be because the latter have been pre-occupied with the precision of definitions, whilst the former have been relatively unconcerned with questions such as "what is light?" or "what is matter?" Popper insists that a pre-occupation with matters of definition, "is itself the main source of vagueness, ambiguity and confusion" (Popper, 1966, p. 19). He urges scientists to eschew questions such as "what is matter?" and to address themselves instead to questions such as "how does this particular piece of matter behave under certain specific conditions?"

Given that the focus of this thesis is on the role of cognitive processes in headache, the interactional conceptualisation of stress, involving a transaction between the environment and the individual, mediated by cognitive appraisal, will be employed as a working definition. The shortcomings of this definition are recognised, but it is employed on the grounds that it may set the stage for a broader understanding of the relationship between stress and headache than one couched exclusively in terms of either the biological reaction of the organism or the stimulus properties of the environment. The cognitive processes involved in theoretical models of stress are considered in Chapter 5.
3.2. Life Events and Headache.

Clinical interviews with headache sufferers have been supportive of the view that stressful events are the most common precipitants of headache attacks (Friedman, 1979; Friedman, von Storch and Merritt, 1954; Howarth, 1965; Selby and Lance, 1960). However, comparisons of headache sufferers and headache-free control subjects on the Social Readjustment Rating Scale, administered with respect to the last twelve months (Blanchard, Radnitz, Evans and Schwartz, 1986) or with respect to the last six months (Andrasik, Blanchard, Arena, Teders et al., 1982) of the subject's life, have failed to reveal significant differences in the severity of life events reported. Using a structured interview method, Invemizzi, Gala and Sacchetti (1985) found headache sufferers to report more life events occurring over the last twelve months than controls.

The above approaches to the assessment of life events are likely to be insensitive to the relatively minor, but more frequent, problems of everyday life that might be associated with the onset of specific headache episodes. When subjects report that stressful events are the most common headache trigger factors (e.g., Henryk-Gutt and Rees, 1973), it is likely that they are referring to the recurrent difficulties of day to day living, rather than to much more significant events such as the death of a friend or the birth of a baby, which would remain salient over a six to twelve month period.

In an effort to tap less significant events, Levor, Cohen, Naliboff, McArthur and Heuser (1986) required migraine sufferers to keep diaries of the number of stressful events they experienced each day, as well as records of their headaches. More stressful events were observed to occur in four day periods leading up to migraine attacks than in corresponding four day periods preceding headache-free days.

Holm, Holroyd, Hursey and Penzien (1986) found that although tension headache sufferers did not differ from controls in the number of life events reported on an adapted version of the Social Readjustment Rating Scale, the headache sufferers reported a greater frequency of common, everyday stressful events, as assessed on the Hassles Scale (Kanner, Coyne, Schaefer and Lazarus, 1981). Holm et al (1986) found their headache sufferers to appraise themselves as having less control over stressful events; to report using higher levels of avoidance and self-blame coping strategies and to be less likely to make use of social support than headache-free controls. Such strategies have been reported to be relatively ineffective ways of coping (e.g., Folkman and Lazarus, 1986; Manne and Sandler, 1984; Wethington and Kessler, 1986).
Employing the Unpleasant Events Schedule (Lewinsohen, Mermelstein, Alexander and MacPhillamy, 1985), which, like the Hassles Scale, is designed to assess frequent stressful events, Kearney, Wilson and Haralambous (1987) found no differences between headache sufferers, pain-free controls and tinnitus patients.

Thus, comparative psychometric studies have yielded inconsistent results on the relationship between stressful events and headache. However, even if one took the conservative view that the frequency of stressful events is equivalent for headache sufferers and controls, this would not rule out the hypothesis that stressful events may trigger headache attacks in headache sufferers. However, it would imply that some predispositional factor would need to be postulated to interact with stressful events to produce headache.

At the present time, the evidence for the existence of a link between stressful events and headache draws most heavily on unstructured retrospective judgements made by headache sufferers themselves (e.g., Friedman et al., 1954; Howarth, 1965). A greater understanding of the functional relationship between such events and headache, awaits the completion of additional prospective studies of the kind reported by Levor et al (1986).

3.3. Personality and Headache.

To the extent that personality factors contribute to individuals' appraisals of and reactions to stressful situations, it has been suggested that aspects of personality might be important variables in the aetiology of headache (e.g., Friedman, 1979; Howarth, 1965; Wolff, 1937).

Much of the evidence for the existence of a specific constellation of personality traits characteristic of tension or migraine headache sufferers is based upon clinical impressions of patients presenting at headache or neurology clinics (e.g., Howarth, 1965; Martin, Rome and Swenson, 1967; Ostfeld, 1962). Claims that migraine headache sufferers are perfectionistic, compulsive, rigid and repressive of hostile feelings (e.g., Alvarez, 1974; Friedman et al., 1954; Wolff, 1937), and that tension headache sufferers are anxious, dependent, hostile and psychosexually conflicted (e.g., Bihldorff, King and Pames, 1971; Martin, 1966; Martin et al., 1967) are likely to be contaminated by the preconceptions of the investigator. Moreover, these inferences are based upon biased samples as less than 50% of headache sufferers actually seek medical advice (Waters and O'Connor, 1970).

Headache sufferers presenting to outpatient clinics have been found to score in the more pathological direction than headache-free controls on various scales of the Minnesota
Multiphasic Personality Inventory (MMPI; Hathaway and McKinley, 1951) (e.g., Kudrow and Sutkus, 1979; Stembach, Dalessio, Kunzel and Bowman, 1980) and on the Neuroticism Scale of the Eysenck Personality Inventory (EPI; Eysenck and Eysenck, 1964) (e.g., Henryk-Gutt and Rees, 1973; Kumar, 1985). However, a number of studies employing non-clinic headache sufferers have not identified significant elevations on the MMPI (Henryk-Gutt and Rees, 1973) or on the clinical scales of the EPI (Henryk-Gutt and Rees, 1973; Philips, 1976). Employing a group of tension, migraine and tension-vascular headache sufferers, half of whom were clinic patients and half of whom were not, and comparing these with headache-free control subjects on the Maudsley Obsessive-Compulsive Inventory (Rachman and Hodgson, 1980), Arena, Blanchard, Andrasik and Applebaum (1986) observed no differences between groups. These findings support the view that clinical samples of headache subjects may report more personality problems than non-clinic samples, and that the study of biased samples may have resulted in an exaggeration of the personality characteristics of headache sufferers (Blanchard and Andrasik, 1982; Philips, 1976).

Blanchard, Andrasik and Arena (1984) administered a wide range of personality tests to 166 headache subjects and 63 headache-free controls. The headache sufferers were divided into cluster, migraine, tension and tension-vascular groups and compared with the controls. On several of the measures, no differences between groups were found. However, on the State and Trait Anxiety Inventories (Spielberger, Gorsuch and Lushene, 1970), Psychosomatic Symptom Checklist (Derogatis, Richels and Rock, 1976) and on the Hypomania, Depression, Hysteria, Paranoia and Psychasthenia Subscales of the MMPI significant differences between groups were observed. Tension headache sufferers scored higher than the tension-vascular group, followed by the migraineurs and, finally, the cluster and headache-free controls who obtained the lowest scores and did not differ significantly from each other on any measure. This general ordering was consistent for all these tests; the significance of the differences between groups varying from test to test. A similar trend was apparent on the Beck Depression Inventory (Beck, Ward, Mendelsohn, Mock and Erbaugh, 1961), except that the migraineurs were more depressed than the tension-vascular group.

Blanchard et al (1984) concluded that the level of psychological disturbance seems to be a function of the amount of time headache sufferers have to endure pain. A similar conclusion was offered by Stembach et al (1980) who also employed the MMPI. Tension headache sufferers may be rarely pain-free, whilst migraine and tension-vascular headache sufferers tend to have more episodic headaches and bouts of cluster headache generally occur only once or twice per year (Lance, 1982). Blanchard et al (1984) speculated that the personality characteristics of headache sufferers might be a consequence of the amount of pain they experience, rather than predispositional factors playing a causal role in the onset of the complaint.
The most pertinent conclusion to come out of this work however, was the following:

"Despite the statistically reliable differences between group means, we are more impressed by the overlap of the scores on the various dimensions across the groups than by the group differences. Certainly these data do not support the notion of a "headache personality"; instead it is clear that individuals with headache are similar to most people without headaches" (Blanchard et al., 1984, p. 342).

Three recent studies have reported associations between Type A behaviour and headache severity amongst college samples (Hicks and Campbell, 1983; Martin, Nathan and Milech, 1987; Woods, Morgan, Day, Jefferson and Harris, 1984). The Type A behaviour pattern has been linked to coronary heart disease (Blumenthal, Williams, Kong, Schonberg and Thompson, 1978; Haynes, Feinleib and Kannel, 1980) and has been thought to be characterised by aggressiveness, time urgency and competitiveness (Friedman and Rosenman, 1974). Type A individuals have been observed to demonstrate greater increases in blood pressure and plasma catecholamine levels under stress than Type B's (see Glass and Contrada, 1982 and Price, 1983 for reviews). Since alterations in autonomic nervous system functioning have been implicated in the pathophysiology of headache (Anthony, 1981; Lance, 1982), it is not surprising that researchers have begun to explore the relationship between Type A behaviour and headache. Martin et al (1987) noted a stronger relationship between mood and headache for Type A than Type B individuals. However, interpretation of this finding is complicated by the fact that the Type A measure employed correlated 0.47 with scores on the Beck Depression Inventory. Thus, an alternative interpretation in terms of depression cannot be ruled out.

As with all correlational and non-experimental group comparison studies, statements regarding cause and effect cannot be inferred. Accordingly, it is not yet clear whether Type A behaviour is a cause or consequence of headache or merely a correlate of some other variable which is also related to headache.

In an attempt to determine whether certain personality characteristics are specific to headache sufferers, some research has compared the psychological functioning of these subjects with headache-free control subjects and included groups of subjects reporting other pain or medical conditions. Kearney et al (1987) found a mixed group of tension, migraine and tension-vascular headache sufferers to score in the more pathological direction than tinnitus patients and normal controls on the Neuroticism Scale of the EPI, on the Taylor Manifest Anxiety Scale (Bendig, 1956) and on the Stress Cognitions Inventory developed by the authors. No differences between groups were observed on the Beck Depression Inventory or on the Emotional Control Scale (Watson and Greer, 1983). The authors point out that although
the differences observed on neuroticism and anxiety were significant, there was a considerable overlap between groups on these two measures.

Maxwell (1966) compared migraine sufferers attending physicians with non-headache medical patients, and found the migraineurs to score higher on the Neuroticism Scale of the EPI. On the other hand Blaszczynski (1984) compared non-clinic classical migraine sufferers, non-clinic tension headache sufferers and normal controls with subjects suffering chronic pain problems other than headache. The three pain groups scored higher than the normals on the Neuroticism Scale, but the tension and migraine headache sufferers failed to differ from each other or from the other chronic pain group. Similar results were observed on the Buss-Durkhee Hostility-Guilt Inventory (Buss and Durkhee, 1957), whilst no differences between groups were observed on the Hysteroid-Obsessoid Questionnaire (Caine and Hope, 1967).

The inconsistency of the findings across studies precludes the conclusion that high scores on neuroticism differentiate headache sufferers from other medical patient groups.

Armentrout (1979) observed that although headache and chronic back pain sufferers had lower self-esteem scores than pain free medical patients, on the Tennessee Self-Concept Scale (Fitts, 1965), no significant difference between the pain groups was found. Ajwani and Ajwani (1983) found non-organic headache sufferers to be more depressed and anxious than headache sufferers for whom an organic cause had been identified. However, a recent comparison of pain-free controls with physician-referred migraineurs, tension headache sufferers and sufferers of irritable bowel syndrome (Blanchard et al, 1986), revealed the latter group to be most depressed (Beck Depression Inventory) and Anxious (State-Trait Anxiety Inventory), followed by the tension headache sufferers, migraineurs and normals. A similar ordering was observed on the Pschosomatic Symptom Checklist.

The fact that headache sufferers have not been shown to score consistently in the more disturbed range on measures of psychological functioning than other pain or patient groups, undermines the premise that there exists some constellation of personality traits that could be said to characterise the headache population.

In a rather different approach to the assessment of personality, Grothgar and Scholz (1987) evaluated the expression of anger in migraineurs, persons with chronic pain problems other than headache and pain-free controls. All groups had to solve difficult anagrams, in a competitive situation, whilst being harassed by the experimenter's confederate. The behaviour of the subject was video taped and rated on the basis of anger expression. The migraineurs were judged to be less expressive of anger than the remaining groups. Subjective reports of anger, failed to differentiate the groups. The authors concluded that headache sufferers tend to
inhibit their expression of anger. However, in the report of the study, it is not stated whether or not the confederate and/or the independent raters were blind to the category to which each subject belonged or to the experimental hypothesis. Furthermore, the migraineurs had suffered with headaches for a mean of 21.4 years, whereas, the other pain sufferers had had pain problems for a mean of only 10.4 years. These factors make the obtained findings difficult to interpret.

A number of authors have suggested that many headache sufferers are depressed, and have implicated this dysphoria in the aetiology of headache (e.g., Dalessio, 1968; Diamond, 1983; Weatherhead, 1980). However, several studies have not revealed significant elevations in depression amongst chronic headache sufferers (e.g., Merskey et al, 1985; Philips and Jahanshahi, 1985), whilst Andrasik, Blanchard, Arena, Teders et al (1982) observed headache sufferers to score no higher on the Beck Depression Inventory than headache-free controls. Furthermore, although there is evidence to suggest that anti-depressant medications, such as amitriptyline, are effective in the treatment of tension headache (Lance and Curran, 1964; Sherwin, 1979), this treatment has been demonstrated to relieve headache regardless of the presence or absence of depression (Carasso, Yehuda and Streifler, 1979; Lance and Curran, 1964). Amitriptyline has also been reported to be effective in the treatment of migraine (Couch, Dewey, Ziegler and Hassanein, 1976; Gomersall and Stuart, 1978), the resultant improvement in headache being independent of the existence of any depression (Couch and Hassanein, 1979). Thus, it cannot be concluded that the treatment relieves headache because it addresses the depression thought to underlie the complaint.

The relationship between depression and headache has also been explored by requiring headache sufferers to record their headaches and mood states each day, and then calculating correlations between the two variables (Arena, Blanchard and Andrasik, 1984; Harrigan, Kues, Ricks and Smith, 1984; Martin et al, 1987). Harrigan et al (1984) employed 17 migraineurs and asked them to rate their headache intensity and mood (on ten scales), three times per day. For each individual, correlations between each mood scale and headache intensity were calculated. The authors reported that 26 of these correlations reached significance, but this is hardly impressive for it must be taken into account that in total, some 170 correlations were computed. Arena et al (1984) studied tension headache sufferers, but observed no significant correlations between headache intensity and daily ratings of anxiety, anger or depression. These findings suggest that there may be little association between daily fluctuations in mood and headache intensity. Mood ratings have been found to be more negative (i.e., indicative of higher levels of depression, anxiety, hostility, tiredness, confusion and unsureness) on headache than on headache-free days (Martin et al, 1987), but no causal relationship can be inferred. Even if a causal relationship between the variables were to exist, the direction of causality could not be specified from these data (Martin et al., 1987).
In general, comparisons of the psychological functioning of headache sufferers with other pain and pain-free control subjects, have not indicated that the former group differ in any specific or reliable manner. Since the design of these studies is such that there can be no separation of cause and effect, their informative content is necessarily limited.

### 3.4. Summary

There is very little evidence to suggest that the psychological functioning of headache sufferers is very different from that of headache-free persons (e.g., Andrasik, Blanchard, Arena, Teders et al., 1982; Blanchard and Andrasik, 1984). Nevertheless, it is widely believed that stressful events are the major triggers of headache attacks (e.g., Friedman et al., 1954; Lance and Selby, 1960). In addition to the case histories upon which this claim was originally based, there is now some evidence suggesting that headache sufferers do report a greater frequency of minor, day to day stressful events than headache-free controls (Holm et al., 1986) and that such stressful events tend to precede periods of headache more often than periods of headache freedom (Levor et al., 1986). However, given the ubiquity of these kinds of stressors, it would seem necessary to hypothesise that they interact with some predisposition for headache in precipitating particular attacks. Cognitive processes, mediating the impact of stressful events upon the well-being of the individual (Cox and Mackay, 1978; Lazarus and Folkman, 1984a, b), may be involved in this interaction.

What is lacking in the literature on psychological variables and headache is a conceptual model, detailing linkages between stressful events, psychological factors and headache. Much of the research conducted thus far has been relatively unconcerned with the formulation of such a model and has, hence, tended to progress in a rather haphazard manner, to seek differences between headache sufferers and control subjects on a wide range of psychological measures.

The main objective of this thesis is to conceptualise the relationship between stressful events and headache in terms of cognitive mediational processes, and to subject this model to empirical scrutiny. Toward this end, it was necessary to examine the literature on the psychological treatment of recurrent headache. This review is presented in the next chapter.
CHAPTER 4

PSYCHOLOGICAL APPROACHES TO THE TREATMENT OF HEADACHE.

The psychological treatments applied most commonly to headache in recent years include biofeedback training, relaxation training and cognitive therapy. The effects of these treatments on headache and their mechanisms of action are considered in this chapter. The task is prefaced by a consideration of the assessment of outcome from such treatments.

4.1. The Assessment of Outcome.

Evaluations of the effects of psychological treatments on headache, have most commonly employed a diary approach to assessment. This involves subjects in keeping a log of their headache activity during a pre-treatment baseline, over the course of treatment and throughout a post-treatment assessment phase. Such diaries require subjects to rate the intensity of their headaches several times per day (e.g., Andrasik and Holroyd, 1983; Blanchard, Andrasik, Neff, Arena et al, 1982; Budzynski, Stovya, Adler and Mullaney, 1970, 1973; Epstein and Abel, 1977).

The diary recording methods have varied from hourly recordings, made on a 6 or 10 point intensity scale (e.g., Andrasik and Holroyd, 1980; Budzynski et al., 1970, 1973; Holroyd and Andrasik, 1978, 1982; Holroyd et al., 1984), to ratings of intensity made only four times per day: at breakfast, lunch, dinner and just before sleep (e.g., Attanasio, Andrasik and Blanchard, 1987; Blanchard et al., 1986; Epstein and Abel, 1977). Other researchers (e.g., Anderson, Lawrence and Olson, 1981; Kohlenberg and Cahn, 1981) have employed a more loosely structured approach, simply requiring that subjects note the time of onset and cessation of each headache and assign a global rating to its intensity.

The above methods of assessing treatment outcome stand in contrast with the practice of asking subjects to assign global ratings of headache improvement at the end of treatment, as has been common practice in pharmacological trials (e.g., Sherwin, 1979; Ward, Bloom and Fridel, 1979). This method is employed less often in behavioural treatment studies (e.g., Abramowitz and Bell, 1985; Sargent, Green and Walters, 1973). Andrasik and Holroyd (1980) found global ratings of headache activity to demonstrate a "minimal" relationship to measures derived from diary recordings. Furthermore, the results obtained by Blanchard, Andrasik, Neff,
Jurish and O'Keefe (1981), suggested that global ratings of improvement may overestimate treatment effects relative to diary recordings.

The diary recording method has been found to be moderately correlated with global ratings of outcome from relaxation training made by a "significant other" living with the patient (Blanchard et al, 1981), allowing some validity to be attached to this procedure. Unfortunately, these researchers did not correlate the global ratings made by the patient with those made by the "significant other". Thus, no comparative evaluation of the validity of the global and diary approaches was carried out.

Further examination of the global and diary methods is required before it can be claimed, with any confidence, that the latter is superior. However, as Andrasik and Holroyd (1980) argue, until evidence is presented to the contrary, it is reasonable to suppose that diary recordings are probably more accurate than global ratings, insofar as the former are likely to be less susceptible to the distortions of memory.

From the diary recording methods discussed above, researchers have derived a number of measures of headache activity. The most popular measure has been the headache index, defined as the sum of the intensity ratings multiplied by the total number of headache hours (e.g., Anderson et al., 1981; Blanchard et al, 1983; Blanchard et al., 1986; Holroyd, Andrasik and Westbrook, 1977; Lake, Rainey and Papsdorf, 1979). When headaches are monitored hourly, it is possible to derive separate measures of intensity, duration, frequency and medication consumption. Some researchers have employed these four measures, as well as the headache index, in their assessment of outcome (e.g., Andrasik and Holroyd, 1980; Brown, 1984; Holroyd et al, 1984). However, the use of a combined index has probably done more to obscure than to clarify specific treatment effects for, as Philips (1977) has pointed out, any given treatment may have differential effects on the intensity, frequency and duration of headache. When two or more of these variables are combined into a composite index, any differential effects are lost. There is, therefore, little to justify researchers' reliance upon the headache index as a measure of headache activity. A greater appreciation of treatment effects seems more likely to be gained from the examination of separate dimensions of headache.

Consideration of the various methods of diary headache recording leads one to wonder whether accurate measures of headache duration can be obtained when subjects rate their headache intensity on only four occasions each day (e.g., Andrasik and Blanchard, 1987; Attanasio et al., 1987), for the period between each recording time could often be in excess of 6 hours. This represents a considerable loss of precision in comparison with headache ratings made hourly (Budzynski et al., 1970, 1973; Holroyd et al., 1984) or once every two hours (e.g., Haynes, Griffin, Mooney and Parise, 1975).
It would seem that, in the assessment of treatment outcome, hourly recordings of headache intensity are preferable to both global improvement ratings and to ratings of intensity made only four times per day. There is little to commend the use of a composite index as a measure of headache severity.

The various approaches to the treatment of headache, commonly employed by psychologists, are considered in the following sections.

4.2. Relaxation Training.

A variety of relaxation training methods have been applied to headache, including progressive relaxation (Jacobson, 1938) and its variants (e.g., Bernstein and Borkovec, 1979; Goldfried and Trier, 1974; Wolpe and Lazarus, 1966), autogenic training (Schultz and Luthe, 1969) and, less commonly, meditation (Kabat-Zinn, Lipworth and Burney, 1985). Of these, the various forms of progressive relaxation training have been employed most widely.

For the present purposes, the term progressive relaxation training will be employed to refer to any relaxation training method, where the focus is predominantly upon the control of muscle tension. In their meta-analytic review involving nine studies of relaxation training for tension headache (usually progressive relaxation), Blanchard, Andrasik, Ahles, Teders and O'Keefe (1980) concluded that relaxation training was more effective than medical or psychological placebos and just as effective as frontal EMG biofeedback alone in reducing headache activity. Furthermore, relaxation training alone was found to be just as effective as a combined treatment involving both relaxation training and frontal EMG biofeedback. However, as recognized by Blanchard et al (1980), such an analysis obscures the methodological differences between studies and fails to take account of the perceived plausibility of the placebo-control conditions employed.

Cox, Freundlich and Meyer (1975) found progressive relaxation training to be more effective than medication placebos in reducing medication consumption amongst tension headache sufferers at post-treatment and four month follow-up assessments. Although the effects of progressive relaxation training and placebo, on the headache index, were not significantly different immediately after treatment, the relaxation treatment was found to be superior at follow-up. No differences in outcome were observed when relaxation training was compared with frontal EMG biofeedback treatment incorporating suggestions for relaxation. Other researchers have also reported equivalent outcomes for progressive relaxation training and relaxation plus frontal EMG biofeedback (e.g., Haynes et al, 1975; Martin and Mathews, 1978) and Chesney and Shelton (1976) found progressive relaxation training to be more
effective than EMG biofeedback alone in reducing headache duration. The relaxation and biofeedback treatments were equivalent to each other, but superior to the no treatment control group, in terms of the extent to which they reduced the frequency of tension headache.

Single subject studies (e.g., Eufemia and Wesolowski, 1983) and uncontrolled treatment trials (Philips and Hunter, 1981a; Tasto and Hinkle, 1973; Warner and Lance, 1975) have also provided results favourable to relaxation training. Teders et al (1984) observed a minimal contact home-based relaxation treatment to be as effective as therapist delivered instructions in reducing diary measures of headache frequency, duration and intensity.

Progressive relaxation training has also been employed in the treatment of migraine. Attfield and Peck (1979) observed a significant improvement in headache activity for migraineurs in this treatment, which was superior to the insignificant effects of finger temperature biofeedback. Blanchard, Theobald, Williamson, Silver and Brown (1978) found migraine sufferers assigned to progressive relaxation training to show greater improvements than those assigned to a waiting list control group.

Janssen and Neutgens (1986) compared progressive relaxation training with autogenic training for tension, tension-vascular and migraine headache. On the headache index, progressive relaxation was found to be more effective than autogenic training for tension headache at the post-treatment and three month follow-up assessments. There was no difference between treatments for the migraineurs. For the tension-vascular headache cases, autogenic training was superior to progressive relaxation training at post-treatment, but this difference was not evident at the follow-up evaluation. The superior effect of progressive relaxation training over autogenic training for tension headache suggests that, at least for this sub-group of headache sufferers, progressive relaxation may have therapeutic effects beyond those attributable to non-specific factors.

Given the studies suggesting that progressive relaxation training may be more effective than a medical or psychological placebo (Cox et al, 1975; Richter et al, 1986) and more effective than finger temperature or EMG biofeedback (Attfield and Peck, 1979; Chesney and Shelton, 1976) in the treatment of tension and migraine headache, progressive relaxation training would appear to generate more than a placebo effect in the treatment of recurrent headache. The evidence, however, is insufficient to warrant a similar conclusion for autogenic training or meditation.

Most often, autogenic training has been applied to migraine in conjunction with finger temperature biofeedback. Very few studies have evaluated the effectiveness of autogenic training itself. Anderson et al (1981) administered autogenic training to two tension headache
sufferers and observed reductions on a combined headache index. However, Collet, Juenet and Cottreaux (1986) compared autogenic training with galvanic skin response biofeedback for tension headache and observed a lesser and insignificant effect for the former on diary measures of headache frequency. As mentioned previously, Jenssen and Neutgens (1986) found little evidence to support the effectiveness of autogenic training over progressive relaxation training for tension, tension-vascular or migraine headache sufferers.

Kabat-Zinn et al (1985) taught mindfulness meditation to subjects suffering back pain (N = 31), neck or shoulder pain (N = 15), migraine or tension headache (N = 24). All pain groups demonstrated improvements on a global rating outcome measure, no significant differences between groups being identified. The paucity of studies on the effectiveness of meditation with headache sufferers, however, precludes any statement regarding the efficacy of this treatment.

The processes by which the various forms of relaxation training might lead to headache reduction are as yet unspecified. The original rationale for the application of progressive relaxation training was based on the Ad Hoc Committee's (1962) view that muscular tension in the neck and cranium constituted the source of pain in tension headache (Chesney and Shelton, 1976; Tasto and Hinkle, 1973). Psychophysiological studies, however, have not supported this assumption (e.g., Anderson and Franks, 1981; Andrasik, Blanchard, Arena, Saunders and Barron, 1982; Martin and Mathews, 1978; Pritchard and Wood, 1983). Indeed, Philips and Hunter (1981a) selected tension headache sufferers with low resting tension levels in the frontalis and temporalis muscles but, despite this, progressive relaxation training did result in significant reductions in headache frequency and duration.

The possibility that some biochemical mechanism involving serotonin turnover may mediate treatment effects is suggested by the observation that increases in the serotonin metabolite 5-HIAA (5-hydroxyindole-acetic acid) occur after periods of transcendental meditation (Bujatti and Riederer, 1976). It is also possible that relaxation training engenders some cognitive change in the subject, leading to an enhanced sense of control over the disorder (Turner and Chapman, 1982).

4.3. Biofeedback Treatment.

Biofeedback is a procedure designed to enable the individual to gain greater voluntary control over his or her physiological functioning. The placement of electrodes at the appropriate site and their connection to an amplifying system, allows the signal from the physiological process in question to be fed back to the individual as a variable light, tone or
meter display. The intensity of the feedback signal is directly proportional to the physiological activity. Accordingly, through regulation of the signal, the subject may learn control over a particular physiological process (e.g., Schwartz, 1973).

Following the traditional view of headache as falling within at least two distinct categories (migraine and tension), early researchers (e.g., Budzynski et al. 1970, 1973; Sargent et al., 1973) reasoned that by teaching the subject voluntary control over the specific mechanisms thought to underlie headache, the condition could be eliminated or at least improved. Thus, biofeedback treatment of migraine has focused on the control of blood flow through the cranial arteries via finger temperature (e.g., Daly et al., 1983; Lacroix et al, 1983) or temporal arterial pulsation (e.g., Feurstein and Adams, 1977; Gauthier, Lacroix, Cote, Doyon and Drolet, 1985) feedback. For tension headache, the emphasis has been on the control over the neck or cranial musculature, through EMG biofeedback (e.g., Hudzynski, 1983; Hart and Chichanski, 1981). The biofeedback treatment is often supplemented with instructions to practise some variant on Jacobson's (1938) progressive relaxation procedure (e.g., Bernstein and Borkovec, 1979) for tension headache, or Schultz and Luthe's (1969) autogenic training for migraine.

4.3.1. Finger Temperature Biofeedback (FTB).

In FTB (sometimes referred to as digital temperature feedback) the skin temperature is taken to reflect the amount of blood flowing through the area. The subject is taught to increase the peripheral blood supply by increasing the temperature of his or her finger (e.g., Blanchard et al, 1978).

FTB has been considered an effective treatment for the following reasons:

1. Increased peripheral blood flow is associated with relaxation (Schultz and Luthe, 1969).

2. It has been assumed that learning to re-direct blood flow to peripheral areas would necessarily reduce blood volume in extracranial vessels, thereby reducing the pressure thought to be responsible for the pain of migraine headache (Sargent et al., 1973).

Sargent et al (1973) treated 28 migraine sufferers with a combination of FTB and autogenic training (Schultz and Luthe, 1969). Independent global ratings of improvement ranged from 68% to 80%. Unfortunately, this study was methodologically weak. The
definition of improvement was not specified and no control or placebo-control groups were included. Furthermore, the research group did not attempt to assess changes in finger temperature and correlate these with improvements in headache severity.

Daly et al (1983) assigned migraine and tension headache sufferers to progressive relaxation training, FTB or EMG biofeedback. Each treatment was supplemented with autogenic relaxation training. Outcome was assessed by means of the headache index, headache intensity and hours of headache per month, as derived from diary recordings. All groups demonstrated significant improvements on all outcome measures assessed at three month follow-up (no immediate post-treatment assessment was conducted). There was no effect of headache type on outcome. Progressive relaxation training was observed to have a lesser effect on headache hours per month than either of the biofeedback treatments. The treatment effects for all groups were equivalent on the remaining outcome measures.

However, as with the study conducted by Sargent et al (1973), it is difficult to attribute the superior effects of the biofeedback treatments to the feedback components as these were combined with autogenic relaxation training which has, itself, been reported to be effective in the treatment of headache (Janssen and Neutgens, 1986; Schultz and Luthe, 1969). The most notable feature of the results reported by Daly et al (1983) is the finding that all treatment effects were unrelated to headache type. This does not augur well for the view that EMG biofeedback and FTB exert their therapeutic effects through specific actions on the pathophysiological processes thought to underlie the disorder. It would seem more likely that a relaxation response (Benson, 1975) may be the important therapeutic ingredient (Holmes and Burish, 1983). Indeed, Attfield and Peck (1979) found progressive relaxation training to be more effective in the treatment of migraine than FTB alone. Similar results were reported by Blanchard et al (1978), except that the superior effect of progressive relaxation training over FTB plus autogenic training was not apparent at the three month follow-up. During the last week of treatment, however, the relaxation group showed greater reductions in headache duration, intensity and medication consumption than the FTB group.

Barrios and Karoly (1983) observed migraineurs to rate their improvement following FTB alone as no greater than that accruing from progressive relaxation training, pharmacotherapy, insight psychotherapy or social skills training. These researchers considered that some common mechanism such as lowered arousal, self-monitoring or the learning of new coping strategies might have been responsible for the treatment gains. Similarly FTB has been found to be no more effective for migraine than temporal artery, frontal EMG or electroencephalographic (EEG) alpha biofeedback (Cohen, McArthur and Rickles, 1980). Gauthier et al (1985) found FTB and temporal artery biofeedback for migraine to be equivalent
to each other, but superior to a waiting list control condition. Diary measures of medication consumption and headache frequency, intensity and duration served as outcome measures.

Studies of FTB alone compared with control and / or placebo- control conditions, have called into question the efficacy of this treatment. The effects of FTB on migraine have been found to be no greater than those occurring for a headache recording control group (Kewman and Roberts, 1980; Mullinex, Norton, Hack and Fishman, 1978). Furthermore, these same researchers found that, when subjects were assigned to finger warming or finger cooling treatment conditions, this made no difference to the outcome.

Jessup, Neufeld and Merskey (1979) described a study where migraineurs were assigned to autogenic training hand warming, FTB hand warming, FTB hand cooling, unassisted hand cooling or to a laboratory visit attention / placebo condition. At post-treatment, all five groups demonstrated significant but equivalent improvements relative to baseline. These gains were maintained by all groups at a six month follow-up. Increases in finger temperature occurred in all groups, regardless of the treatment received.

Reading (1984) found frontalis EMG, finger temperature, galvanic skin response or false EMG biofeedback conditions to be equally effective in the treatment of migraine. The subjects were exposed to cognitive stressors (arithmetic and word recognition tasks) before and after treatment. At post-treatment, the effects of stressors on frontalis EMG, skin conductance level, finger temperature, heart rate and respiration rate were no different from these effects at pre-treatment. This indicated that, on these measures, the subjects' physiological reactivity was not modified by any of the treatments. Lake et al (1979) observed no effect for FTB alone, or in combination with rational-emotive therapy, over a waiting list control condition in the treatment of migraine.

In summary, it would appear that FTB in isolation is no more effective in the treatment of migraine than a credible placebo, and certainly no more effective than some form of relaxation practice.

Damaging to the rationale underlying FTB is the observation that relief from migraine following this treatment, or autogenic training, does not correlate with learned control over peripheral blood flow (Gauthier et al, 1985; Mullinex et al 1978; Werbach and Sandweiss, 1978).

Blanchard et al (1983) treated migraine and tension-vascular headache sufferers with FTB and observed a significant correlation (r = 0.3) between the number of sessions in which any increase in finger temperature occurred and improvements on the headache index. Despite
its being significant, the correlation pertains to but one of six measures of finger temperature control. The five remaining measures failed to correlate significantly with the headache index. Moreover, the significant correlation observed by Blanchard et al (1983), was not replicated by Gauthier et al (1985), despite the fact that the latter research group employed a very similar measure of finger temperature control.

Lacroix et al (1983) administered an unspecified relaxation treatment, frontal EMG biofeedback or FTB to 23 migraine sufferers. In the biofeedback conditions, subjects were instructed to practise at home the response they achieved in the laboratory. These researchers claimed that FTB was the most effective treatment, and that there existed a relationship between finger temperature and the frequency of migraine attacks. However, examination of their results reveals that such bold conclusions are not supported by the data. First, the group x time interaction on global improvement ratings made at post treatment, eight week and six month follow-up fell short of significance (F (4,48) = 2.41; p > 0.05). A similar result was obtained for questionnaire responses concerning headache frequency (F (4,47) = 2.26; p > 0.05). Secondly, although all three groups demonstrated significant increases in finger temperature over the course of treatment, this effect was no greater for the FTB group. Thirdly, when the data from all groups were pooled and correlations between global improvement ratings and various measures of finger temperature control calculated, none of these reached significance.

Thus, the results obtained by Lacroix et al (1983) are consistent with those reported by Cohen et al (1980). The latter research group observed migraineurs to be no more responsive to FTB than to temporal artery, EMG or alpha biofeedback. The EMG biofeedback group demonstrated a reduction in finger temperature over treatment (despite this, their headaches improved), whilst the FTB group did not differ from the temporal artery or alpha biofeedback groups on this measure. The capacity of the subjects in the FTB group to increase finger temperature was unrelated to headache improvement. Similarly, in each of the remaining treatment conditions, learned control over the targeted physiological process failed to predict the extent of therapeutic gains.

It has been found that teaching subjects to increase or decrease finger temperature makes no difference to cerebral blood flow (Largen, Mathew, Dobbins, Meyer and Claghorn, 1978) and that increasing finger temperature is associated with increased, rather than decreased, temporal artery blood flow (Price and Tursky, 1976). Such findings contradict the mechanism assumed to underlie the therapeutic effects of FTB.
Given the unsympathetic outcome research and the lack of support for the assumptions upon which FTB is predicated, it must be concluded that there is very little justification for the continued application of the technique.

4.3.2. Temporal Artery Biofeedback (TAB).

As with finger temperature biofeedback, the rationale for TAB (sometimes referred to as cephalic biofeedback) rests on the notion that dilatation of the temporal arteries constitutes the source of pain in migraine headache (Ad Hoc Committee, 1962; Tunis and Wolf, 1953). Subjects receive feedback concerning the vasomotor functioning of the extra cranial arteries and attempt to learn a vasoconstrictive response (e.g., Bild and Adams, 1980; Friar and Beaty, 1976). Vasoconstriction is thought to alleviate the pain by countering the vasodilative phase of migraine attacks. Accordingly, the technique has been applied almost exclusively to the treatment of migraine. Given that the source of feedback in TAB is closer to the presumed source of pain than that of finger temperature training, it is somewhat surprising to find that far fewer studies have been published on the efficacy of TAB.

In the first published investigation into the effects of TAB employing a group treatment outcome design (Friar and Beaty, 1976), subjects in the experimental condition received feedback from the temporal artery on the side of the head on which pain occurred most often, whilst those in the control condition received feedback for pulse amplitude in the hand (the authors did not specify from which hand feedback was received). A comparison of temporal artery pulse amplitudes across conditions revealed no significant difference between groups. Furthermore, both groups of subjects failed to demonstrate any significant reduction in headache activity relative to baseline. As the authors pointed out, the limited reduction in headache activity that did occur was probably explicable in terms of placebo effects.

Feurstein and Adams (1977) administered TAB followed by EMG biofeedback to one migraine and one tension headache sufferer, and the reverse order of treatments to a second migraine and tension headache sufferer. These researchers reasoned that the migraineurs would respond only to TAB and that the tension headache subjects would respond only to the EMG biofeedback phase of treatment. One tension and one migraine headache sufferer responded as expected, but the second migraineur improved more in frontalis EMG biofeedback treatment than in TAB treatment, and the second tension headache sufferer responded better to TAB than to frontalis EMG biofeedback. Accordingly, the results do not support the premise that these two treatments exert their therapeutic effects by addressing separate underlying pathophysiologies.
Bild and Adams (1980) assigned migraine sufferers to two treatment conditions, one where they received TAB from the temporal artery on the habitually affected side of the head, or to another where they received EMG biofeedback from the frontalis muscle, or to a waiting list control condition. Although a number of measures were derived from subjects' diary recordings of headache activity, it was only on the measure of headache frequency that the TAB group demonstrated a greater improvement than the remaining groups. At the three month follow-up, headache frequency for the TAB group was no different from that observed at the post-treatment assessment. However, since there was no follow-up assessment for the remaining groups, it was impossible to evaluate the relative efficacy of TAB at this stage of the study.

The observations of Bild and Adams (1980) were not replicated by Cohen et al (1980), who found TAB, EMG biofeedback, alpha biofeedback or finger temperature training to exert equivalent effects on diary measures of headache frequency, intensity and disability ratings. Furthermore, although there was some evidence to suggest that subjects in the TAB condition were able to demonstrate greater vasoconstriction towards the end of treatment, the extent of these physiological responses was unrelated to headache improvement. A similar absence of any significant correlation between control over temporal artery blood volume and headache improvement following TAB was reported by Gauthier et al (1985). These researchers observed TAB to be no more effective for migraine than FTB. When the data from these two treatments were combined, greater reductions in headache intensity duration and medication consumption were observed relative to a waiting list control group.

It would appear that there is no consistent evidence to support either the efficacy of TAB or its rationale. When the technique has been evaluated against frontal EMG biofeedback (Cohen et al, 1980) or against feedback from the hand (Friar and Beaty, 1976), each of which have been employed by researchers as placebo-control conditions (Bild and Adams, 1980; Friar and Beaty, 1976), no clear cut demonstration of the superior efficacy of TAB has been forthcoming. It seems unlikely that TAB has anything more than a placebo effect on migraine headache. The rationale underlying the technique is questioned by the observation that alterations in vasomotor functioning, following treatment, are unrelated to improvements in headache (Cohen et al., 1980; Gauthier et al., 1985). Finally, the fact that migraine can occur in the absence of detectable changes in extracranial blood flow (e.g., Drummond, 1984) also questions the utility of this procedure.
4.3.3. Electromyographic Biofeedback.

EMG biofeedback treatment is directed towards teaching subjects to reduce the level of tension in the cranial musculature (usually the frontalis muscle). It has thus been applied to the treatment of tension headache, following the view that this disorder stems from sustained contraction of the skeletal muscles in the region of the head and neck (Ad Hoc Committee, 1962; Dalessio, 1980; Diamond and Dalessio, 1978). The technique has been applied to migraine only as a placebo control, not as an active treatment (e.g., Cohen et al., 1980; Reading, 1984). As was the case with finger temperature feedback, appraisal of the effectiveness of EMG biofeedback is complicated by some researchers' tendency to combine this treatment with instructions to engage in regular relaxation practice (usually progressive relaxation) (e.g., Bell, Abromowitz, Folkins, Spensley and Hutchinson, 1983; Budzynski et al., 1970, 1973; Hudzynski, 1983).

In the first trial of EMG biofeedback from the frontalis muscle, five subjects received this treatment and were asked to practise at home the same relaxation they learned in the laboratory (Budzynski et al., 1970). Improvements in group ratings of headache intensity were reported to occur over the course of treatment. In a subsequent study, these researchers (Budzynski et al., 1973) assigned tension headache sufferers to EMG biofeedback plus home relaxation practise, false feedback or to a waiting list control group. The active treatment group demonstrated lower frontalis muscle tension levels in the last two weeks of the eight week treatment than subjects receiving false feedback. This finding persisted at the three month follow-up. Only subjects in the active treatment demonstrated significant reductions on the headache index. After treatment, headache index scores were lowest for the active treatment. At follow-up, the genuine feedback group was said to have a lower level of headache activity than the false feedback group, but no inferential statistic was given. A notable feature of the results is the finding that, over the course of the active treatment, a very high correlation \( (r = 0.9) \) emerged between weekly frontal EMG levels and the size of the weekly headache index derived from diary recordings. In the false feedback condition, no significant correlation between these variables was observed. However, many subsequent studies have failed to confirm the association between reductions in headache activity and diminishations in cranial muscle tension levels over the course of EMG biofeedback (e.g., Abramowitz and Bell, 1985; Andrasik and Holroyd, 1980; Cram, 1980; Epstein and Able, 1977; Holroyd, et al., 1977; Martin and Mathews, 1978; Philips and Hunter, 1981a). Martin and Mathews (1978) also observed no significant correlation between reductions in neck muscle tension and headache activity over a course of EMG biofeedback. Andrasik and Holroyd (1980) found that improvements in tension headache occurred irrespective of whether subjects were trained to increase, decrease or hold constant their frontalis muscle tension levels during EMG biofeedback treatment. These results persisted at a three month follow-up assessment.
(Andrasik and Holroyd, 1980). Cram (1980) observed that tension headache subjects, taught to hold their frontalis muscle tension levels constant, were more improved on the headache index at a six month follow-up assessment than those trained to reduce frontalis tension levels. Thus, it would appear that reductions in cranial EMG levels may not represent the process by which EMG biofeedback occasions any improvement in tension headache.

On the question of the efficacy of EMG biofeedback, in the treatment of tension headache, it is important to note that, although some studies report EMG biofeedback alone to be more effective than medical placebos (Cox et al., 1975), attention placebos (Cram, 1980) or pseudo biofeedback (Budzynski et al., 1973; Kondo and Canter, 1977), Holroyd, Andrasik and Noble (1980) observed frontal EMG biofeedback to be no more effective than bogus medication. Furthermore, frontal EMG biofeedback has been reported to be no more effective than no-treatment control conditions (e.g., Chesney and Shelton, 1976; Holroyd et al., 1977; Janssen, 1983). These inconsistent findings cast doubt on the efficacy of EMG biofeedback training and raise the question of whether this treatment is any more effective than a much simpler technique such as progressive relaxation training.

Cox et al (1975) combined EMG biofeedback with regular home relaxation practice and compared this treatment with progressive relaxation training in the treatment of tension headache. On the basis of headache index scores, the combined treatment proved to be more effective at the post-treatment assessment. By the six month follow-up assessment, however, the two treatment groups were equivalent in terms of outcome, and superior to a medication placebo control group.

Comparisons of frontalis EMG biofeedback alone with progressive relaxation training have also resulted in equivalent outcomes (e.g., Haynes et al., 1975; Martin and Mathews, 1978), whilst Chesney and Shelton (1976) found progressive relaxation training to be more effective than frontal EMG biofeedback alone in reducing the duration of tension headache. Equivalent outcomes were observed on other measures of headache activity.

There is one study claiming frontalis EMG biofeedback alone to produce greater reductions on the headache index than progressive relaxation training (Hutchings and Reinking, 1976). However, it was subsequently reported that at six and twelve month follow-up assessments, no difference between the treatment groups was evident (Reinking and Hutchings, 1976). Furthermore, these researchers employed taped, rather than "live", relaxation training which may have attenuated the effects of this treatment (Borkovec and Sides, 1979).
Additional studies seeking to demonstrate the value of EMG biofeedback have investigated the hypothesis that tension headache sufferers, who achieve a minimal response to relaxation training, may derive benefit from a course of frontalis EMG biofeedback training, (e.g., Balanchard, Andrasik, Neff, Arena et al., 1982; Blanchard, Andrasik, Neff, Teders et al., 1982). In these studies, all subjects were started on a course of relaxation training, after which those achieving a reduction in headache activity of more than 60% were assigned to follow-up, whilst those failing to achieve such a reduction received EMG biofeedback. The relaxation training "non-responders" were found to have improved significantly after biofeedback training. This was interpreted as evidence for the additive therapeutic effect of biofeedback, at least for a subgroup of tension headache sufferers (Blanchard, Andrasik, Neff, Arena et al., 1982; Blanchard, Andrasik, Neff, Teders et al., 1982). However, since the relaxation non-responders received biofeedback immediately after their relaxation course, it is possible that the additive treatment effect reported was constituted, at least in part, by the continuing benefits of the relaxation training. Accordingly, it cannot be concluded that biofeedback contributes to outcome when relaxation training is only minimally effective, as these studies provided no control for any lagged effects of the latter treatment.

The body of evidence cited above, suggests that frontalis EMG biofeedback may be no more effective than relaxation training in the control of tension headache. As has been noted in a number of reviews of the literature (e.g., Blanchard and Andrasik, 1982; Holmes and Burish, 1983), this conclusion would seem to hold regardless of whether or not the subject is classified as a migraine or tension headache sufferer. No significant differential outcome for migraine and tension headache sufferers has been observed for frontalis EMG biofeedback procedures (e.g., Cohen et al., 1980; Daly et al., 1983) or for progressive relaxation training (e.g., Blanchard et al., 1978; Blanchard, Andrasik, Neff, Arena et al., 1982; Daly et al., 1983). Furthermore, unless EMG biofeedback is supplemented with instructions to practise some sort of relaxation technique at home, be it progressive relaxation (e.g., Chesney and Shelton, 1976) or some idiosyncratic method acquired in the laboratory (e.g., Cox et al., 1975), it is probably no more therapeutic than a placebo.

The mechanisms through which progressive relaxation training and biofeedback alleviate headache remain unspecified. Although the regular attainment of a relaxation response (Benson, 1975) may be an important ingredient, as suggested by the observation that improvement is correlated with frequency of home relaxation practice (Blanchard et al., 1983), the evidence clearly does not warrant the conclusion that the various biofeedback procedures produce therapeutic gains by specifically altering the pathophysiological mechanisms presumed to underlie the tension and migraine headache conditions.

The development of cognitive therapies, where the focus of the intervention is on the thoughts assumed to mediate an individual's emotional response to a given situation, owes much to the work of Albert Ellis (1962, 1977a, b). Ellis' rational-emotive therapy seeks to identify irrational beliefs, persuade the individual to give up this style of thinking and replace it with more rational philosophies and self-statements. Ellis (1962) identified ten irrational beliefs which he regarded as being causally related to emotional distress. He considered beliefs such as "I must be thoroughly competent in everything I do" or "It is a dire necessity that I be liked or loved by every one" as irrational. According to Ellis, persons who hold these beliefs are vulnerable to emotional disturbance in the face of life events. The theoretical status of the concept of an irrational belief will be considered in detail in Chapter 6.

Similar interventions focusing on cognitions have been developed by Beck and his associates (Beck, 1976; Beck et al., 1979) in relation to depression. Here the emphasis is on challenging clients' negative views of themselves and the future.

Meichenbaum (1975, 1977) developed self-instructional training which, like Ellis' rational-emotive therapy, focuses on self-statements. However, in Meichenbaum's (1975, 1977) treatment, the emphasis is upon teaching the client specific cognitive coping skills rather than on challenging irrational assumptions. This treatment has been applied successfully to speech anxiety (Fremouw and Zitter, 1978), anger control (Novaco, 1976), test-anxiety (Meichenbaum, 1972) and weight control (Dunkel and Giaros, 1978).

The application of cognitive therapy to headache was first referred to as "cognitive coping skills training" (Holroyd et al., 1977). These researchers listed the following components:

1. An educational phase, where the pathophysiology underlying the condition is explained and the rationale for treatment is presented with an emphasis upon the cognitive mediation of stress reactions.

2. A self-monitoring phase, where the client is taught to record situations, thoughts, feelings and behaviours associated with headache attacks and stressful transactions.

3. A skills training phase which may include self-control relaxation (Goldfried and Trier, 1974), causal re-attribution (Beck et al., 1979), rational re-structuring (Goldfried, Decenteceo and Weinberg, 1974), calming imagery (Holroyd et al., 1977) or problem-solving (Goldfried and Davison, 1976).
Needless to say, the various studies employing cognitive interventions for headache have not all followed the regimen laid down in "cognitive coping skills training" (Holroyd et al., 1977; Holroyd and Andrasik, 1982b). The term "cognitive coping skills training" is somewhat ambiguous, for it may encompass a broad range of both cognitive and behavioural techniques. Accordingly, the term "cognitive therapy" is preferred. This term will be applied to any treatment study where the intervention was concerned with the restructuring of cognition. Where additional procedures were employed, these will be specified.

The first study to evaluate cognitive therapy for headache compared this treatment with frontalis EMG biofeedback (with instructions for between sessions practise of the acquired technique) and evaluated both against a headache monitoring control group (Holroyd et al., 1977). Tension headache sufferers served as subjects. Subjects' ratings of treatment credibility were obtained at the end of the first treatment session, at post-treatment and follow-up. Subjects in the cognitive treatment demonstrated greater improvements on the headache index than those in the biofeedback group who were no more improved than the headache monitoring controls. These results persisted at the 15 week follow-up assessment. A two year follow-up of the two treatment groups revealed that only the cognitive therapy group evidenced a significant reduction in headache activity (frequency, duration and headache index) relative to the pre-treatment baseline (Holroyd and Andrasik, 1982b). The cognitive group was also more improved than the biofeedback group on headache duration, with trends in this direction being evident on headache frequency and the headache index.

In their initial study (Holroyd et al., 1977), all subjects were treated individually. In a second study (Holroyd and Andrasik, 1978), cognitive therapy was conducted in groups. Tension headache sufferers were assigned to a headache recording control condition or to one of three treatment conditions. Common to all treatments was the requirement that subjects monitor situations, cognitions and feelings associated with headache and stress. In addition, these subjects received cognitive therapy, cognitive therapy plus self-control relaxation training (Goldfried and Trier, 1974) or took part in a headache discussion group emphasising the "historical roots" of their symptoms, rather than the acquisition of specific coping skills. All treatments were conducted over five weekly sessions, each of 1 3/4 hours duration.

At the post-treatment and six week follow-up assessments, and on all measures of headache activity (headache index, frequency, duration and intensity), all three treatment groups were found to be significantly more improved than those simply recording their headaches, with no differences between treatments being evident. Holroyd and Andrasik (1978) observed that those subjects in the headache discussion group, who demonstrated improvements in headache, reported developing their own cognitive strategies for coping with stress and headaches. The authors concluded that self-control relaxation training does not
contribute to the effectiveness of cognitive therapy and that the essential therapeutic ingredient in cognitive therapy may be the simple self-monitoring of cognitive and somatic reactions to stress and headache.

Efforts to assess the efficacy of cognitive therapy for tension headache by means of multiple baseline single subject designs have produced mixed results. One study found reductions in headache to be more dependent on the introduction of cognitive therapy than on the introduction of frontalis EMG biofeedback plus suggestions to practise relaxation at home (Kremsdorf, Kochanowicz and Costell, 1981). A second study found little evidence of any differential therapeutic effects of autogenic training, cognitive therapy or various combinations thereof (Anderson et al., 1981). All therapeutic procedures were equally effective in reducing headache index scores to near zero levels.

Cognitive therapy has also been applied to migraine. Knapp (1982) compared cognitive therapy, temporal artery biofeedback and sequential combinations of these two treatments with a headache recording control condition. Although equivalent significant reductions in headache frequency and duration occurred for all treatment groups over the course of therapy, these improvements failed to differ from the non-significant changes occurring in the waiting list control group. At the two month and twelve month follow-up assessments, all treatment groups demonstrated significant improvements relative to baseline on diary measures of headache frequency, duration, intensity and medication consumption. The controls were not followed up. The failure of the treatment groups to differ from the controls over the course of treatment is difficult to interpret in view of the very small number of subjects in each condition (n = 4).

Brown (1984) demonstrated that teaching migraineurs to employ imagery coping strategies under stress, as well as immediately before and during headache, was effective in reducing headache activity (total intensity ratings averaged over days) relative to an elaborate placebo condition emphasising the need to unlearn "...subconscious stressful thoughts...(through)...subliminal deconditioning ..." (Brown, 1984, p. 60). These improvements remained evident at the eight week follow-up. Brown (1984) speculated that the imagery treatment may have been effective because it aided relaxation, interrupted catastrophising ideation, which experimental pain studies have shown to be associated with a lowered tolerance for pain (e.g., Spanos, Brown, Jones and Homer, 1981; see Appendix A), and / or because the treatment increased subjects' sense of self-efficacy with respect to headache management, thus leading to more effective coping.

Additional studies of cognitive therapy for headache have often mixed cognitive elements with behavioural techniques such as relaxation training or biofeedback, making it
difficult to make any statement about the specific effects of the cognitive procedures themselves.

Bakal, Demjen and Kaganov (1981) combined cognitive therapy, relaxation training and frontal EMG biofeedback into a single package and applied this to the treatment of migraine, tension and tension-vascular headache sufferers. At the post-treatment and six month follow-up assessments significant reductions were observed for all headache groups on diary recordings of headache hours, headache index and medication consumption. There was no significant relationship between headache type and outcome.

Steger and Harper (1980) compared cognitive therapy plus frontal EMG biofeedback with audiotaped relaxation training comprising, "...autogenic phrases, breathing exercises and various relaxation related instructions" (Steger and Harper, 1980, p. 138). Subjects in the relaxation group were given audio tapes and then had no further contact with the therapist for six weeks. In contrast, the subjects in the cognitive therapy plus biofeedback treatment had eight sessions with the therapist. Only subjects in the combined treatment demonstrated significant improvements on diary measures of headache frequency and intensity. After treatment, there was no difference between the groups on headache frequency. Subjects in the combined treatment, however, reported lower levels of headache intensity than those in the relaxation group. The minimal response of the relaxation group may have been due to their lesser contact with the therapist, rather than to any difference in treatment effects.

Figueroa (1982) compared group cognitive therapy plus progressive relaxation training with "traditional psychotherapy" (group discussion of events associated with headache) and evaluated each of these treatments against a headache monitoring control group. Diary measures of headache activity revealed no differences between the psychotherapy and control groups. Reductions in headache frequency and duration were greater for the cognitive therapy plus relaxation group than for the remaining groups. A significant reduction in headache intensity occurred only for the combined treatment, but this was not significantly greater than that observed for the psychotherapy group. Both treatment groups demonstrated greater reductions in headache intensity than the headache recording control group.

Two studies have employed cognitive therapy in the home based treatment of headache. Kohlenberg and Cahn (1981) found that a written self-help programme, comprising a liquid crystal device for finger temperature feedback, plus instructions for relaxation training and cognitive therapy was significantly more effective in reducing the frequency, intensity and duration of migraine headache than a book on the diagnosis and management of the disorder. Explanation of these results in terms of differing expectations of outcome across the groups seems unlikely, as both groups gave equivalent confidence ratings for each treatment both
before and after reading the material. Attanasio et al (1987) assigned tension headache sufferers to a combined office-based treatment, comprising cognitive therapy and progressive relaxation, training or a home based version of this treatment consisting of written materials and involving 50% less therapist contact, or to a second self-help treatment involving progressive relaxation training only. All groups demonstrated equivalent and significant reductions (relative to baseline) on all measures of headache activity (headache index, peak intensity, frequency and medication consumption). The important result in this study is the finding that relaxation training plus cognitive therapy was no more effective than relaxation training alone.

Mitchell and White (1977) evaluated the various components of a cognitive-behavioural treatment programme for migraine by means of a dismantling design. Twelve subjects commenced diary recordings of headache. Three subjects received only this phase of the package, whilst the remaining nine also engaged in self-monitoring of stressful events. Twelve weeks later, six of these subjects progressed on to relaxation training. After another twelve weeks, three of the six subjects who received relaxation training received cognitive therapy. After a further twelve weeks (at week 48), there had been no change in headache frequency for the two self-monitoring groups. The two treatment groups were significantly more improved than the self-monitoring groups, with the relaxation training plus cognitive therapy group being more improved than the group receiving relaxation training but not cognitive therapy. These results were also evident 12 weeks later at the week 60 assessment. The findings suggest that cognitive therapy may be an effective adjunct to relaxation training in the treatment of migraine. The increased effectiveness of the cognitive intervention is unlikely to have been a function of the subjects greater exposure to the therapist, as only one additional group session with the therapist was involved, the remainder of the cognitive treatment being self-administered through audio tapes. However, it is possible that the larger number of techniques to which these subjects were exposed served to increase the strength of placebo effects. The findings are also limited by the fact that at the end of the study, there were only three subjects in each condition.

Newton and Barbaree (1987) found a cognitive intervention, focused principally upon pain control, plus some unspecified form of relaxation training, to be more effective in reducing headache frequency and intensity, amongst a group of tension and migraine headache sufferers, than a headache monitoring control condition. Treatment effects were independent of headache type.

It is unfortunate that so many studies of cognitive therapy for headache have combined this treatment with other interventions such as biofeedback or relaxation training, thereby precluding any statements about the effectiveness of cognitive therapy per se. Very few studies
have examined the effects of cognitive therapy in isolation. Two studies have found cognitive therapy to be more effective for tension headache than a headache monitoring control condition (Holroyd and Andrasik, 1978; Holroyd et al 1977). Knapp (1982) observed no effect for cognitive therapy on migraine headache over a waiting list control condition, although the group sizes (n = 4) in this study may have obscured any treatment effects. Brown (1984) found cognitive imagery to be a more effective treatment for migraine than placebo. Holroyd et al (1977) observed cognitive therapy to be more effective than frontalis EMG biofeedback in the treatment of tension headache.

The only direct comparison of cognitive therapy with relaxation training is afforded by a study of paediatric migraine (Richter et al., 1986). These researchers observed both treatments to be more effective than placebo (a condition in which subjects were taught to label their emotions, to relate them to situations and were provided with "sham coping skills") in reducing headache index scores and headache frequency. However, there were no differences between treatments at the post-treatment or 16 week follow-up assessments. The subjects in this study were aged 9-18 years. It is difficult to appreciate how the group cognitive treatment could have been presented in such a way that it would have appealed to both 9 year olds and 18 year olds. The variability in the age of the subjects may have diluted the effects of cognitive therapy. Relaxation training, being a much simpler procedure, may have been less susceptible to this influence.

Given that Brown (1984) found a cognitive intervention alone to be superior to an elaborate placebo procedure in the management of migraine and, given also that Holroyd and his colleagues (Holroyd and Andrasik , 1982b; Holroyd et al 1977) found cognitive therapy alone to be superior to EMG biofeedback in the treatment of tension headache over a period of up to two years after treatment, it would seem reasonable to conclude that cognitive therapy may be more effective than placebo. More comparisons of cognitive therapy alone, with simpler procedures such as relaxation training, are required in order to establish whether or not the former is of any additional benefit. Headache type (migraine or tension) appears to have no bearing on outcome from cognitive interventions (Bakal et al., 1981; Newton and Barbaree, 1987), although the level of confidence that one can have in these findings is tempered by the fact that, in these studies, the cognitive procedures were combined with other behavioural treatments.

Although many studies have been concerned with evaluating the effects of therapeutic regimens, involving cognitive procedures, on headache activity, very few studies have explored the question of what kinds of cognitive changes might underlie these treatment effects. Lake et al (1979) administered the Irrational Beliefs Test (Jones, 1968) to migraineurs before and after a treatment involving finger temperature biofeedback and three sessions of
rational-emotive therapy. The treatment had no effect on Irrational Belief Test scores, possibly because of the very small amount of rational-emotive therapy delivered to subjects (three 40 minute sessions). Unfortunately, Lake et al (1979) did not attempt to correlate changes in Irrational Belief Test scores with changes in headache activity. The rational-emotive plus finger temperature biofeedback treatment was no more effective in reducing headache activity than a waiting list control condition.

Over the course of their cognitive therapy plus relaxation training programme for tension and migraine headache sufferers, Newton and Barbaree (1987) attempted to assess subjects' thought processes by asking them to telephone an answering machine and to thus record their thoughts during headaches. Subjects receiving treatment, as well as those in the headache monitoring control group, engaged in these telephone recordings for three weeks before and after treatment. Each thought report was rated by independent raters on the following scales:

1. Headache appraisal (3-point scale: negative, neutral, positive).

2. Affective response (5-point scale)

3. Coping rated on the following subscales:
   a) Problem-focused: Thoughts about how to manage the pain.
   b) Cognitive avoidance: Wishing the pain would go away.
   c) Unclassifiable thoughts.

4. Sensory focus: Thoughts about the sensory qualities of the pain.

At the end of the study, the treated subjects reported more positive appraisals of headache, more problem focused thinking and greater reductions in headache frequency and intensity than those subjects assigned to the headache monitoring control condition.

In the pre-treatment data, ratings of headache intensity were associated with more negative appraisal of headache, more cognitive avoidance and greater negative affect. Multiple regression analysis revealed that only the negative affect measure made a significant independent contribution to the prediction of headache intensity. Over the course of treatment, positive shifts in appraisal were significantly correlated with reductions in headache intensity. The authors concluded that thoughts about headache are related to its intensity and that a
positive shift in headache appraisal might represent one mechanism by which cognitive therapy reduces headache intensity.

One of the problems with this study is that cognitive therapy was combined with relaxation training, making it difficult to determine whether the relationships observed were a function of one type of treatment or the other. Furthermore, it was made abundantly clear to subjects in the treatment group that the intervention was concerned primarily with their experience of headache pain. This would have been clear from the use of such treatment strategies as attention diversion, transformation of pain and reappraisal of headache. Thus, the observed relationship between shifts in headache appraisal and changes in headache intensity may have represented simply the simultaneous effect of the demand characteristics of the therapeutic situation on these two variables. It is also difficult to appreciate how appraisals of headache as "positive" or "negative", can be considered conceptually distinct from ratings of headache intensity. Indeed, as an example of a negative headache appraisal, Newton and Barbaree (1987) give the thought: "It's getting worse".

In an investigation of the role of cognitive changes underlying EMG biofeedback training for tension headache (Holroyd et al., 1984), it was found that changes in headache self-efficacy (defined as subjects' judgements of their ability to prevent headache onset) and headache locus of control (defined as the extent to which subjects believe that they can control their headaches) were both correlated significantly with improvement based on the headache index. Reductions in frontalis EMG levels were uncorrelated with changes in headache activity. However, since the self-efficacy and locus of control ratings were very specific to headache, it is possible that headache improvement was mediated by some other mechanism and that, as this improvement occurred, subjects' ratings of their own headache control skills increased, perhaps only as a side-effect of obtaining headache relief. That is, the changes on the rating scales may have reflected headache improvement rather than any underlying mechanism.

4.5. General Comments.

There have been very few investigations into the cognitive process variables that may underlie the effects of psychological interventions on recurrent headache. Those which have been conducted have tended to confound their measures of cognitive processes with outcome variables (Holroyd et al., 1984; Newton and Barbaree, 1987). Thus, the research conducted up to the present time, has yielded little in the way of information on the cognitive changes that may be critical to the success of psychological interventions in general, and to cognitive interventions in particular. This is unfortunate, for some description of the cognitive processes mediating outcome from cognitive therapy would facilitate the development of theoretical
models linking stressful events with headache attacks. This thesis is concerned with the formulation and empirical examination of such a theoretical model. Some progress towards this goal might be achieved by conducting an examination of the cognitive processes underlying cognitive interventions for headache. Such a study is described in Chapter 6. However, before theorising about the kinds of cognitive processes that may mediate the cognitive treatment of headache, it is necessary to attempt some clarification of the concept of cognition and consider its relationship to emotion. This task is undertaken in the next chapter. The relationship between cognition and pain (other than headache) is considered, along with theories of pain and methods of pain measurement, in Appendix A.
CHAPTER 5

COGNITIVE PROCESSES AND EMOTION:

IMPLICATIONS FOR HEADACHE.


The dominant view, expressed in the psychological literature, contends that cognition is a private process that cannot be exposed to public scrutiny (e.g., Mahoney, 1974; Turner, 1967).

Towards the end of the last century, students of experimental psychology studied aspects of cognition, as components of the stream of consciousness, using the method of systematic introspection developed by Wundt. With the advent of behaviourism, heralded by the publication of J.B. Watson's (1913) paper entitled "Psychology as the Behaviourist Views It", there began a vigorous attack on the methods and subject matter of introspection (Boring, 1950). Psychoanalytic theory, which regards emotions as the result of instinctual gratification or frustration, and cognitive processes as developing in Darwinian fashion to service the needs of the organism (Freud, 1920), was also criticised severely for its concern with unobservable structures and processes. Watson's positivism dictated that psychologists ignore the phenomena of consciousness and concepts such as the unconscious, and concern themselves only with observable behaviour. Watson considered thinking to be nothing more than subvocal speech and, in the words of Boring (1950, p. 644), he suggested that feeling "might turn out to be glandular activity or tumescence and detumescence of genital tissues." Such a position has been termed radical behaviourism (Mahoney, 1974).

Beginning with Tolman's (1927) efforts to offer behavioural definitions of cognition, and Mower's (1960) attempts to demonstrate that emotions can be conditioned, cognition and emotion came to be recognised, once again, as legitimate fields of inquiry within experimental psychology.

As pointed out by Mahoney (1977b), within the domain of behavioural psychology, the main impetus for the shift towards the consideration of private events in explanations of human behaviour came from the field of psychotherapy, where Albert Ellis (1962) was the
main protagonist. His emphasis upon cognitive activity, as crucial in the determination of emotion, set the stage for what Dember (1974) has termed a "cognitive revolution".

As a result of the work on cognition and emotion which has mushroomed in recent years (e.g., Beck, 1976; Bandura, 1977; Lazarus, 1966, 1982; Mahoney, 1974; Meichenbaum, 1977), many behaviourally-minded psychologists have been compelled to study these private events and to grapple with the problem of their definition.

5.1.1. Defining Cognition.

The usage of the term "cognition" in psychology has been so broad as to make definition very difficult. One of the major problems in this area is one of defining cognition independently of emotion. This is particularly important for those wishing to study relationships between these two classes of variables (e.g., Folkman, Lazarus, Gruen and De Longis, 1986; Goldfried and Sobocinski, 1975; Harrell, Chambless and Calhoun, 1981). For these purposes, the definition of cognition as "a Person's present thinkings, feelings and willings, his perceivings, rememberings and imaginings" (Ryle, 1949, p. 13) is inadequate, for some cognitive theorists, such as Ellis (1962) and Lazarus (1966), would almost certainly object that feelings are consequences of cognition and must, therefore, be considered as distinct from cognition.

Neisser (1967, p. 4) offered a definition which remains very broad, but is more independent of emotion:

"...cognition refers to all the processes by which the sensory input is transformed, reduced, elaborated, stored, recovered and used. Such terms as sensation, perception, imagery, retention, recall, problem-solving and thinking, among others, refer to hypothetical stages or aspects of cognition."

Theorists concerned with the cognitive processes involved in psychopathology and psychotherapy have focused on such aspects of cognition as belief systems, thoughts, assumptions or self-statements (e.g., Beck et al., 1979; Ellis, 1962; Mahoney, 1974; Meichenbaum, 1977). For example, Ellis (1962) regards irrational beliefs as being causally related to emotional distress. His system of psychotherapy is designed to persuade clients to give up such beliefs and replace them with more rational ones.

It is important to note that cognition has been defined as a process for becoming aware of reality, (Anderson, 1927; Rachlin, 1977), and as a process by which reality is interpreted and evaluated against a background of expectations and values derived from previous
experience (e.g., Beck, 1976; Ellis, 1977). This distinction has been drawn by referring to "cold" and "hot" cognitions (Abelson, 1963); to "knowing" and "appraising" (Lazarus, 1982) or to "non-evaluative" and "evaluative" beliefs (Wessler, 1982). For example, the statement "his name is John" represents a non-evaluative belief, whilst the statement "I don't like John" is an example of an evaluative belief. Theoreticians who draw this distinction are concerned to speak of relations (usually causal relations) between cognition and emotion. Problems arise in this regard when one considers the distinction between evaluative beliefs and emotional reactions. For example, is the statement "I am afraid of the dark" a statement of an emotion (fear) or a statement of an evaluative belief?

At the present moment, only the broadest definitions of cognition can be offered, such that the term refers to the range of perceptual and mental processes of which the human being is capable (e.g., Neisser, 1967). Because of the wide range of processes subsumed into definitions of cognition, certain classes of cognitive processes such as evaluative beliefs (Wessler, 1982) or cognitive appraisal (Lazarus, 1982) are difficult to distinguish from self reports of emotional states.

5.1.2. Defining Emotion.

It is widely recognised that emotion is an ill-defined concept within psychology (Frijda, 1986; Izard, Kagan and Zajonc, 1984; Mandler, 1979). As Mandler (1979, p. 279) notes, "there is no commonly, even superficially, acceptable definition of what a psychology of emotion is about." William James (1890) defined emotion in terms of the sensations produced by bodily changes:

"...bodily changes follow directly the perception of the exciting fact, and...our feeling of the same changes as they occur is the emotion" (James, 1890, p. 449).

The implication of James'(1890) position is that each of the various emotions should be linked to specific differentiable physiological processes. This suggestion generated much research aiming to associate particular patterns of physiological activity with particular feeling states. This work was summarized by Cannon (1929) who concluded that "the same visceral changes occur in very different emotional states and in non-emotional states" (p. 351). Thus, it was recognised that emotion cannot be defined completely in terms of bodily responses. Accordingly, it was proposed that physiological arousal determines the strength of the emotion, but that the quality of the emotion is determined by the cognitive process of labelling the arousal (e.g., Schachter and Singer, 1962). Thus, arousal was regarded as a necessary but not as a sufficient condition for the occurrence of emotion. Emotion itself was conceived as an interaction of cognitive and physiological activity (Schachter and Singer, 1962). Reisenzein
(1983) argued that the process of labelling arousal as a particular emotion involves two cognitions: one which evaluates a situation as having implications for one's well-being and a second which attributes the arousal experienced in the situation to the evaluative cognition. For example, in a given situation, one may experience a high degree of arousal and know that the situation is dangerous, but the emotion of fear will not be experienced unless one perceives a causal relation between the danger and the arousal (Reisenzein, 1983).

Within the frameworks of Schachter and Singer (1962) and Reisenzein (1983), cognition is regarded as an essential element of any emotional state to such an extent that it is impossible to speak of emotion without reference to cognition.

It is because emotion is such a complex phenomenon that it has proved so difficult to define. Given this complexity, it may be unwise to attach a definition which would necessarily limit the range of phenomena which many theoreticians take to be encompassed by the term, for this could hinder, rather than facilitate, efforts to better understand its nature (c.f., Popper, 1966).

5.1.3. Distinguishing Between Cognition and Emotion.

The separation of cognition and emotion is crucial for those wishing to examine inter-relationships between these variables. The approach taken most commonly by researchers concerned with exploring relationships between cognition and emotion is to delimit those aspects of cognition and emotion that are of interest and attempt to offer independent operational definitions of each (e.g., Folkman, Lazarus, Gruen et al., 1986; Goldfried and Sobocinski, 1975; Harrell et al., 1981; Lazarus, 1966). What is necessary for this kind of approach, however, is that theoreticians conceptualise their cognitive constructs such that these can, as far as possible, be assessed independently of emotional responses.

Thus, for the purposes of identifying cognitive aspects of stress that may contribute to the onset and/or severity of headache attacks, the concept of cognition will be employed to refer to all of the mental processes described by Neisser (1967), of which the process of cognitive appraisal (Lazarus, 1982), will be regarded as a sub-category. However, at each stage of the research, the particular cognitive appraisal processes under investigation will be conceptualised and assessed so as to minimise any overlap with emotion.
5.2. Relationships Between Cognition and Emotion.

The nature of the relationship between cognition and emotion has been a matter of considerable debate during recent years. The main protagonists in the argument have been Richard Lazarus (1982, 1984) and Robert Zajonc (1980, 1984).

Lazarus (1982, 1984) champions the view that cognition is both necessary and sufficient for the occurrence of an emotional response. In this respect, he is arguing the line taken by the cognitive therapists (e.g., Beck, 1976; Ellis, 1962), namely, that cognition antecedes emotional responses. The essential point made by Lazarus is that, if some input (an environmental event, sensation, thought or memory) is to trigger an emotional response, it must first be recognised by the receiver as having implications for his or her well-being.

In support of their position, Lazarus and his colleagues (e.g., Koriat, Melkman, Averill and Lazarus, 1972; Lazarus, 1966) have shown that emotional reactions to unpleasant films can be manipulated by altering the accompanying commentary. The conclusion offered is that the commentary altered subjects' cognitive activity, thereby, effecting some change in their emotional response.

In opposition to Lazarus, Zajonc (1980, 1984) views cognition as a sufficient but not as a necessary condition for emotion. He speaks in terms of the processing of sensory input by neurophysiological structures such as the non-dominant cerebral hemisphere, limbic system and hypothalamus. He argues that this processing can generate an emotional response in the absence of cognitive activity.

Zajonc (1980, 1984) supports his position by reference to the observation that subliminally presented stimuli, that cannot be recognised by the subject, produce predictable emotional reactions (Kunst-Wilson and Zajonc, 1980). In addition, Zajonc (1980) reports a series of studies demonstrating that subjects can make reliable like-dislike judgements about subliminally presented material. However, these studies do not rule out the possibility that cognitive processes might operate outside the field of conscious awareness.

The positions taken by Lazarus (1982, 1984) and Zajonc (1980, 1984) may both be oversimplifications of what is undoubtedly a complex relationship between cognition and emotion. For example, the evidence presented by both sides could be accommodated by the hypothesis that there exists a bidirectional relationship between cognition and emotion (e.g., Gilligan and Bower, 1984; Meichenbaum and Butler, 1980). That is, cognition may determine emotional responses and vice versa. Indeed, both Lazarus (1982) and Zajonc (1980) recognise a reciprocal relationship between the variables. Their argument concerns the determination of
the initial emotional or cognitive response to a stimulus. If one recognises that these phenomena operate in a causal chain, then the proponents of cognitive therapy are justified in proposing that it may be possible to eliminate, or ameliorate, emotional distress by manipulating cognitive activity (e.g., Beck, 1976; Ellis, 1962; Meichenbaum, 1977). The position taken by these theorists is outlined in the next section.

### 5.3. Theories Relating Cognition and Emotional Distress

Albert Ellis (1962) was one of the first to implicate specific cognitive processes in the aetiology of such human problems as anxiety and depression, and to apply such a theory to the practice of psychotherapy. His writings on rational-emotive theory and therapy have had a profound influence upon the field of behaviour therapy, and laid the ground work for the development of a variety of approaches to the treatment of psychological disorders, all of which share an emphasis upon the modification of cognitive appraisal as crucial to the effective treatment of these disorders. Such procedures as Beck's cognitive therapy for depression (Beck, 1976; Beck et al., 1979), Meichenbaum's (1977) self-instructional training, as well as the various coping skills training (e.g., Goldfried, 1977; Kazdin, 1973) and problem solving (e.g., D'Zurilla and Goldfried, 1971; Mahoney, 1974) therapies owe their origins in large part to Ellis' (1962) contributions.

Of all the cognitive therapists outlining their approach in the psychological literature, Ellis (1977a, b) has been most concerned with examining the theoretical constructs underlying his method of psychotherapy. Beck, for example, is content to speak of "negative" views of the self and the future, and of "selective-abstraction" of the "negative" aspects of situations (Beck et al., 1979), with little regard for specifying the nature of these constructs. The only clue he gives is that they are associated with dysphoria. Similarly, Meichenbaum (1975) differentiated between "worrying" self-statements and "positive coping" self-statements. Both Beck and Meichenbaum invest little effort in elaborating the specific nature of their theoretical constructs. As a result, these constructs are easily confused with the emotional reactions they seek to explain.

In contrast, Ellis (1977a, b) has drawn a distinction between rational and irrational belief systems and has, on occasions, attempted to describe these independently of emotional responses. He regards irrational beliefs as those beliefs which demand that the environment, self or others, be different from the way they are in reality and conform, instead, to one's own desires and expectations (Ellis, 1977b). According to Ellis (1962, 1977a, b), individuals who hold irrational beliefs are more likely to become emotionally distressed in the face of stressful events than those who hold rational beliefs (beliefs that reflect an acceptance of the
environment, self and others). Thus, for Ellis (1962), the task of psychotherapy is one of persuading clients to give up their irrational beliefs and to replace these with more rational philosophies. There remains the problem of specifying the extent to which a thought must be demanding before it can be termed irrational. This issue will be considered in the next chapter. However, it is probably because Ellis (1977a, b) has attempted to ascribe some empirical quality to his concept of an irrational belief that his theoretical framework has generated more research into its assumptions than those of Beck (1976) or Meichenbaum (1977).

The distinction between rational and irrational beliefs and the research pertaining to the relationship between such beliefs and emotional distress will be considered in the next chapter.

Bandura's (1977) concept of self-efficacy is allied to the position taken by Ellis (1962), Beck (1976) and Meichenbaum (1977), insofar as it is conceptualised as a cognitive mechanism, regulating behavioural and emotional responses (Bandura, 1977, 1982, 1986).

Self-efficacy is defined as "the conviction that one can successfully execute the behaviour required to produce the outcome" (Bandura, 1977, p. 193). Bandura differentiates self-efficacy from outcome expectations, which he defines as "a person's estimate that a given behaviour will lead to certain outcomes" (Bandura, 1977, p. 193). It has been argued that the concept of self-efficacy becomes vacuous unless it is tied to specific outcomes and that it is, therefore, impossible to define self-efficacy independently of outcome expectations (e.g., Eastman and Marzillier, 1984; Marzillier and Eastman, 1984). This assertion will be examined in Chapter 7.

Bandura (1977) postulated a relationship between self-efficacy and emotion, mediated by the acquisition of coping responses. He argued that a low level of self-efficacy in managing the aversive aspects of a situation leads to avoidance behaviour which, in turn, stifles the development of coping skills. In Bandura's view, it is the perceived lack of competency in the face of aversive environmental conditions which generates the accompanying emotional distress. Bandura (1977) points out, however, that emotional arousal can also be a source of feedback which may contribute to the level of self-efficacy.

In support of his theory, Bandura (1977, 1982) has conducted a number of studies demonstrating that the fear experienced by snake and spider phobics, when approaching the feared object, is a function of their level of self-efficacy. Behavioural procedures, such as modelling, were found to result in an increased willingness to approach the feared object and in reductions in the level of self-reported anxiety. These responses were observed to be correlated with measures of subjects' perceived self-efficacy to manage anxiety and the feared
object. Furthermore, self-efficacy to approach feared objects has been found to be a better predictor of avoidance behaviour than indices of autonomic arousal (Bandura, 1978; Bolles, 1972).

Laboratory studies, indicating that subjects report less anxiety, demonstrate lower levels of autonomic arousal and show less impairment on task performance, when they believe they have control over an aversive stimulus (e.g., Averill, 1973; Miller, 1979, 1980), are also consistent with Bandura's (1977, 1982, 1986) position.

Lazarus and Folkman (1984a, b) have focused upon primary and secondary appraisal processes as determinants of coping responses and emotion. A primary appraisal refers to the degree of threat the situation is judged to present to the person. A secondary appraisal refers to the person's evaluation of his or her capacity to manage or obviate the threat. According to Lazarus (1977), emotion is a direct result of these appraisal processes. Lazarus and his colleagues (Lazarus, 1977; Lazarus and Folkman 1984a, b) argue that when a person's perceived capacity to manage the demands of a threatening situation are taxed, that person will experience emotional distress. There follows a complex sequence of coping actions, reappraisal processes and emotional responses as the individual attempts to resolve the situation:

"As a result of constant feedback and continuing efforts to cope with the situation or to regulate the emotional response, the person is also constantly re-appraising his relationship with the environment, with consequent alterations in the intensity and quality of the emotional reaction" (Lazarus, 1977, p. 149).

The above review of some of the ways in which cognition has been related to emotional distress is not meant to be exhaustive. Other formulations such as Kelly's (1955) personal construct theory, and Seligman's reformulated theory of learned helplessness (Abramson, Garber and Seligman, 1980) are recognised as having much in common with the theoretical positions already considered. Cognitive theories of emotional distress, whilst differing in their specification of the relevant cognitive constructs, all view human emotional reactions as being determined by symbolic representations of reality.

5.4. Implications for Headache.

Stress is widely recognised as the most common precipitant of headache attacks (e.g., Friedman, 1979; Friedman et al., 1954; Howarth, 1965; Selby and Lance, 1960). The application of such stress management techniques as relaxation training, biofeedback training
and cognitive therapy reflects this assumption. As discussed in Chapter 4, however, the processes by which relaxation and biofeedback treatments produce relief are not clear, whilst the processes underlying the effects of cognitive therapy have barely been addressed.

Given that cognitive processes have occupied central positions in theories of emotional distress (e.g., Beck, 1976; Ellis, 1962; Lazarus, 1977) and that, therapy designed to manipulate cognition has been found to be more effective for headache than EMG biofeedback (Holroyd et al., 1977; Holroyd and Andrasik, 1982b) or placebo (Brown, 1984; Figueroa, 1982), it would seem reasonable to suggest, by way of an hypothesis, that cognitive appraisal processes may play a role in the aetiology and / or maintenance of recurrent headache. However, very little research has been conducted on the role of cognitive appraisal as a mediator of improvements in headache activity following psychological interventions. Those studies that have attempted to address this issue have confused measures of cognitive constructs with outcome variables (e.g., Holroyd et al., 1984; Newton and Barbaree, 1987), thereby limiting the value of the results obtained (see Chapter 4).

5.5. The Focus of the Present Research.

The studies reported in Chapters 6 and 7 of this thesis aim to identify the particular cognitive appraisal processes that may mediate the relationship between stressful events and recurrent headache. The thesis is also concerned with examining the prospects for linking a cognitive conceptualisation of the stress-headache relationship with the mechanism of sensory modulation, this task being undertaken in the final study, reported in Chapter 8.

In a step towards the description of the cognitive appraisal processes that may contribute to headache, the next chapter is concerned with the distinction between rational and irrational beliefs (Ellis, 1962, 1977a, b) and with an examination of the role of rationality, as a mediating variable, in the rational-emotive therapy of recurrent headache.
CHAPTER 6

STUDY I

THE EFFECTS OF RATIONAL-EMOTIVE THERAPY ON HEADACHE:

THE ROLE OF RATIONALITY IN THE MEDIATION

OF TREATMENT RESPONSE.

6.1. INTRODUCTION.

6.1.1. The Distinction Between Rational and Irrational Beliefs.

Ellis' (1962, 1977a, b) rational-emotive theory asserts that it is the way in which individuals interpret situations that determines their emotional response. Ellis contends that the process of interpretation is carried out within the context of a belief system. It is the nature of this belief system which shapes the interpretive process, and thus generates the emotional response. In order to account for the wide variation in the intensity of the human emotional response to situations, Ellis (1962, 1977a, b) has characterised belief systems as being rational or irrational. According to rational-emotive theory, stressful events lead a person to experience aversive emotions regardless of whether their belief system is rational or irrational; the intensity of that aversive emotion being a function of the extent to which the belief system is irrational. Thus, the distinction between rational and irrational belief systems is a crucial conceptual issue in rational-emotive theory.

Ellis (1977b, p. 15) defined irrationality as:

"any thought, emotion or behaviour that leads to self-defeating or self-destructive consequences that significantly interfere with the survival and happiness of the organism."

In a later article (Ellis, 1980, p. 2) he defined rationality in these terms:

"If people choose to stay alive and try to be happy, they think "rationally" or "self-helpfully" when they think, emote or behave in any way that abets these goals."
These definitions, however, are far too broad. If irrationality is to be a characteristic of cognition within a theoretical system seeking to explicate relationships between cognition, emotion and behaviour, then to apply the term to all three sets of phenomena makes it difficult to speak of irrationality as a quality of cognition which affects emotion and behaviour. Furthermore, both definitions imply that rationality and irrationality can be known only by their effects on the "happiness of the organism"; but it is precisely such emotional responses that Ellis is seeking to explain. If rational and irrational cognitions can be known only by their emotional or behavioural effects, it becomes logically impossible to test the theory, as one is left with no way of independently assessing the variables alleged to be inter-related.

Later in his article "The Basic Clinical Theory of Rational Emotive Therapy", Ellis (1977b, p. 9) offers a more promising definition of irrational beliefs as:

"...demanding, commanding statements about what should and must happen so that you can absolutely and necessarily get what you desire."

It follows from this definition that an irrational belief is a belief which demands particular outcomes or alterations to reality, while a rational belief is one which is accepting of oneself, others and the environment as they are (Ellis, 1962). Following his argument that irrational beliefs contain demandingness components, Ellis (1977a, b, 1980) suggests that statements involving the words "should" or "must" are often irrational. He refers to these as "musturbatory ideas" (Ellis, 1977b, 1980). However, complications arise for Ellis when he attempts to draw a distinction between the rational desire for reality to be different and the irrational demand that it should or must be different. This distinction blurs very quickly unless we are very particular about our usage of the English language. People often use words such as "must" or "should" when the meanings ascribed by Ellis to these terms are clearly not intended. For those not acquainted with rational-emotive theory, the following statements would almost certainly be regarded as meaning much the same thing:

1. I must get to the post office before it closes.

2. I desire to get to the post office before it closes.

If one takes Ellis' position to its extreme, the first statement is irrational; the second is rational. Although this point might seem rather trite in the above context, it becomes crucial when we consider the assessment of rational or irrational beliefs. For example, the first item on Jones' (1968) Irrational Beliefs Test states: "It is important to me that others approve of me." If the respondent agrees with the item, he or she obtains a score in the irrational direction. It is
scored as if it read "others must approve of me". However, as the item appears on the test, it would seem to be the statement of a desire or preference and would, thus, be regarded by Ellis as rational.

The distinction between demandingness and desires or preferences would appear difficult to sustain. Confusion surrounding the distinction has pervaded assessment instruments such as the Irrational Beliefs Test, making many of the items ambiguous with respect to the concepts of rationality and irrationality. It is very difficult to conceive of an individual who would agree with statements expressing desires or preferences for certain outcomes, but disagree with statements expressing demands for those same outcomes.

A clearer appreciation of the central construct in rational-emotive theory may follow from a focus on rationality rather than irrationality. Rationality may be defined as an acceptance of oneself, others and the environment (Ellis, 1962). Irrationality may then be conceived as departures from this position. This circumvents the problem of distinguishing between desires and demands.

When acceptance is recognised as a defining characteristic of a rational belief, it becomes possible to speak of a rational or irrational belief system as standing in relations with emotion and behaviour, and to formulate testable hypotheses concerning these relations.

For the purposes of the present study, acceptance is recognised as the essential feature of rationality and will therefore dictate the particular approach to assessment undertaken herein. Existing approaches to assessment will be considered in the next section.

6.1.2. The Assessment of Irrational Beliefs.

The measure of irrational beliefs employed most commonly by researchers is the Irrational Beliefs Test (IBT; Jones, 1968). The instrument consists of 100 items organised into ten different categories, each designed to assess one of ten irrational beliefs specified by Ellis (1962). The items are scored on a 5-point scale ranging from "strongly disagree" to "strongly agree". Summing the scores within categories gives a measure of the strength with which each irrational belief is held. Summing across categories gives a total irrationality score.

By means of factor analysis, Jones (1968) established that the 100 items could be separated into 10 different factors, each representing one of the irrational beliefs described by Ellis (1962).
Jones (1968) demonstrated that all but two of the IBT subscales were correlated significantly with maladjustment scores on the 16 PF questionnaire (Cattell and Stice, 1957), and claimed this as evidence supporting Ellis (1962) assertion that irrational beliefs are related to emotional distress. However, as has been pointed out by a number of critics (e.g., Malouff and Schutte, 1986; Smith and Zurawski, 1983), the fact that scores on the IBT correlate with such aspects of emotional distress as depression (Nelson, 1977; Vestre, 1984) and anxiety (Deffenbacher, Zwemer, Whisman, Hill and Sloan, 1986; Smith and Zurawski, 1983) raises the question of whether the IBT assesses dimensions any different from those which might be more parsimoniously subsumed under the concept of emotional distress. Indeed, many of the items on the IBT seem to refer to emotional states. For example: "I often worry about how much people will approve of and accept me"; "I feel little anxiety over unexpected dangers or future events"; "I often get disturbed over situations I don't like"; "I often become quite annoyed over little things". This confounding appears to have arisen because Jones (1968) did not ascribe any particular quality to the concept of an irrational belief, but instead followed one of Ellis' (1962) rather confused definitions, and finished up writing many items which refer to the effects of irrational beliefs rather than to the beliefs themselves.

The Idea Inventory (Kassinove, Crisci and Tiegerman, 1977) also purports to measure irrational beliefs but, like the IBT, many items refer to emotions such as worry, anxiety or depression.

Shorkey and Whiteman (1977) developed the Rational Behaviour Inventory (RBI), a confusing title given that, within Ellis' (1962) model, rationality is regarded as a characteristic of cognition rather than of behaviour. The instrument was developed along similar lines to those followed by Jones (1968). This scale consists of eleven subscales. Once again, the development of the scale was not based upon a clear conceptualisation of the nature of rationality and many of the items refer to emotional states. Accordingly, the RBI contains the same deficiencies as those outlined for the IBT.

In a recent effort to construct a unitary measure of irrationality, Malouff and Schutte (1986) developed a 20-item scale drawn from a large pool of items considered by the authors to reflect the content of the ten irrational beliefs identified by Ellis and Harper (1961) and attempted to exclude references to emotional states. Although the scale represents an improvement over previously developed scales and is reported to have a high internal consistency, what it is that is actually being measured is not clearly specified. The current usage of the term rationality within the cognitive-behavioural literature is far too vague for it to be treated as the focus of an assessment instrument without some qualification of the kind outlined in the previous section.
In view of the above considerations, existing measures of rationality were deemed inadequate. Accordingly, and as a prelude to the first study, a scale designed to assess rationality, conceptualised in terms of acceptance and excluding emotional states, was developed.

6.1.3. Relationships Between Irrational Beliefs and Emotional Distress.

As mentioned previously a number of studies report significant correlations between measures of irrational beliefs and pencil and paper measures of emotional distress (e.g., Deffenbacher et al., 1986; Nelson, 1977; Vestre, 1984). The extent to which these studies can be regarded as supportive of the rational-emotive model is limited by the degree of confounding between the different measures of the constructs. Furthermore, these studies involve only two of the three features which make up the rational-emotive model, ignoring the relationship between irrational beliefs and stressful events. This latter issue was addressed by Smith, Boaz and Denney (1984) who examined relationships between the RBI and a measure of life events in the prediction of emotional (Psychological Screening Inventory; Lanyon, 1970) and physical (Health Problems Inventory developed by the authors) distress. Multiple regression analysis revealed a significant interaction between life events and the RBI on the measure of physical distress, such that the correlation between life events and the Health Problems Inventory was greater for subjects scoring high on irrational beliefs than for those scoring low. This result is consistent with the rational emotive model. However, no such interaction was observed on the Psychological Screening Inventory. The pattern of findings is difficult to interpret in terms of rational-emotive theory, but as the authors point out, any interaction between the life events index and the RBI on the Psychological Screening Inventory may have been attenuated by the contamination of the two latter scales. The RBI is far less likely to have been contaminated with the Health Problems Inventory.

Goldfried and Sobocinski (1975) required subjects to imagine scenes involving social rejection. In comparison with those scoring low on irrational beliefs, as assessed by the IBT, those scoring high demonstrated greater increases in hostility and anxiety, as assessed on the Multiple Affect Adjective Checklist (Zuckerman and Lubin, 1965). Although the results of this study are consistent with rational-emotive theory, Craighead, Kimball and Rehak (1979) were unable to replicate the findings. However, Craighead et al (1979) did observe subjects scoring high on the IBT to make more negative self-referent statements following social rejection imagery than their low scoring counterparts; this finding being consistent with Ellis' (1962; 1977a, b) position.
Cash, Rimm and MacKinnon (1986) attempted to induce differential mood states in subjects scoring high or low on irrational beliefs by means of the Velten (1968) Mood Induction Procedure, in which subjects read sets of statements emphasising negative, positive or neutral self-evaluations. In response to the negative statements, high scorers on the IBT demonstrated greater increases in anxiety and depression on the Multiple Affect Adjective Check List than subjects scoring low on the IBT (Cash et al., 1986). No such interactions were observed for the positive mood induction condition. The results support the contention that irrational beliefs render individuals vulnerable to the experience of anxiety and depression (Ellis, 1962; 1977a, b). However, in order for it to be demonstrated that irrationality is the essential dimension constituting this vulnerability, it would have to be shown that measures of irrationality predict responsiveness to negative mood induction better than trait measures of emotional distress such as the various anxiety and depression scales.

Although the studies cited above present few findings which directly contradict rational-emotive theory, the supportive results reported are tempered by the existence of alternative explanations of the data and by the contamination between measures of irrational beliefs and emotional distress. It has been suggested that any causal relationship between irrational beliefs and emotional distress might be elucidated most effectively by treatment studies (Smith, 1982). This body of research is considered in the following paragraphs.

Reviews of the effects of rational-emotive therapy indicate that the treatment is effective in reducing emotional distress in clients presenting with a range of problems (e.g., Rachman and Wilson, 1980; Sutton-Simon, Di Giuseppe and Miller, 1978). However, as pointed out by several authors (e.g., Mahoney, 1977a; Smith, 1982), favourable outcome studies bear no logical relation to the truth of rational-emotive theory. In addition, consideration of the outcome literature is complicated by the lack of uniformity in the treatment procedures employed (Rachman and Wilson, 1980). It is those studies concerned with an evaluation of the role of irrational beliefs in the process of rational-emotive and related cognitive interventions, which are most likely to cast light on the question of whether reductions in emotional distress are mediated by alterations in irrational beliefs. It is to those studies that we now turn.

Consistent with the rational-emotive model, several studies report that, in the rational-emotive therapy of anxiety (Lipsky, Kassinove and Miller, 1980; Trexler and Karst, 1972), depression (Lipsky et al., 1980), and under-assertiveness (Alden, Safran and Weideman, 1978), improvements on outcome measures are accompanied by diminishments in irrational thinking. However, these studies provide rather weak evidence for the theory, for no attempt was made to correlate changes in irrational beliefs with changes on outcome measures. According to rational-emotive theory, alterations in irrational thinking constitute the specific mechanism mediating treatment effects. It follows, therefore, that reductions in emotional
distress will be achieved to the extent that irrational beliefs are modified and that these two classes of variables will thus be correlated over the course of treatment. To the author's knowledge, only one study (Smith, 1983) has pursued this line of inquiry.

Smith (1983) re-analysed data originally collected by Lipsky et al (1980). Lipsky et al (1980) assigned 50 community mental health patients seeking psychotherapy to one of five conditions: rational-emotive therapy, rational-emotive therapy plus rational role reversal (a technique whereby the therapist and client switch roles for a short period), rational-emotive therapy plus rational-emotive imagery (a technique whereby clients imagine themselves disputing irrational beliefs) an alterative treatment (relaxation training plus supportive counselling) or to a waiting list control condition. Lipsky et al (1980) observed that on a number of measures of emotional distress (the Anxiety and Depression subscales of the Multiple Affect Adjective Check List, the Trait Anxiety Scale and the Neuroticism Scale of the Eysenck Personality Inventory), as well as on the Idea Inventory, equivalent reductions were observed for each of the rational-emotive conditions, which were greater than those observed for the alternative treatment and waiting list control conditions. Smith (1983) determined change scores for each variable and then adjusted these for baseline levels by means of multiple regression analysis. He then correlated the adjusted irrational beliefs scores with those for each of the outcome measures. The correlations were calculated for subjects receiving some form of rational-emotive therapy; separate correlations were calculated for those placed in one or the other of the control conditions. Of the eight correlations calculated, seven were statistically significant and in the expected direction. Significant correlations were observed irrespective of treatment condition. Such a pattern of results is consistent with the rational-emotive model.

Once again, however, and as Smith (1983) acknowledges, the possibility that the measure of irrational beliefs employed was confounded with the measures of emotional distress cannot be discounted as an alternative interpretation of the findings. Indeed, inspection of the baseline correlations between the outcome measures and the irrational beliefs measure tends to support this view. For example, in the pre-treatment scores, the Idea Inventory was found to correlate -0.68 with the Neuroticism Scale.

In conclusion, efforts to elucidate relationships between irrational beliefs and emotional distress have been plagued by the potential for confounding of the measurement instruments which seem to have flowed from ill-conceived notions concerning the nature of rationality. This state of affairs has precluded a thorough examination of the basic tenets of the rational-emotive model.
6.1.4. Aims of Study I.

If stress is a major precipitant of headache attacks (e.g., Friedman et al., 1954; Levor et al., 1986) and if the impact of stressful events upon an individual's emotional and physical well-being is a function of the extent to which that individual thinks irrationally (Ellis, 1962; Ellis, 1977a, b) it follows that, interventions which strengthen the tendency to think rationally, will lead to reductions in headache activity. Study I aimed to design a measure of rationality reflecting the quality of acceptance (outlined at the beginning of this chapter) rather than aspects of emotional distress, and apply this to an investigation of the role of rationality in the rational-emotive therapy of recurrent headache. In this manner, the contribution of rationality to headache was evaluated.

6.2. ASSESSMENT OF THEORETICAL CONSTRUCTS.

6.2.1. Development of The Rationality Scale.

Following the definition of rationality in terms of acceptance of oneself, others and the environment, and bearing in mind the need to separate measures of rationality from measures of emotional distress, a twelve item scale assessing rationality (the Rationality Scale) was developed.

The one hundred items on the Irrational Beliefs Test (Jones, 1968) served as the item pool. All items involving the self-disclosure of emotional states were eliminated. Twenty four items were eliminated on this basis (see appendix B). From the 76 remaining items, those scored by Jones (1968) in the direction of irrationality were eliminated (see Appendix B). Presumably, Jones (1968) regarded the remaining 41 items as in some way being measures of rationality, as these items are reverse scored on the original test. From these 41 items, the author selected 12 items considered to be representative of statements pertaining to acceptance of oneself, others and the environment. The Rationality Scale consisted of these 12 items (see Appendix B). As with the irrational Beliefs Test, each item is scored on a 5-point scale with responses ranging from "strongly agree" to "strongly disagree". The sum of the scores obtained for each item gives an index of rationality, such that higher scores are indicative of higher levels of rationality.

All items on the Rationality Scale are scored in one direction, making it susceptible to response bias. Accordingly, it was decided to leave the twelve items embedded in the Irrational Beliefs Test, administer this test to subjects in its entirety, and then separate the twelve
rationality items prior to conducting the statistical analysis. It was considered that such a procedure would be less likely to result in response bias than simply presenting subjects with the twelve Rationality Scale items.

Psychometric data pertaining to the Rationality Scale is presented later in this chapter and in Appendix B.

6.2.2. The Assessment of Headache Activity.

Following previous research, headache activity was assessed by means of a diary which subjects completed daily (e.g., Budzynski et al., 1970, 1973; Holroyd and Andrasik, 1978, 1982a). Headaches were recorded in booklets, each consisting of seven printed sheets. The booklets were designed to be small and easy to carry so that subjects could keep a booklet with them at all times. Provision was made to record the intensity of headache hourly between 6.00 am and 5.00 am on the following day (Budzynski et al., 1970, 1973). However, instead of the six category scales employed as measures of headache intensity by Budzynski et al (1970, 1973), it was decided to present subjects with visual analogue scales on which they could indicate their headache intensity for each hour of the day. Visual analogue scales were preferred to category rating scales in view of the evidence suggesting that the former may be more sensitive to treatment effects in the study of chronic pain patients (Scott and Huskisson, 1976). The visual analogue scales consisted of horizontal lines, one hundred millimetres in length. The left hand anchor point was labelled "No headache", and the right hand anchor point, "Headache as bad as it could be". Thus, subjects indicated the intensity of headache by placing a mark at the appropriate point on the scale. For hours that they were asleep or headache-free, subjects simply left the scale blank. This step was taken in order to minimise the effort involved in recording, by allowing subjects to be unconcerned with the diary during the hours that they were headache-free.

The headache recording sheets required subjects to indicate the type and quantity of medication consumed on a particular day. They were also required, where possible, to provide a brief description of the circumstances surrounding the onset of any headache that occurred.

An example of a headache recording sheet and its associated instructions appear in Appendix C.
From the headache diaries, the following measures of headache activity were derived:

1. **Headache Frequency:** Defined as the number of days in the week on which the subject recorded a headache. It was recognised that rather than experiencing a single headache on a day, subjects might suffer two or more discrete headaches, separated by a number of headache-free hours. However, it was considered that such a pattern of headache could often be the result of repeated doses of medication. That is, the headache may disappear for the period over which any medication is active, only to return once the analgesic effects wear off. Cycles such as these would result in a spuriously inflated measure of headache frequency if one were to count the number of discrete headache episodes occurring each day. Accordingly, this practice was avoided in favour of a method whereby headache frequency is defined as the number of days in the week for which some headache activity is recorded, regardless of the number of discrete headache episodes appearing on those days.

2. **Headache Duration:** Defined as the mean number of headache hours recorded on headache days. Other researchers have defined headache duration as the total number of headache hours recorded over any given period (e.g., Andrasik and Holroyd, 1980; Budzynski et al., 1970, 1973). However, this method of measurement was not preferred as it is less independent of headache frequency than one defined in terms of the mean number of headache hours recorded on headache days.

3. **Headache Intensity:** The intensity of headache for each hour was determined simply by measuring the distance, in millimetres, from the left hand anchor point on the visual analogue scale to the mark made by the subject. In the determination of the mean headache intensity over any given period, zero ratings were not included. Thus, headache intensity was defined as the mean intensity rating for those hours on which subjects indicated that a headache was present.

4. **Medication Consumption:** Defined as the mean number of tablets consumed per day for the purposes of headache relief. Such a measure has been employed by researchers on many occasions (e.g., Andrasik and Holroyd, 1980; Bakal et al., 1981; Budzynski et al., 1970, 1973; Figueroa, 1982). Although medications for headache have been scaled according to the strength of their analgesic effects (Coyne, Sargent, Segerson and Obourn, 1976) and these weightings applied to the assessment of medication intake in some treatment studies (e.g., Attanasio et al., 1987; Blanchard et al., 1982a), such a level of precision was considered unnecessary in the present instance, in view of the fact that all subjects were asked not to change their medication during the period of the
study. Thus changes in medication consumption were assessed in terms of quantity rather than quality of medication.

To conceptualise pain measures in unidimensional terms, as is implied by the use of category or visual analogue scales, is a simplification of a multidimensional phenomenon (Melzack and Wall, 1982; see Appendix A). It is recognised that the current assessment device is open to this criticism and that multidimensional instruments such as the McGill Pain Questionnaire (Melzack, 1975) and the Headache Assessment Scale (Hunter, 1983) are available. However, it was decided not to employ these instruments in the assessment of headache in view of the fact that four dependent measures were already under examination, and that the use of a comparative treatment outcome design necessarily restricted the number of subjects that could be included.

6.3. EXPERIMENTAL DESIGN.

The present study was concerned with evaluating the role of rationality in the aetiology and/or maintenance of recurrent headache. It was proposed to carry out this investigation by comparing the effects of rational-emotive therapy and relaxation training on headache activity, and by determining the strength of any correlation between improvements in headache activity and increases in rationality.

Rational-emotive therapy was preferred to the cognitive therapy that has been applied to headache on several previous occasions (e.g., Figueroa, 1982; Holroyd and Andrasik, 1978; Holroyd et al., 1977), in view of the fact that the former employs a much more circumscribed set of procedures with a specific focus on the modification of irrational beliefs. It was therefore considered most suitable for the purposes of the present inquiry. Cognitive therapy for headache, as described in the literature (e.g., Holroyd and Andrasik, 1982a; Holroyd et al., 1977), involves the acquisition of a range of coping skills including problem solving, coping self-statements and calming imagery, as well as an emphasis upon irrational cognition. This multifaceted approach to treatment was not employed because teaching clients various coping skills may bear little relation to rationality and could exert independent effects on headache, thereby obscuring any relationship that may exist between rationality and headache activity.

A relaxation training treatment was employed as a comparison condition against which the effects of rational-emotive therapy could be evaluated. Relaxation training has been reported to be an effective treatment for headache (e.g., Cox et al., 1975; Tasto and Hinkle, 1973; Warner and Lance, 1975), but does not seek to alter irrational beliefs. This treatment,
therefore, provides a control for the effect of non-specific factors (i.e., non-specific with respect to rationality) involved in the psychological therapy of headache.

To investigate the hypotheses of the present study (see Section 6.4.), a 2 x 3 factorial design (with repeated measures on the time factor) was chosen.

The two experimental factors were as follows:

1. Treatment Group with two levels:
   - Rational-emotive therapy.
   - Relaxation training.

2. Time with three levels:
   - Pre-treatment assessment.
   - Post-treatment assessment.
   - Follow-up assessment.

Scores on the Beck Depression Inventory (Beck et al., 1961) have been found to be predictive of headache improvement following progressive relaxation training (Jacob et al., 1983), with subjects scoring high on this inventory having poorer outcomes. Similarly, headache sufferers who spend large proportions of their waking time with headache have been observed to have poorer outcomes from a multi-faceted treatment involving relaxation training, EMG biofeedback and cognitive therapy, than those subjects who have longer periods of headache freedom (Bakal et al., 1981). Thus, in an attempt to increase the precision of the experiment, it was decided to match subjects on each of these variables before assigning them to treatments on a random basis.

6.4. HYPOTHESES.

The hypotheses formulated prior to the collection of data were as follows:

1. Within each treatment there will be significant reductions in headache frequency, intensity, duration and medication consumption at the post-treatment and follow-up assessments, relative to pre-treatment baseline levels. The treatment outcome literature reviewed in Chapter 4 is not sufficiently sophisticated for one to predict that particular dimensions of headache activity will be most responsive to psychological interventions.
2. Rational-emotive therapy will be more effective in reducing headache frequency, intensity, duration and medication consumption than relaxation training.

3. For subjects receiving rational-emotive therapy, there will be a significant increase in rationality over the course of treatment.

4. Any Increase in rationality over the course of treatment will be greater for subjects receiving rational-emotive therapy than for those receiving relaxation training.

5. Within each treatment group, reductions in headache frequency, intensity, duration and medication consumption will be correlated significantly with increases in rationality.

6. Within each treatment group, any reductions in anxiety and depression will be correlated significantly with increases in rationality. This hypothesis follows from the view of rationality as a variable mediating the effects of therapy on emotional distress (Ellis, 1962, 1977a, b).

7. Any reductions in anxiety and depression will be greater for the rational-emotive treatment than for the relaxation treatment.

6.5. METHOD.

6.5.1. Subjects.

The sample of subjects participating in the study comprised 36 headache sufferers aged between 25 and 61 years, with a mean age of 40.5 years (S.D. = 10.2 years). Their years of full-time education ranged from 9 to 25 years with a mean of 14.4 years (S.D. = 3.3 years). There were 20 females and 16 males.

In view of the evidence suggesting that headaches often classified as "tension" or "migraine", tend to fall on a continuum of severity, rather than into distinct subtypes (e.g., Drummond and Lance, 1984a; Waters, 1974), this continuum model (Bakal et al., 1981; Bakal and Kaganov, 1977, 1979) was employed as the conceptual basis for subject selection. In the interests of ensuring some homogeneity of subjects, it was decided to select headache sufferers from the "milder" or "tension" end of the continuum. For this purpose, the exclusion criteria developed by Philips (1977) and also adopted by Janssen (1983) were employed. Prospective
subjects were excluded from participation in the study on the basis of the presence of migrainous features in association with their usual headache. Headache sufferers were excluded if they met either of the following criteria:

1. They reported vomiting in association with their usual headache on more than 50% of occasions.

2. They reported one or more of the following features in association with their usual headache on more than 50% of occasions.
   - Unilateral onset of headache.
   - Nausea.
   - Sensory prodromata.

Given the suggestion that oral contraceptives may exacerbate headache (e.g., Kudrow, 1975), females using oral contraceptives were excluded from the study if they admitted to a worsening of headache at the time that they first began taking birth control tablets.

In addition to meeting the requirements of the above exclusion criteria, those subjects selected were also required to satisfy the following inclusion criteria:

1. They had been suffering from headaches for at least one year.

2. They estimated their headaches to occur on an average of at least 2 days per week.

3. They had made no change to their usual medication for headache in the past month.

4. They had not been involved in any relaxation training, meditation or other stress-management programme at any time during the previous 5 years.

5. They were not currently practising any relaxation or meditation technique.

6. They were aged between 18 and 65 years.

The criteria outlined above resulted in the selection of the 36 headache sufferers described at the beginning of this section.

All subjects selected were required to visit their local medical practitioner and obtain a certificate stating that, to their doctor's knowledge, there was no organic basis to their headaches, and no reason why they should not participate in a stress-management programme.
Subjects were also required to pay a deposit of $10.00 which was refundable upon completion of the experiment.

Prospective subjects were recruited from the Canberra Community by means of newspaper articles and radio announcements, calling for headache sufferers to participate in a study designed to investigate the effectiveness of stress-management methods for headache. The specific details of each treatment were not released.

6.5.2. Measures.

In addition to the headache diary and Rationality Scale described previously, the Beck Depression and Trait-Anxiety Inventories were also administered pre-and post-treatment. These measures have been widely employed in the assessment of emotional distress and have been reported to possess satisfactory levels of reliability and validity (e.g., Anastasi, 1982; Beck, Steer, and Garbin, 1988; Bumberry, Oliver and McClure, 1978; Knight, Hendrika, Waal-Manning and Spears, 1983; Oliver and Burkham, 1979). These measures were included in the present study so as to allow for the examination of any relationship between anxiety, depression and rationality over the course of treatment.

In order to examine whether or not the treatments differed in terms of non-specific variables such as expectations of success and perceived credibility, which have been implicated in the therapeutic process (Frank, 1961), subjects were asked to complete two visual analogue scales assessing these factors. On the first scale they were required to indicate the likelihood of their recommending the treatment to a friend also suffering from headache. On the second scale they indicated how important they thought it that the treatment be available to other headache sufferers (see Appendix D). These scales were administered after the first treatment session and at the post-treatment assessment. The method employed is similar to that described by Holroyd and Andrasik (1978).

6.5.3. Treatments.

The two treatments to be compared in the present study were rational-emotive therapy (Ellis, 1962, 1977a, b) and progressive relaxation training (Bernstein and Borkovec, 1979; Jacobson, 1938), each conducted in groups of five or six subjects. Each type of treatment was delivered by the author, a graduate student in clinical psychology aged 26 years, with some previous experience in the application of rational-emotive therapy and progressive relaxation training in group settings.
Each treatment comprised six weekly sessions, each of 75 minutes duration. Thus, each group of subjects received a total of 7 1/2 hours of therapist contact, which is comparable with the amount of contact offered to subjects in previous group treatment studies for headache (e.g., Figueroa, 1982; Holroyd and Andrasik, 1978).

Throughout both treatments, subjects recorded their headache activity and, where possible, they recorded the situation associated with headache onset (Bakal et al., 1981; Holroyd et al., 1977). Subjects receiving rational-emotive therapy were also asked to record any thoughts associated with those situations.

In the first session of each treatment, subjects were provided with a brief overview of the pathophysiology of tension and migraine headache. This was followed by a description of the relationship between stress and headache. In the progressive relaxation condition, a relationship between muscle tension and headache was emphasised. In the rational-emotive condition, the therapist focused upon the role of cognitive processes, particularly Ellis' (1962) ten irrational beliefs, in the generation of physiological responses likely to precipitate headache episodes.

The rational-emotive therapy followed the procedures outlined by Ellis and Grieger (1977) and Grieger and Boyd (1980). After being introduced to rational-emotive theory, subjects' thoughts regarding stressful events and headache were examined, with the therapist illustrating linkages between these thoughts and Ellis' (1962) irrational beliefs. Through such techniques as rational-emotive imagery, rational role reversal and role plays, as well as in the course of interacting with the therapist, subjects were taught how to detect irrational cognition and how to substitute it with more rational thoughts and self-statements (Ellis and Grieger, 1977; Grieger and Boyd, 1980). Subjects were provided with homework exercises requiring them to analyse their thoughts during stressful encounters and to dispute any irrational thoughts.

Subjects receiving progressive relaxation training were provided with a 40-minute relaxation audio tape of the therapist delivering relaxation instructions along the lines specified by Jacobson (1938). Relaxation exercises were carried out during the sessions by the therapist, and subjects were asked to practise at home at least once per day. As treatment continued, subjects were weaned off the tape and taught how to abbreviate the procedure so as to allow relaxation to be achieved quickly and at the first indication of any increase in tension (Bernstein and Borkovec, 1979). Subjects were encouraged to apply their relaxation skills in every day situations of stress and at the onset of headache attacks (c.f. Goldfried and Trier,
The demand that subjects apply their relaxation skills in every day life was made so that the treatments were similar insofar as they both required the practice of newly acquired skills in daily life.

6.5.4. Procedure.

Prospective subjects telephoned the author at the Psychology Department, Australian National University and were interviewed by him in order to determine whether or not they met the criteria outlined in Section 6.5.1. Those subjects who failed to meet the selection criteria were thanked for their interest and were advised of locations within the community where they could expect to obtain stress-management training. Those subjects who did meet the selection criteria were told that two treatments were being offered, each known to be effective for headache, and that the purpose of the study was to determine whether one treatment was superior to the other. They were told that both treatments involved techniques of stress-management and that they would be conducted in small groups.

Subjects were kept unaware of the details of treatment until they presented for their first treatment session. They were not informed of the nature of the alternative treatment until after the collection of follow-up data, twelve weeks after the final treatment session.

During the initial telephone interview, subjects were informed that the study would be written-up as a thesis to be submitted to the University, but that full confidentiality would be maintained. They were told that they would be required to record their headaches for 4 weeks prior to treatment, throughout the course of treatment for two weeks after treatment and for a further two weeks three months after treatment.

The 36 subjects recruited by means of the above procedures were all sent letters informing them that they had been accepted into the study. These letters detailed the need for subjects to attend a pre-treatment assessment session to which they were asked to bring their medical certificates and deposit of $10.00. A form requiring subjects to give their written consent to participate in the study was also included with the letter. They were asked to bring the completed form to the pre-treatment assessment session. Subjects were subsequently telephoned to arrange appointments for this assessment.

The pre-treatment assessments were conducted in groups of between five and ten subjects. The experimenter reiterated the purpose of the study, collected medical certificates and deposits and then administered the Irrational Beliefs Test, incorporating the Rationality Scale, and the Beck Depression Inventory.
Subjects were informed that all treatment sessions would be held on four evenings of each week, and that they would be required to attend one of these treatment sessions each week for six weeks. Subjects were provided with a form detailing the days and times on which it was proposed to conduct the sessions and required to indicate those times at which they would be unable to attend because of scheduling difficulties.

The method of headache recording was explained and each subject was provided with six headache recording booklets, allowing for six weeks of continuous headache recordings. The importance of maintaining accurate records was impressed upon subjects. They were encouraged to keep a booklet with them at all times so that when a headache occurred, they could record its intensity on an hourly basis.

Subjects commenced their headache recordings immediately after the pre-treatment assessment. As soon as they had completed two weeks of recording, they were asked to return the completed booklets to the experimenter in stamped self-addressed envelopes which were provided. This material was used to match subjects on the number of hours for which headache was present. Thus, subjects were told that treatment could not begin until two weeks after all of them had returned the first two weeks of headache recording. All subjects returned the booklets promptly.

In order to match the headache sufferers on Beck Depression Scores and on the total number of headache hours suffered in the initial two week period, subjects were divided first into depressed and non-depressed groups on the basis of a median split on Beck Depression Scores. Those scoring at 6 or above were classified depressed; those scoring below 6 were classified non-depressed. Thus the median score fell towards the lower end of the Beck Depression Inventory. Scores of 8 or more on this inventory have been observed to be predictive of a poor response to relaxation training, while scores of 3 or less have been associated with better outcomes (Jacob et al., 1983). Since the obtained median score fell between these two figures, it was deemed an appropriate cut-off level.

Within each of these classifications subjects were then matched on the basis of the number of headache hours recorded on the two booklets returned by mail to the experimenter. As far as their schedules would allow, subjects within each matched pair were assigned to one of the two treatments on a random basis. As a result, 8 men and 10 women were assigned to each treatment.

The following two weeks of headache recording made by subjects served as the pre-treatment baseline measure of headache activity (c.f. Blanchard, Andrasik, Neff, Arena et al., 1982; Blanchard, Andrasik, Neff, Teders et al., 1982; Budzynski et al., 1970, 1973; Holroyd
and Andrasik, 1978). Subjects continued to record headaches throughout the duration of treatment and for two weeks after treatment. Data from this latter period were employed in the determination of post-treatment measures of headache activity. Twelve weeks after the completion of treatment, subjects recorded their headaches for a further two weeks; this recording period allowing for follow-up measures of headache activity.

Subjects brought the headache recordings pertaining to the pre-treatment baseline with them to the first treatment session and returned them to the experimenter. Prior to the commencement of this session, the Irrational Beliefs Test, incorporating the Rationality Scale, and the Beck Depression Inventory were re-administered. The Trait Anxiety Inventory was also administered. These questionnaire responses were employed to assess pre-treatment levels of rationality, depression and anxiety. The same questionnaires were administered at the close of the final treatment session and thereby provided post-treatment measures of these same variables. Subjects were provided with enough headache recording booklets to last for the duration of treatment. As they attended for treatment, they brought their completed headache recording booklets with them and passed these on to the experimenter. At the final treatment session, they were provided with two more booklets (required for the post-treatment assessment) and asked to return these completed booklets to the experimenter by mail. The follow-up measures of headache activity were obtained by sending booklets to subjects through the mail with an accompanying letter and stamped self-addressed envelope.

When the experimenter received the final set of headache diaries, the deposits of $10.00 were returned to subjects by mail. During the pre-treatment, post-treatment and follow-up headache recording phases of the study, the experimenter maintained weekly telephone contact with subjects in an effort to maximise compliance.

6.6. RESULTS.

Of the 36 subjects who entered the study, three withdrew during the pre-treatment headache recording period. Two of these subjects (one male assigned to rational-emotive therapy, and one female assigned to relaxation training) claimed that their headaches were no longer a problem, while the third (assigned to rational-emotive therapy) said that he could not afford the time. Each of these subjects had completed the initial two-week headache recording period and returned their diaries to the experimenter. During the first three weeks of treatment one male and one female subject dropped out of each group. Thus by the post-treatment assessment phase, data on headache activity was available from 14 subjects in the rational-emotive treatment (9 females and 5 males) and from 15 subjects in the relaxation treatment (8 females and 7 males). Follow-up data was available from 11 subjects in the rational-emotive
treatment (6 females and 5 males) and from 12 subjects in the relaxation treatment (5 females and 7 males). Three females from each treatment failed to provide follow-up data. These observations indicate that the drop-out rate was very similar for both treatments.

The attrition of subjects from the study made it impossible to maintain the randomised block structure of the design. Accordingly, for the purpose of testing for the significance of differences between treatments, it was decided to analyse the experiment as a completely randomised design (Kirk, 1968).

Two subjects in the rational-emotive treatment group failed to complete the pre-treatment measure of trait anxiety. Thus, analyses involving this variable involved fewer degrees of freedom.

The Beck Depression Inventory scores and the medication consumption scores, were observed to have skewed distributions. Since parametric statistics assume a normal distribution (Kirk, 1968), the obtained values were subjected to a natural logarithmic transformation prior to analysis.

6.6.1. Data Reduction.

The visual analogue headache intensity scale values were determined by measuring the distance from the left hand anchor point to the mark indicated by the subject. This was achieved by means of a bit-pad device which calculated the mean headache intensity of all headache hours recorded by the subject on any day when headache occurred. In this manner, the mean intensity of headaches was determined for each subject within the pre-treatment, post-treatment and follow-up periods. Headache frequency was determined by simply counting the number of days on which headache occurred within each of the recording periods and dividing the total by the number of weeks of recording in each period (2). The mean duration of headaches was determined by calculating, for each subject, the mean number of hours of headache recorded on days when headache occurred within each of the three specified periods.

6.6.2. Treatment Credibility Ratings.

Scores on the two treatment credibility rating scales were highly correlated. Ratings made at the end of the first treatment session correlated 0.94 (N = 33). Those made at the end of the final session correlated 0.91 (N = 29). Accordingly, in comparing the perceived credibility of the two treatments, it was decided to employ the mean of the two scale scores
obtained for each subject. Two-tailed t-tests revealed no significant differences between the treatment groups (see Table 6-1).

Table 6-1
Mean Treatment Credibility Rating Scale Scores.

<table>
<thead>
<tr>
<th>Session</th>
<th>Rational-Emotive Therapy</th>
<th>Relaxation Training</th>
<th>t-value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>One*</td>
<td>78.4 (1.1)</td>
<td>77.4 (1.0)</td>
<td>0.95</td>
<td>N.S.</td>
</tr>
<tr>
<td>Six+</td>
<td>88.7 (0.9)</td>
<td>87.6 (0.8)</td>
<td>1.29</td>
<td>N.S.</td>
</tr>
</tbody>
</table>

*Indicates degrees of freedom = 31.
+Indicates degrees of freedom = 27.

Note: In the above table and in subsequent tables, N.S. indicates a result which fell short of the p < 0.05 level. The standard deviations are given in brackets.

Thus, there were no significant differences between treatments in terms of subjects' credibility ratings.

6.6.3. Rationality Scale.

The reliability and validity of the Rationality Scale were evaluated by recourse to the data obtained when the scale was administered to subjects during the pre-treatment assessment and again at the commencement of the first treatment session. The scale was observed to have a test-retest reliability coefficient of 0.81 for the total score. Employing the data obtained in the initial administration, the scale was observed to have an internal consistency reliability coefficient (Cronbach, 1951) of 0.82. The construct validity of the scale was evaluated by correlating the total scores obtained at the initial administration with the total scores obtained for the remaining 88 items on the Irrational Beliefs Test. The product-moment correlation coefficient was r (36) = -0.48 (2-tailed p < 0.005).

As predicted by rational-emotive theory, scores on the Rationality Scale were found to be negatively correlated with scores on both the Beck Depression Inventory (r (36) = -0.46, 2-tailed p < 0.005) and Trait Anxiety Inventory (r (34) = -0.52, 2-tailed p < 0.005).
6.6.4. Planned Analyses.

The pre-treatment values of all variables studied were compared across the rational-emotive and progressive relaxation groups (for the 29 subjects who completed treatment) by means of t-tests. No significant differences between groups were observed (see Appendix E).

In an analysis of changes in variables occurring over time it is important to consider that pre-treatment, post-treatment and follow-up scores are almost invariably inter-correlated, suggesting that the pre-treatment variable values may be contributing to the magnitude of those values at subsequent assessments (Kinsman and Staudenmayer, 1978). Thus, minor variations in pre-treatment values could influence the significance of any differences between treatments observed at subsequent assessments (Kinsman and Staudenmayer, 1978). It has been suggested that this problem may be overcome by conducting an analysis of covariance on the post-treatment and follow-up scores, with the pre-treatment scores as covariates (e.g., Benjamin, 1967; Kinsman and Staudenmayer, 1978; Lord, 1963). Thus, it was decided to adopt this approach in the analysis of between treatment effects. Following this same logic, it was decided to determine the magnitude of correlations between pairs of variables assessed over the course of treatment, by correlating the post-treatment scores, and controlling any contribution of pre-treatment values by means of partial correlation analysis.

Within treatment effects on headache activity were examined by means of one-tailed paired t-tests on each of the dependent measures. One-tailed tests were carried out because the literature on the effects of progressive relaxation training and cognitive therapy suggest that these techniques are associated with improvements rather than with exacerbations of headache activity (e.g., Cox et al., 1975; Figueroa, 1982). Thus, since the direction of differences could be predicted, one-tailed tests were deemed appropriate (Welkowitz, Ewen and Cohen, 1976).

Since four dependent measures were employed in the assessment of headache activity analysing each separately would involve an inflation of the Type I error rate (Kirk, 1968). It was decided to control this error rate with the Bonferroni correction (Kirk, 1968). This involves splitting the nominal significance level of $p = 0.05$ between the number of comparisons made. Thus, for analyses involving headache activity, the Type I error rate was set at $0.05 / 4 = 0.0125$. This probability level was termed the Bonferroni corrected Type I error rate, as distinct from the nominal Type I error rate ($p = 0.05$). For the examination of hypotheses involving a single dependent variable the nominal significance level was employed.

Given that reliance upon the Bonferroni corrected error rate results in a loss of power to detect significant differences, it was decided to examine each result for significance at both the
Bonferroni corrected and nominal significance levels, but to be cautious about results which are significant only at the nominal level.

A multivariate approach to the analysis of between-groups effects was not employed in view of the observation that this technique is less powerful than the Bonferroni procedure (Bird, 1975).

**Hypothesis 1.**

The significance of any reductions in headache activity that occurred within each treatment group from pre-treatment to post-treatment, and from pre-treatment to follow-up, were evaluated by means of one-tailed paired t-tests. At the post-treatment assessment, subjects receiving rational-emotive therapy demonstrated reductions in headache frequency and intensity which were significant at the Bonferroni corrected level ($p < 0.0125$). The reductions in headache duration and medication consumption were significant only at the nominal level ($p < 0.05$). This pattern of results persisted at the follow-up assessment except that the result for medication consumption was no longer significant at the nominal level (see Tables 6-2 and 6-3).

**Table 6-2**

Pre-Treatment to Post-Treatment Changes in Headache Activity for the Rational-Emotive Group ($N = 14$).

<table>
<thead>
<tr>
<th>Headache Activity</th>
<th>Pre-Treatment Mean</th>
<th>Post-Treatment Mean</th>
<th>Paired t-value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency</td>
<td>5.21 (1.57)</td>
<td>3.36 (2.18)</td>
<td>3.88</td>
<td>0.001</td>
</tr>
<tr>
<td>Intensity</td>
<td>20.20 (12.37)</td>
<td>12.51 (10.63)</td>
<td>3.87</td>
<td>0.001</td>
</tr>
<tr>
<td>Duration</td>
<td>7.60 (4.06)</td>
<td>5.80 (3.98)</td>
<td>1.86</td>
<td>0.043</td>
</tr>
<tr>
<td>Medication*</td>
<td>1.91 (1.41)</td>
<td>1.32 (1.33)</td>
<td>1.87</td>
<td>0.042</td>
</tr>
</tbody>
</table>

*Inspection of the distributions of the pre-treatment, post-treatment and follow-up
medication scores revealed these to be skewed. Thus, the transformation: \( \ln(\text{Medication Score} + 1) \) was carried out on these scores. All medication scores entered in subsequent tables were transformed in this manner.

Table 6-3

Pre-Treatment to Follow-up Changes in Headache Activity for the Rational-Emotive Group (N = 11).

<table>
<thead>
<tr>
<th>Headache Activity</th>
<th>Pre-Treatment Mean</th>
<th>Follow-up Mean</th>
<th>Paired t-value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency</td>
<td>4.73 (1.40)</td>
<td>2.50 (0.98)</td>
<td>5.40</td>
<td>0.001</td>
</tr>
<tr>
<td>Intensity</td>
<td>22.96 (12.63)</td>
<td>17.66 (12.97)</td>
<td>2.71</td>
<td>0.011</td>
</tr>
<tr>
<td>Duration</td>
<td>7.23 (4.48)</td>
<td>5.57 (2.97)</td>
<td>2.28</td>
<td>0.046</td>
</tr>
<tr>
<td>Medication</td>
<td>1.87 (1.54)</td>
<td>1.46 (1.05)</td>
<td>1.55</td>
<td>N.S.</td>
</tr>
</tbody>
</table>

For the subjects receiving progressive relaxation training, the reductions in headache frequency were significant at the Bonferroni corrected level at both the post-treatment and follow-up assessments. For the remaining dimensions of headache activity, no reductions were significant at the nominal level (see Tables 6-4 and 6-5).
Table 6-4

Pre-Treatment to Post-Treatment Changes in Headache Activity for the Relaxation Group (N = 15).

<table>
<thead>
<tr>
<th>Headache Activity</th>
<th>Pre-Treatment Mean</th>
<th>Post-Treatment Mean</th>
<th>Paired t-value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency</td>
<td>4.50 (1.72)</td>
<td>2.47 (2.22)</td>
<td>4.48</td>
<td>0.001</td>
</tr>
<tr>
<td>Intensity</td>
<td>17.76 (12.64)</td>
<td>17.45 (13.94)</td>
<td>0.11</td>
<td>N.S.</td>
</tr>
<tr>
<td>Duration</td>
<td>7.57 (3.18)</td>
<td>7.38 (4.17)</td>
<td>0.27</td>
<td>N.S.</td>
</tr>
<tr>
<td>Medication</td>
<td>1.64 (1.22)</td>
<td>1.16 (1.21)</td>
<td>1.69</td>
<td>0.056</td>
</tr>
</tbody>
</table>

Table 6-5

Pre-Treatment to Follow-up Changes in Headache Activity for the Relaxation Group (N = 12).

<table>
<thead>
<tr>
<th>Headache Activity</th>
<th>Pre-Treatment Mean</th>
<th>Follow-up Mean</th>
<th>Paired t-value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency</td>
<td>4.67 (1.50)</td>
<td>2.75 (2.34)</td>
<td>3.81</td>
<td>0.003</td>
</tr>
<tr>
<td>Intensity</td>
<td>17.66 (14.04)</td>
<td>17.44 (14.16)</td>
<td>0.07</td>
<td>N.S.</td>
</tr>
<tr>
<td>Duration</td>
<td>7.75 (3.50)</td>
<td>7.85 (4.42)</td>
<td>-0.09</td>
<td>N.S.</td>
</tr>
<tr>
<td>Medication</td>
<td>1.71 (1.33)</td>
<td>1.37 (1.26)</td>
<td>1.34</td>
<td>N.S.</td>
</tr>
</tbody>
</table>

Thus, adopting the conservative Bonferroni decision rule, the hypothesis that each treatment would lead to diminutions in headache activity was supported with respect to headache frequency for both treatments and with respect to headache intensity for rational-
emotive therapy. The hypothesis was not supported with respect to reductions in headache duration or medication consumption.

**Hypothesis 2.**

The significance of any differences between treatments in their capacity to reduce headache activity was evaluated by means of analyses of covariance (ANCOVA) on the post-treatment and follow-up scores, employing the pre-treatment scores as covariates. The hypothesis that rational-emotive therapy would be superior to relaxation training in reducing headache activity was not supported for any headache measure when the Bonferroni correction was employed ($p > 0.0125$). Employing the nominal decision rule, a greater reduction in headache intensity was observed to occur for the rational-emotive than for the relaxation group ($p < 0.05$) at post-treatment, but this was not maintained at the 12-week follow-up (see Tables 6-6 and 6-7 where the means are adjusted for the covariates). Accordingly, it was concluded that there was no clearly significant difference between the treatments in their capacity to reduce headache activity (see Appendix F for the ANCOVA summary tables).

**Table 6-6**

Adjusted Post-Treatment Mean Headache Activity Scores for Rational-Emotive Therapy and Relaxation Training Groups ($N = 29$).

<table>
<thead>
<tr>
<th>Headache Activity</th>
<th>Rational-Emotive Adj. Mean</th>
<th>Relaxation Adj. Mean</th>
<th>F-value (df=1, 26)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency</td>
<td>3.06</td>
<td>2.75</td>
<td>0.21</td>
<td>N.S.</td>
</tr>
<tr>
<td>Intensity</td>
<td>11.60</td>
<td>18.32</td>
<td>4.34</td>
<td>0.047</td>
</tr>
<tr>
<td>Duration</td>
<td>5.79</td>
<td>7.39</td>
<td>1.94</td>
<td>N.S.</td>
</tr>
<tr>
<td>Medication</td>
<td>1.32</td>
<td>1.16</td>
<td>0.08</td>
<td>N.S.</td>
</tr>
</tbody>
</table>
Hypothesis 3.

The significance of any increase in Rationality Scale scores over the course of rational-emotive therapy was evaluated through a comparison of the pre-treatment and post-treatment mean rationality scores, with a one-tailed paired t-test. A trend for the post-treatment rationality scores (mean = 32.71; S.D. = 8.37) to be greater than the pre-treatment scores (mean = 29.93; S.D. = 6.21) was observed, but this fell short of the 0.05 significance level (t (13) = 1.64; p = 0.06). Thus, the increase in Rationality Scores over the course of rational-emotive therapy only bordered on significance and cannot, therefore, be regarded as clear-cut.

Hypothesis 4.

It was hypothesised that rational-emotive therapy would lead to greater increases in Rationality Scale scores than progressive relaxation training. An analysis of covariance (employing the pre-treatment scores as covariates) revealed no significant difference (F (1, 26) = 0.02; p > 0.05) between the rational-emotive therapy (adjusted mean = 30.45) and progressive relaxation (adjusted mean = 30.24) conditions on this variable (the means are adjusted for the covariates; see Appendix G for the ANCOVA summary table). Thus, the results did not support the hypothesis.
Hypothesis 5.

Second order partial correlations (controlling for pre-treatment scores) between post-treatment rationality scores and post-treatment headache activity scores were calculated for each treatment.

There was no evidence to support the hypothesised correlation between reductions in headache activity and increases in rationality. All correlations fell short of both the Bonferroni corrected ($p > 0.0125$) and nominal ($p > 0.05$) significance levels (see Table 6-8).

Table 6-8

Partial Correlations: Post-Treatment Rationality Scores with Post-Treatment Headache Activity Measures.

<table>
<thead>
<tr>
<th>Headache Activity</th>
<th>Rational-Emotive (N=14) r (df=10)</th>
<th>Relaxation (N=15) r (df=11)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency</td>
<td>-0.37</td>
<td>-0.19</td>
</tr>
<tr>
<td>Intensity</td>
<td>-0.17</td>
<td>-0.01</td>
</tr>
<tr>
<td>Duration</td>
<td>-0.32</td>
<td>0.25</td>
</tr>
<tr>
<td>Medication</td>
<td>0.05</td>
<td>-0.15</td>
</tr>
</tbody>
</table>

Hypothesis 6.

It was hypothesised that reductions in Beck Depression and Trait Anxiety scores would be correlated significantly with increases in Rationality Scale scores within both treatments. Inspection of the distributions of the pre-treatment and post-treatment Beck Depression Scale Scores revealed these to be skewed. Thus, the transformation $\ln (\text{Beck Depression Score} + 1)$ was carried out.

Second order partial correlations (controlling pre-treatment scores) between post-treatment rationality scores and post-treatment Beck Depression and Trait Anxiety scores were calculated. The Type I error rate was set at 0.05. Significant partial correlations for post-treatment Rationality Scale scores with post-treatment Beck Depression scores ($r (10) = -0.66$; one-tailed $p = 0.01$) and post-treatment Trait Anxiety scores ($r (8) = -0.54$; one-tailed $p =$...
0.05) were observed for the rational-emotive treatment. However, for the relaxation treatment, these partial correlations were not significant, being \( r (10) = -0.165 \) and \( r (8) = -0.29 \) (one-tailed \( p > 0.05 \)) respectively.

Thus, the results from the rational-emotive treatment were supportive of the hypothesised correlations between increases in rationality and reductions in anxiety and depression. The results from the relaxation treatment were not supportive of this hypothesis.

**Hypothesis 7.**

The significance of any difference between the treatments in their capacity to reduce scores on the Beck Depression and Trait Anxiety Inventories was evaluated by analyses of covariance on the post-treatment scores for each variable, with the respective pre-treatment scores as covariates. The Type I error rate was set at 0.05. The hypothesis that rational-emotive therapy would be associated with greater reductions in anxiety and depression than relaxation training was not supported (see Table 6-9 where the means are adjusted for the covariates; see Appendix G for the ANCOVA summary table).

**Table 6-9**

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Rational-Emotive Adj. Mean</th>
<th>Relaxation Adj. Mean</th>
<th>F-value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beck Depression *</td>
<td>1.21</td>
<td>1.64</td>
<td>2.54</td>
<td>N.S.</td>
</tr>
<tr>
<td>Trait Anxiety +</td>
<td>35.68</td>
<td>37.60</td>
<td>0.60</td>
<td>N.S.</td>
</tr>
</tbody>
</table>

*Indicates degrees of freedom = 1, 26.

+Indicates degrees of freedom = 1, 24 (two subjects in the rational-emotive group failed to complete the pre-treatment Trait Anxiety Inventory).
6.6.5. Supplementary Analyses.

Pre-Treatment Relationships Between Psychological Measures and Headache Activity.

Within the pre-treatment data (N = 36), relationships between rationality and headache activity were evaluated by correlating Rationality Scale scores with headache frequency (r (34) = -0.24), intensity (r (34) = 0.13), duration (r (34) = 0.11) and medication consumption (r (34) = -0.26). None of these correlations were significant (2-tailed p > 0.05).

Relationships Between Changes in Anxiety and Depression and Changes in Headache Activity.

In order to investigate relationships between changes in headache activity and changes in emotional distress over the course of treatment, second order partial correlations (controlling for pre-treatment scores) between post-treatment headache activity scores and post-treatment Trait Anxiety and Beck Depression scores were calculated. None of these partial correlations were significant at the Bonferroni corrected level (p > 0.0125). The partial correlations between changes in headache frequency and changes in depression and anxiety were positive and significant at the nominal level (p < 0.05) for the relaxation treatment, but no other partial correlations were significant by this criterion (see Tables 6-10 and 6-11). Thus, taking account of the inflated Type I error rate which occurs when more than a single statistical test is applied, the results can only be regarded as tentatively suggestive of a relationship between changes in anxiety and depression and changes in headache frequency for subjects receiving progressive relaxation training.
Table 6-10

Partial Correlations: Post-Treatment Trait Anxiety Scores with Post-Treatment Headache Activity Measures.

<table>
<thead>
<tr>
<th>Headache Activity</th>
<th>Rational-Emotive (N=14)</th>
<th>Relaxation (N=15)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>r</td>
<td>p-value*</td>
</tr>
<tr>
<td></td>
<td>(df=8)</td>
<td></td>
</tr>
<tr>
<td>Frequency</td>
<td>0.34</td>
<td>N.S.</td>
</tr>
<tr>
<td>Intensity</td>
<td>-0.03</td>
<td>N.S.</td>
</tr>
<tr>
<td>Duration</td>
<td>0.03</td>
<td>N.S.</td>
</tr>
<tr>
<td>Medication</td>
<td>0.14</td>
<td>N.S.</td>
</tr>
</tbody>
</table>

*Indicates a 2-tailed p-value.

Table 6-11

Partial Correlations: Post-Treatment Beck Depression Scores with Post-Treatment Headache Activity Measures.

<table>
<thead>
<tr>
<th>Headache Activity</th>
<th>Rational-Emotive (N=14)</th>
<th>Relaxation (N=15)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>r</td>
<td>p-value*</td>
</tr>
<tr>
<td></td>
<td>(df=10)</td>
<td></td>
</tr>
<tr>
<td>Frequency</td>
<td>0.18</td>
<td>N.S.</td>
</tr>
<tr>
<td>Intensity</td>
<td>0.12</td>
<td>N.S.</td>
</tr>
<tr>
<td>Duration</td>
<td>0.21</td>
<td>N.S.</td>
</tr>
<tr>
<td>Medication</td>
<td>0.14</td>
<td>N.S.</td>
</tr>
</tbody>
</table>

*Indicates a two-tailed p-value.

Relationships Between Pre-Treatment Depression Scores and Outcome on Headache Activity Measures.

High pre-treatment scores on the Beck Depression Inventory have been reported to be associated with poorer outcomes from relaxation training for tension headache (e.g., Jacob et
Thus, it was decided to investigate the predictive role of pre-treatment depression scores by calculating first order partial correlations between this measure and post-treatment and follow-up headache activity measures (controlling for pre-treatment headache activity scores). Employing two-tailed tests, all correlations fell short of both the Bonferroni corrected ($p > 0.0125$) and nominal ($p > 0.05$) significance levels (see Tables 6-12 and 6-13).

**Table 6-12**

Partial Correlations: Pre-Treatment Beck Depression Scores with Post-Treatment Headache Activity Measures.

<table>
<thead>
<tr>
<th>Headache Activity</th>
<th>Rational-Emotive (N=14) $r$ (df=11)</th>
<th>Relaxation (N=15) $r$ (df=12)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency</td>
<td>0.14</td>
<td>-0.32</td>
</tr>
<tr>
<td>Intensity</td>
<td>0.00</td>
<td>0.08</td>
</tr>
<tr>
<td>Duration</td>
<td>0.05</td>
<td>0.30</td>
</tr>
<tr>
<td>Medication</td>
<td>-0.47</td>
<td>0.37</td>
</tr>
</tbody>
</table>

**Table 6-13**

Partial Correlations: Pre-Treatment Beck Depression Scores with Follow-Up Headache Activity Measures.

<table>
<thead>
<tr>
<th>Headache Activity</th>
<th>Rational-Emotive (N=11) $r$ (df=8)</th>
<th>Relaxation (N=12) $r$ (df=9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency</td>
<td>-0.49</td>
<td>-0.06</td>
</tr>
<tr>
<td>Intensity</td>
<td>0.15</td>
<td>-0.07</td>
</tr>
<tr>
<td>Duration</td>
<td>-0.10</td>
<td>-0.46</td>
</tr>
<tr>
<td>Medication</td>
<td>-0.32</td>
<td>-0.04</td>
</tr>
</tbody>
</table>
These results indicate that there was no significant correlation between pre-treatment Beck Depression scores and outcome on the various measures of headache activity for either treatment group.

**Effect of Treatments on Anxiety and Depression.**

The effects of each treatment on Beck Depression and Trait Anxiety scores was evaluated by testing the significance of the difference between pre- and post-treatment scores by means of two-tailed t-tests. The Type I error rate was set at 0.05. Significant reductions in anxiety and depression occurred in the rational-emotive group (see Table 6-14) but not in the relaxation group (see Table 6-15). There was a trend towards a lowering of Trait Anxiety within the relaxation group but this did not reach significance.

**Table 6-14**

Mean Beck Depression and Trait Anxiety Scores for Rational-Emotive Group (N = 14).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pre-Treatment Mean</th>
<th>Post-Treatment Mean</th>
<th>Paired t-value</th>
<th>2-tailed p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beck Depression*</td>
<td>1.88 (0.96)</td>
<td>1.21 (1.09)</td>
<td>2.98</td>
<td>0.011</td>
</tr>
<tr>
<td>Trait Anxiety+</td>
<td>43.58 (10.40)</td>
<td>36.25 (6.96)</td>
<td>3.13</td>
<td>0.010</td>
</tr>
</tbody>
</table>

*Indicates degrees of freedom = 13.

+Indicates degrees of freedom = 11.
Table 6-15

Mean Beck Depression and Trait Anxiety Scores for Relaxation Group (N = 15).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pre-Treatment Mean</th>
<th>Post-Treatment Mean</th>
<th>Paired t-value</th>
<th>2-tailed p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beck</td>
<td>1.88 (0.77)</td>
<td>1.63 (0.75)</td>
<td>1.43</td>
<td>N.S.</td>
</tr>
<tr>
<td>Depression</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trait</td>
<td>41.33 (9.85)</td>
<td>37.15 (8.26)</td>
<td>1.92</td>
<td>0.08</td>
</tr>
<tr>
<td>Anxiety</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Indicates degrees of freedom = 14.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

6.7. DISCUSSION.

The results obtained in the present study were not supportive of a rational-emotive conceptualisation of the relationship between stress and recurrent headache. There was a suggestion that at the post-treatment assessment, rational-emotive therapy was superior to relaxation training in reducing headache intensity, but this difference was not maintained at the 12-week follow-up assessment. Furthermore, the result for headache intensity at post-treatment was not significant when the Bonferroni correction was applied. On all other aspects of headache activity, no differences between treatments were observed at any stage of the experiment. Contrary to expectations, reductions in headache activity occurring within the rational-emotive group were not associated with increases in rationality to any significant extent. Similar results were observed for the relaxation group.

The fact that pre-treatment measures of rationality and headache activity were not correlated significantly, whilst high negative correlations between the former variable and measures of anxiety and depression were observed suggests that, although rationality may be related to emotional distress, it may have little to do with headache. This does not augur well for a rational-emotive conceptualisation of any relationship between stressful events and headache.

The increase in rationality which occurred in the rational-emotive group fell just short of significance and was not significantly greater than the increase in the relaxation group. However, in spite of the limited effect of rational-emotive therapy on rationality, significant reductions in headache frequency and intensity were observed for this treatment at the post-
treatment and follow-up assessments. Furthermore, within the relaxation treatment, where the focus was not upon rationality, a significant reduction in headache frequency occurred, this result being maintained at the follow-up assessment. This pattern of findings does not support the notion that there exists any firm linkage between rationality and headache activity. However, it is possible that such a relationship does exist, but that the Rationality Scale was insensitive to those aspects of rationality that may be involved. For example, rational-emotive therapy may have increased the level of rational thinking in situations associated specifically with headache onset or increases in headache intensity, without such an increase being registered on the Rationality Scale. However, the fact that, in the present study, there was no significant difference between the effects of relaxation training and rational-emotive therapy on headache activity tends to argue against the view that the degree of rationality plays a significant role in the aetiology and/or maintenance of headache. Rather, it would seem that if some cognitive process did play a role in the observed treatment effects, such a process is likely to have been altered to a similar extent by both treatments. That is, some cognitive change common to both treatments might underlie the observed reductions in headache activity.

It would seem difficult to argue that the absence of significant correlations between changes in rationality and changes in headache frequency and duration over the course of rational-emotive therapy was due to the small number of subjects involved in this treatment. The respective correlations were -0.37 and -0.32. They were in the predicted direction but were not significant. However, the number of subjects in this treatment was adequate for the identification of significant correlations between rationality and depression and between rationality and trait anxiety over the course of rational-emotive therapy, as predicted by the rational-emotive model (Ellis, 1962, 1977a, b). Although a significant relationship between rationality and emotional distress was evident during rational-emotive therapy, consistent with the view that rationality may mediate improvements in emotional distress during this treatment (Ellis, 1962; 1977a, b), no such significant relationship between rationality and headache activity was found. This undermines the hypothesis that rationality plays a significant role in the aetiology or maintenance of recurrent headache.

The significant correlations observed between rationality and anxiety and depression over the course of rational-emotive treatment are consistent with the rational-emotive theory of emotional disturbance. Against the theory, however, is the absence of any such significant relationships within the relaxation treatment group. The latter observation suggests that any alterations in the level of emotional distress need not be necessarily related to alterations in rationality. However, Smith (1983) observed changes in anxiety and depression, occurring in subjects placed in waiting list control or relaxation plus supportive counselling groups, to be correlated significantly with changes in irrational beliefs as assessed by the Idea Inventory
(Kassinove et al., 1977). Smith (1983) suggested that these results could have been due to a confounding of items on the Idea Inventory with those on the measures of emotional distress. In the present study, efforts were made to avoid such confounding. This may account for the absence of significant correlations of Rationality Scale scores with scores on the Beck Depression or Trait Anxiety Inventories over the course of relaxation training.

Within the relaxation treatment, reductions in headache frequency were associated with reductions in anxiety and depression. These findings are difficult to interpret, given that no such relationships were observed within the rational-emotive group. However, the results could indicate that the capacity of relaxation training to reduce headache frequency may be limited by the extent to which this treatment generates alterations in emotional distress. Such a suggestion must be considered very speculative until further research is conducted.

Unlike Jacob et al (1983), no significant correlation between reductions in headache activity and pre-treatment Beck Depression scores was noted for the subjects receiving relaxation training. Unfortunately, Jacob et al (1983) provided little in the way of detail concerning their progressive relaxation training programme. In the present study, subjects receiving relaxation training were encouraged to apply their newly acquired skills in specific situations and to thereby employ the technique as a coping skill (Goldfried and Trier, 1974). This active approach to relaxation training may have challenged depressed subjects' view of themselves as helpless in the face of their headaches to a greater extent than the procedures of Jacob et al (1983), and this may have weakened the predictive power of pre-treatment Beck Depression scores.

Unlike the rational-emotive treatment described in the present study, which focused specifically on rationality, previous cognitive treatment studies have employed a range of techniques including imagery, coping self-statements, and cognitive re-appraisal, as well as emphasising the alteration of irrational beliefs (e.g., Holroyd and Andrasik, 1978; Holroyd et al., 1977). Thus, it is possible that the treatment effects reported for cognitive therapy could stem from the acquisition of specific cognitive coping skills or from the restructuring of cognitive appraisal processes other than rationality.

Holroyd and Andrasik (1978) required subjects to record their thoughts in situations associated with emotional distress or headache. These subjects were then assigned either to a treatment designed to teach specific relaxation and cognitive coping skills or to a headache discussion group, which focused on the historical roots of their headaches and did not include the presentation of any coping skills for dealing with stress or headache. Relative to a control group, who simply recorded their headaches but not their cognitive responses, subjects in the two experimental conditions demonstrated significant reductions in headache frequency,
intensity and duration. These effects were maintained at a 6-week follow-up assessment. There were no significant differences between the two experimental conditions on any measure of headache activity. Holroyd and Andrasik (1978) concluded that it may be the self-monitoring of cognitive responses which is the essential therapeutic ingredient in cognitive therapy for headache. They noted, however, that all but one of the subjects in the headache discussion group developed their own cognitive strategies (e.g., imagery or praying) for dealing with stress and headache. Holroyd and Andrasik (1978) suggested that providing subjects with a causal account of their headaches may have led them to believe that they possess the ability to manage their headaches themselves.

In the present study the treatments emphasised that subjects had the potential to master their headaches. A number of authors have argued that the provision of a rationale for the origin and treatment of clients' difficulties contributes to their sense of mastery over the problem, and is an essential feature of any effective psychotherapy (e.g., Bandura, 1977; Frank, 1961; Murray and Jacobson, 1978). For example, Frank (1961, pp. 328-330) has pointed out that:

"The therapeutic rationale, finally, enables the patient to make sense of his symptoms. Since he often views them as inexplicable, which increases their ominousness, being able to name and explain them in terms of an over-reaching conceptual scheme is in itself powerfully reassuring. The first step in gaining control of any phenomenon is to give it a name... all successful therapies implicitly or explicitly change the patient's image of himself as a person who is overwhelmed by his symptoms and problems to that of one who can master them... as a result, he becomes able to tackle the problems he had been avoiding and to experiment with new, better ways of handling them".

Bandura (1977) has referred to the degree of mastery that a person believes he or she has over a particular problem or situation as self-efficacy. Increases in self-efficacy have been found to be associated with reductions in phobic symptoms (Bandura, 1977, 1982) increased tolerance for experimental pain (Dolce, Doleys et al., 1986; Vallis and Bucher, 1986), reduced ratings of arthritic pain severity (Shoor and Holman, 1984) and with better outcomes from chronic pain management programmes (Dolce, Crocker and Doleys, 1986; Dolce, Crocker, Moletteire and Doleys, 1986; Kores, Murphy, Rosenthal, Ellias and Rosenthal, 1985). Furthermore, there is some evidence to suggest that perceived self-efficacy to withstand pain may be related to the functioning of the endogenous pain inhibitory system (Bandura, O'Leary, Taylor, Gauthier and Gossard, 1987) which has been implicated in the aetiology of headache attacks (e.g., Lance, 1982; Sicuteri, 1982).

Thus, with respect to the present study, outcomes from both rational-emotive therapy and relaxation training could have been mediated by increases in self-efficacy to reduce emotional distress and headache in stressful situations, rather than by increases in rationality.
Bandura (1977, 1982) has postulated that the level of self-efficacy is an important determinant of the extent to which individuals will persist in their efforts to cope with stressful situations. Appraisal processes related to self-efficacy, such as the extent to which the individual believes that he or she can control a given situation, have been observed to be related to coping behaviour (e.g., Folkman and Lazarus, 1980; Folkman, Lazarus, Dunkel-Schetter, De Longis and Gruen, 1986; Parkes, 1984). Both appraisal and coping have been found to be related to emotional responses (e.g., Aldwin and Revenson, 1987; Folkman and Lazarus, 1986, 1988). Thus, an examination of these concepts may shed light on the nature of the relationship between stress and headache.

In the next chapter, the concepts of self-efficacy, appraisal and coping are elaborated. A conceptual model, proposing that these constructs operate as intervening variables, mediating the relationship between stressful events and headache, is developed and subjected to empirical examination.

6.8. SUMMARY.

Study I was concerned with evaluating the role of rationality, defined as the acceptance of oneself, others and the environment, in the aetiology and / or maintenance of headache. Following the rational-emotive model of human distress (Ellis, 1962, 1977a, b), wherein the magnitude of the emotional response to stressors is considered to be a function of the extent to which the individual's belief system is irrational, increases in rationality were postulated to mediate outcome from rational-emotive and progressive relaxation treatments for headache. The results obtained did not support this hypothesis. Rational-emotive therapy was no more effective in producing lasting improvements in headache frequency, duration, intensity or medication intake than progressive relaxation training. Furthermore, within each treatment, changes in headache activity were not correlated significantly with alterations in rationality. It was concluded that rationality may not make a significant contribution to headache attacks.

In the rational-emotive treatment, significant reductions in headache frequency and intensity were observed to occur and to be maintained at the 12-week follow-up assessment. In the progressive relaxation treatment significant reductions in headache frequency only, were observed at the post-treatment and 12-week follow-up assessments. It was suggested that these reductions in headache activity may have been mediated by increases in self-efficacy (Bandura, 1977, 1982, 1986) which may have followed from the acquisition of skills for the management of stressful events and headache.
CHAPTER 7

STUDY II

RELATIONSHIPS BETWEEN SELF-EFFICACY, STRESS, APPRAISAL, COPING AND HEADACHE: A LONGITUDINAL STUDY.

7.1. INTRODUCTION.

7.1.1. The Concept of Self-Efficacy.

The term self-efficacy, as defined by Bandura (1977, 1986), has been discussed in previous chapters of this thesis. The purpose of the present section is to attempt some clarification of the concept by raising the question of the tenability of the distinction between efficacy expectations and outcome expectations (Eastman and Marzillier, 1984; Marzillier and Eastman, 1984).

Bandura (1977, p. 193) defines an outcome expectation as "...a person's estimate that a given behaviour will lead to certain outcomes", and an efficacy expectation as "...the conviction that one can successfully execute the behaviour required to produce the outcomes." He is very concerned to maintain this distinction, so as to argue that his concept of self-efficacy is distinct from Rotter's (1966) concept of locus of control. This latter concept was introduced into psychology in an effort to distinguish persons who perceive reinforcement as contingent upon their own actions (internal locus of control) from those who believe that such outcomes occur independently of their own actions, being determined by factors outside their own scope of influence (external locus of control) (Rotter, 1966).

Bandura (1977, p. 204) states that locus of control refers to beliefs about "action-outcome contingencies", and that what is being defined is an outcome expectation. He argues that the concept cannot be regarded as synonymous with self-efficacy because "...people who regard outcomes as personally determined, but who lack the requisite skills would experience low self-efficacy" (Bandura, 1977, p. 204). That is, they would perceive themselves as lacking the capability to influence their situation. However, it would seem equally true that persons
who believe that they possess the skills to execute a particular behaviour, but who also believe
that the execution of that behaviour pattern would have no effect on their environment, would
similarly perceive themselves as incapable of influencing their situation, and could thus be
regarded as being low in self-efficacy. In both scenarios the person would experience low self-
efficacy, albeit for different reasons.

It would appear that far from being independent of self-efficacy, expectations about
outcome are crucial determinants of the level of self-efficacy. However, this hardly implies
that self-efficacy is a redundant concept. Rather, self-efficacy, as defined by Bandura (1977),
is a function of both the extent to which outcomes are perceived to be personally determined
and the extent to which it is believed that one is in possession of the skills to effect the
outcome. Problems arise for Bandura (1977) when he attempts to speak of outcome
expectations as independent of perceived capabilities.

As Teasdale (1978, pp. 211-212) has pointed out:

"By including the words "successfully" and "required to produce the outcomes"
in his definition of efficacy expectations, Bandura combines belief about ability
to make a response with expectations concerning the outcome of the response..."

Nevertheless, Bandura (1978) has persisted with his argument that efficacy and outcome
expectations are conceptually distinct, but his position is not defensible. He has argued that:

"...the expectation that one can jump 6 feet is an efficacy judgement; the social
recognition, applause, trophies and self-satisfactions anticipated for a
performance constitute outcome judgements" (Bandura, 1978, p. 240).

However, this distinction between outcome expectations and efficacy expectations is
entirely arbitrary and question-begging. As Eastman and Marzillier (1984, p. 220) point out,
"When is an outcome part of a behaviour pattern and when is it an outcome in the Bandura
sense?"

Jumping 6 feet could be regarded as an outcome expectation following from the efficacy
expectation that one can approach the jump at the correct speed. Approaching the jump at the
correct speed, could be conceived as an outcome expectation following from the efficacy
expectation that one can execute the appropriate muscle movements. However, this constitutes
an infinite regress, wherein, any behaviour can be labelled an outcome expectation, with its
antecedents labelled efficacy expectations. As Wolpe (1978) has pointed out, on this logic,
efficacy expectations reduce quickly to judgements regarding our capability to perform the
motor components of an act, thereby trivialising the concept. Although Bandura (1978, p. 241)
insists that "...one must not confuse efficacy judgements with trivial skeletal movements", it is difficult to appreciate how this can be achieved when he insists that efficacy and outcome expectations are conceptually distinct.

It would appear impossible to conceive of an appraisal of one's capacity to perform a given task which does not take account of outcome expectations. The confidence that one has in one's capacity to reach a certain level of task performance surely requires that one be cognisant of that level of performance for which one is striving (i.e., the outcome). The efficacy expectation requires the anticipation of task completion and, therefore, involves an outcome expectation. Efficacy expectations cannot be formulated without reference to outcome and it is, therefore, absurd to speak of an efficacy judgement as independent of any consideration of outcome.

Thus, for the purposes of Study II, which is concerned with an exploration of the relationship between self-efficacy and recurrent headache, Bandura's definition of self-efficacy as "the conviction that one can successfully execute the behaviour required to produce the outcome" (Bandura, 1977, p. 193) will be accepted. However, outcome expectations will be regarded as essential features of self-efficacy (Eastman and Marzillier, 1984; Kirsch, 1985; Marzillier and Eastman, 1984).

7.1.2. Self-Efficacy and Headache.

In order to study relationships between self-efficacy and headache it is necessary to specify those kinds of efficacy expectations that are likely to have implications for the disorder. The evidence reviewed in Chapter 4 suggests that self-control procedures, such as relaxation training, biofeedback and cognitive therapy, do occasion improvements in recurrent headache. If, as was suggested in the last chapter, these procedures produce their effects by altering efficacy expectations, the focus of this increase in self-efficacy may be upon headache sufferers' perceived capability to exercise self-control responses in the face of stressful events and headache. The self-control therapies have emphasised control over cognitive, behavioural and affective responses by means of cognitive therapy (e.g., Figueroa, 1982; Holroyd and Andrasik, 1978), relaxation training (e.g., Chesney and Shelton, 1976) or biofeedback (e.g., Budzynski et al., 1973; Daly et al., 1983). Such control may mitigate against the occurrence of headache in the face of stressful events. The term self-control-efficacy shall be employed to refer to the individual's perceived capacity to exercise self-control over cognitive, behavioural and affective responses.
The relationship between life events and emotional and physical distress has been the subject of much research over the last twenty years. However, as a number of authors have pointed out, the strength of the relationship between stressful events and measures of psychological or somatic functioning has tended to be weak, with stressful events typically accounting for less than 10% of the variance in measures of emotional and physical distress (e.g., Andrews and Tennant, 1978; Elliott and Eisdorfer, 1982; Nezu and Ronan, 1985; Rabkin and Struening, 1976). It has been shown that many people do not develop psychological or physical symptoms despite exposure to stressful events (Kobasa, 1979). Furthermore, from a theoretical perspective, the identification of a relationship between life events and psychological and physical health does not, in itself, shed light on the psychological processes that may mediate such a link. These concerns have led a number of researchers to hypothesise that the impact of stressful events on the individual's well-being may be buffered or moderated by psychological and / or social resources (e.g., Kobasa, Maddi and Kahn, 1982; Pearlin and Schooler, 1978; Wethington and Kessler, 1986).

Johnson and Sarason (1978) found that the correlation of the severity of undesirable life events with depression and anxiety was greater for subjects with an external locus of control than for those with an internal locus of control. Employing a multiple regression analysis, Krause (1985) observed an interaction between locus of control and the frequency of undesirable life events in the prediction of depression. Once again, subjects with an external locus of control demonstrated strong relationships between the frequency of undesirable events and the severity of depression. Thus, it was concluded in each of these studies that, locus of control buffers or moderates the strength of the relationship between stressful events and well-being (Johnson and Sarason, 1978; Krause, 1985).

Aspects of personality such as hardiness (Kobasa, 1979; Kobasa et al., 1982) and mastery (Pearlin and Schooler, 1978) have also been implicated in the buffering process.

Hardiness has been conceptualised as a multidimensional construct comprising commitment to one's goals, the belief that outcomes are within one's scope of influence, and the tendency to appraise change as a challenge rather than as a threat (Kobasa et al., 1982).

The concept of mastery has been defined in terms very similar to those used to characterise an internal locus of control:

"Mastery ... concerns the extent to which one regards one's life-changes as being under one's own control in contrast to being fatalistically ruled" (Pearlin and Schooler, 1978, p. 114).
Given that stressful events are widely regarded as the most common precipitants of headache attacks (e.g., Friedman et al., 1954; Selby and Lance, 1960; Henryk-Gutt and Rees, 1973), and given also that self-control therapies have been found to reduce headache activity (e.g., Budzynski et al., 1973; Cox et al., 1975; Figueroa, 1982), self-control-efficacy may moderate the strength of any relationship between the frequency of stressful events and the frequency of headache. This chapter is concerned with an investigation of this hypothesis. The instrument developed to assess self-control-efficacy is described in a later section.

It has been suggested that the relationship between psychological resources (such as mastery or locus of control) and well-being may be mediated by the processes of appraisal and coping (Folkman, Lazarus, Gruen and De Longis, 1986; Parkes, 1984). The following section is concerned with these mediational processes.

7.1.3. Appraisal and Coping.

According to Lazarus and his colleagues (Folkman, Lazarus, Dunkel-Schetter, De Longis and Gruen, 1986; Lazarus and Folkman, 1984a, b), it is the way in which an individual appraises a situation that determines coping actions, with both appraisal and coping contributing to subsequent well-being. The distinction between the concepts is not always clear, for in a number of instances the process of re-appraisal is termed a coping response (Lazarus and Folkman, 1984a, b). Furthermore, the relations between the theoretical constructs may be bidirectional with, for example, appraisal influencing coping and subsequent coping influencing appraisal (Lazarus and Folkman, 1984a, b).

Lazarus and Folkman (1984a, b) have distinguished between primary and secondary appraisals. A primary appraisal is an evaluation of the degree of threat that a situation poses to the individual's well-being. A secondary appraisal refers to the individual's evaluation of his or her capacity to manage or obviate the threat.

Folkman and Lazarus (1980, p. 223) defined coping as:

"...the cognitive and behavioural efforts made to master, tolerate or reduce external and internal demands and conflict among them."

They distinguished between problem-focused coping (efforts to modify the source of stress) and emotion-focused coping (efforts to regulate one's emotional response to the stressor). Folkman and Lazarus (1980) developed the Ways of Coping Scale to assess these two broad categories of coping and found that, when individuals appraised situations as amenable to change, they were most likely to engage in problem-focused coping. When they
appraised situations as ones which could not be influenced, they tended to employ emotion-focused coping. These findings are consistent with the view that appraisal influences coping actions (Folkman and Lazarus, 1980; Lazarus and Folkman, 1984a, b).

Other researchers have been concerned with describing relationships between particular styles of coping and psychological and physical well-being. In these studies, subjects are typically asked to describe a stressful event and then to complete a questionnaire detailing the manner in which they coped with the event (e.g., Aldwin and Revenson, 1987; Billings and Moos, 1984; Holm et al., 1986).

Researchers in the field of coping do not adhere to any accepted classification of coping strategies. Instead, they select from a wide range of strategies, and design assessment scales for each on a rational basis. Some of the most commonly assessed coping strategies are listed by Moos and Billings (1982). These include cognitive avoidance (trying to forget about the problem or engaging in wishful fantasies), problem solving action (this is sometimes referred to as direct coping and involves efforts to deal directly with the situation), affective regulation (efforts to control one's emotional response to the situation), and emotional discharge (venting feelings of frustration or anger on oneself or others). Behavioural avoidance of the situation has also been assessed (e.g., Holahan and Moos, 1986; Holm et al., 1986), with these researchers combining this measure with cognitive avoidance in the construction of a single avoidance scale.

Avoidance and emotional discharge coping strategies have been associated with depressed mood and more psychological symptoms (e.g., Aldwin and Revenson, 1987; Billings and Moos, 1984; Cronkite and Moos, 1984), whilst tendencies to employ low levels of avoidance coping, high levels of direct coping and high levels of affective regulation have been associated with lower levels of depression and fewer psychological symptoms (Billings and Moos, 1981, 1984; Holahan and Moos, 1985, 1986).

Felton and Revenson (1984) studied patients suffering from hypertension, diabetes, arthritis or cancer. Those patients who employed an active coping strategy in the face of their illness (information seeking), demonstrated better psychological adjustments (as assessed on sickness impact scales developed by the authors) than those relying on an avoidance strategy (wish-fulfilling fantasy). Similarly, Manne and Sandler (1984) found that persons with genital herpes demonstrated lower self-esteem, poorer sexual adjustment and higher levels of depression when they coped with their disease by means of wishful thinking (cognitive avoidance) and characterological self-blame (emotional discharge).
In a study of coping amongst tension headache sufferers, Holm et al (1986) observed these subjects to employ more avoidance and self-blame coping than headache-free control subjects. Furthermore, Philips and Jahanshahi (1986) found that avoidance behaviour accounted for 60.5% of the variance in the pain behaviour of chronic headache sufferers. Philips (1987a) suggested that this avoidance behaviour may be maintained by the belief that withdrawal from activity reduces the severity of pain. That is, a low level of self-efficacy for engaging in activities without triggering or aggravating pain, may foster avoidance behaviour (Philips, 1987a).

Although it has only been in recent years that psychologists have attempted to study relationships between coping and psychological adjustment in a systematic fashion, the evidence appears to indicate that coping strategies involving avoidance or emotional discharge may be associated with poorer adjustments, whilst those involving direct action or affective regulation may be associated with better adjustments. The question of whether there exists any causal relationship between coping and adjustment requires the conduct of prospective studies with repeated observations of coping and emotional distress.

Folkman, Lazarus, Gruen et al (1986) hypothesised that mastery is related to psychological and somatic symptoms through the processes of appraisal and coping. Subjects' primary and secondary appraisals, as well as their coping responses, were assessed by requiring them to describe their responses to five stressful situations. The number of variables assessed (seven primary appraisals, four secondary appraisals and eight coping strategies) makes it difficult to interpret their results. However, multiple regression analyses revealed that secondary appraisals did not account for any significant proportion of the variance in psychological or somatic symptoms. The primary appraisal and coping variables did account for a significant proportion of the variance in psychological symptoms, but did not explain the variance in somatic symptoms to any significant extent. Although high mastery scores were associated with fewer psychological and somatic symptoms, mastery was not correlated consistently with measures of appraisal or coping. Thus, as Folkman, Lazarus, Gruen et al (1986) point out, these results leave unclear the processes by which mastery might influence psychological and somatic functioning.

Parkes (1984) was able to identify inter-relationships between locus of control, appraisal and coping. Locus of control was weakly correlated with direct coping. Subjects with an internal locus of control tended to appraise situations as less important than those with an external locus control. In situations appraised as amenable to change, subjects with an internal locus of control reported high levels of direct coping and low levels of suppression (a type of coping involving efforts to ignore the situation and inhibit action), whilst those with an external locus of control reported the converse pattern of coping. The results of this study
suggest that the moderating effect of locus of control on well-being (e.g., Johnson and Sarason, 1978; Krause, 1985) may be mediated by appraisal and coping processes.

This chapter is concerned with the development and examination of a model of the relationship between stressful events and headache. It is proposed that this relationship is moderated by self-control-efficacy and that this moderating function is mediated by the processes of appraisal and coping. Such a model is detailed in the following section.

7.1.4. Theoretical Model of Relationships Between Stressful Events and Headache.

Although there is little evidence to support a role for major life events in the aetiology of headache (e.g., Andrasik, Blanchard, Arena, Teders et al., 1982; Blanchard et al., 1986; Holm et al., 1986), there is some evidence suggesting that, less severe, more frequent stressful events, may contribute to headache attacks (e.g., Holm et al., 1986; Levor et al., 1986). Since headache afflicts a large proportion of the community and is often an episodic complaint, occurring several times per week in many sufferers, it follows that, if the onset of discrete attacks is precipitated by stressful events, these events must be of a fairly common nature similar to those listed on the Daily Hassles Scale (Kanner et al., 1981). Indeed, it was on this scale that Holm et al (1986) observed tension headache sufferers to report more stressful events than headache-free control subjects.

It is postulated that frequent stressful events precede headache onset, but that the strength of the relationship between these variables is moderated by the level of self-control-efficacy (defined as the individual's perceived capacity to self-regulate cognitive, behavioural and affective responses). It is proposed that persons with a high level of self-control-efficacy will appraise stressful events as less threatening and as more amenable to change than persons with low levels of self-control-efficacy. Persons with high levels of self-control-efficacy are recognised as those who are self-efficacious with respect to control over their cognitive, behavioural and emotional responses to stressors and are, therefore, considered to have a high degree of confidence in their capacity to meet the demands of stressful encounters.

Lazarus and his colleagues (Lazarus, 1977; Lazarus and Folkman, 1984a, b) have proposed that the more individuals believe they have at stake in an encounter, and the less they appraise the impact of an encounter as amenable to change, the more emotional distress they will experience in the face of that encounter. If these appraisal processes do operate to increase the strength of the emotional response to stressors and if the strength of this response plays a role in the onset of headache attacks (e.g., Friedman et al., 1954; Henryk-Gutt and Rees,
1973), it follows that appraising stressful events as highly important (primary appraisal) and as not amenable to change (secondary appraisal), will be associated with headache onset. Similarly, it follows that the stronger the emotional response to the stressful event, the greater the likelihood of the person suffering a headache.

A high level of self-control-efficacy is expected to foster higher levels of affective regulation coping in stressful encounters. This follows from the conceptualisation of self-control-efficacy as involving control over cognitive and emotional responses, each of which may contribute to affective regulation coping (Moos and Billings, 1982).

As discussed previously, affective regulation skills are taught in the successful application of self-control therapies to headache (e.g., Chesney and Shelton, 1976; Cox et al., 1975; Figueroa, 1982). Furthermore, affective regulation coping in response to stressors has been associated with lower levels of depression (Billings and Moos, 1984). Thus, it is proposed that high levels of affective regulation will mitigate against the occurrence of headache attacks in the face of stressful events.

Appraising events as amenable to change has been associated with active coping methods directed towards the modification of the stressor (direct coping) (Folkman and Lazarus, 1980; Parkes, 1984). Conversely, stressful events that are not appraised as amenable to change have been observed to be associated with an increased use of avoidance coping strategies (Folkman, Lazarus, Dunkel-Schetter et al., 1986). Direct coping has been associated with fewer psychological symptoms (Aldwin and Revenson, 1987) and lower levels of depression (Billings and Moos, 1984), whilst avoidance coping has been associated with higher frequencies of psychological and physical symptoms (Cronkite and Moos, 1984; Aldwin and Revenson, 1987; Billings and Moos, 1981; Holahan and Moos, 1985, 1986). Furthermore, the frequency of avoidance coping has been found to be greater amongst tension headache subjects than headache-free controls (Holm et al., 1986). Accordingly, it is proposed that direct coping, in the face of stressful events, will mitigate against the occurrence of headache whilst avoidance coping will increase the probability of headache occurring.

To summarize the theoretical model, it is proposed that the action of stressful events upon headache frequency is moderated by the level of self-control-efficacy, this moderating action being mediated by appraisal, coping and emotional response variables. Lower levels of self-control-efficacy will lead persons to appraise stressful events as less amenable to change and as more threatening. These appraisals will increase the strength of the aversive emotional response to the stressor, thereby increasing the probability of headache occurring. Lower levels of self-control-efficacy will be associated with lower levels of affective regulation coping which will, in turn, increase the probability of headache onset in the face of stressful events.
Appraising stressful events as amenable to change will foster direct coping which will mitigate against the onset of headache. Appraising stressful events as not amenable to change will increase the frequency of avoidance coping which will in turn increase the probability of headache developing in the face of stressful events.

7.2. LONGITUDINAL DESIGN.

The present study was concerned with an empirical examination of the model proposed in the previous section. In order to study the temporal relationships between stressful events, appraisal, coping, emotional upset and headache onset, a daily self-observation methodology was chosen, similar to that developed by Levor et al (1986).

Lever et al (1986) required migraine sufferers to record their headaches, stressful events and emotional states over a four week period. For the purposes of examining relationships between these variables, this group of researchers identified migraine and migraine-free cycles. A migraine cycle was defined as three migraine-free days followed by a day on which migraine occurred. A migraine-free cycle was defined as four consecutive migraine-free days. Non-migrainous headache days were counted as migraine-free days.

Lever et al (1986) chose cycles of four days on the basis of the observed vasomotor instability of migraine sufferers said to be evident as much as 72 hours prior to the actual attack (Tunis and Wolff, 1953). However, this is a rather weak rationale for the determination of cycle length. Firstly, Tunis and Wolff’s (1953) observations were based on a sample of only ten migraine sufferers who were selected on the basis of previous observations indicating the occurrence of vascular changes during headache. Secondly, the evidence on the pathophysiology of migraine, reviewed in Chapter 2, indicates that, although vascular changes do occur during some migraine episodes, these alterations are not necessary to the occurrence of many attacks (e.g., Drummond and Lance, 1983; Thompson and Adams, 1984).

For many of their subjects, Lever et al (1986) were unable to identify a single migraine and migraine-free cycle, necessary for their repeated measures analysis of variance. Although they started with 33 migraineurs, only 14 subjects recorded patterns of headache activity wherein at least one of each migraine and migraine-free cycle could be identified. No cycles could be identified for subjects reporting frequent migraine attacks. Lever et al (1986) observed that a greater number of stressful events were recorded in the migraine than in the migraine-free cycles, and that there was a trend (p = 0.06) towards greater emotional arousal during the migraine cycles.
The methodology of Levor et al (1986) promises to shed more light on the relationship between stressful events and headache than studies which have simply relied on headache sufferers' retrospective reports (e.g., Henryk-Gutt and Rees, 1973; Holm et al., 1986). Accordingly, it was decided to adapt this methodology, which allows for the study of the temporal relationship between stressful events and headache, to the present research problem.

The main difficulty with the methodology described by Levor et al (1986) is their choice of four day cycles. This resulted in a considerable loss of data. Furthermore, as discussed previously, their rationale for the selection cycle lengths is suspect.

The present study was concerned with the spectrum of headache ranging from tension headache sufferers through to migraineurs, as specified in the continuum model (Bakal and Kaganov, 1979; Drummond and Lance, 1984). It would be impossible to identify headache and headache-free cycles of four days in length if subjects were experiencing more than one or two headaches per week. Thus, the present study aimed to employ shorter cycles, so as to permit the study of a wider range of headache activity patterns, and to minimise the loss of data that invariably occurs when cycles are selected from records of headache activity.

There is very little data or theory to guide the selection of cycle length. However, there have been a number of recent studies suggesting that there may be same-day and one-day lagged relationships between the occurrence of stressful events and the report of physical symptoms (e.g., De Longis, Folkman and Lazarus, 1988; Stone, Jandore and Neale, 1986; Verbrugge, 1985). Each of these studies employed a daily self-observation methodology and assessed physical symptoms by means of The Daily Health Record (Verbrugge, 1985) which assesses such symptoms as flu, sore throat, backache and headache. Thus, on the basis of this evidence, it was decided to employ cycles of two days in length. Accordingly, a headache cycle was defined as a headache-free day followed by a day with headache; a headache-free cycle was defined as two consecutive headache-free days.

It was decided to alter further the methodology of Levor et al (1986) by requiring subjects to indicate the time at which stressful events occurred so that, within any given day for which a headache was present, it would be possible to establish whether the event occurred before, during or after the headache. This represents an advance over previous self-observation methodologies which have not noted the time of onset of symptoms, or the time at which stressful events occurred, thus making it impossible to determine the temporal relationship between stressful events and symptoms when these two classes of phenomena occur on the same day (e.g., De Longis et al., 1988; Levor et al., 1986; Stone et al., 1986; Verbrugge, 1985).
The proposed refinement of previous methodologies implies that stressful events occurring on the headache day of the headache cycle, will be counted within that cycle (and included in between-cycles analyses) only if they precede headache onset. Since the headache will occupy a period of time on the headache day of the headache cycle, there is necessarily a reduction in the time available, within that cycle, for a stressful event to occur and be coded as preceding the headache. Thus, when all events are coded, the headache cycles will be biased towards the inclusion of fewer stressful events than the headache-free cycles. However, the direction of this bias runs counter to the hypothesis that a greater number of stressful events will occur in the headache cycles (prior to headache onset) than in the headache-free cycles. Accordingly it was considered unnecessary to attempt to exert any control over this biasing factor.

Although the studies cited above allowed subjects to record a number of stressful events on each day, in the present study it was decided to confine subjects to one stressful event per day (the most distressing). This step was taken in view of the need to obtain data on subjects' appraisals, coping strategies, and emotional response to the event. It was considered that requiring subjects to complete the questionnaire items on these variables more than once per day might over burden them and reduce compliance.

### 7.3. HYPOTHESES.

The hypotheses derived from the theoretical model outlined in section 7.1.4, and formulated prior to the collection of data were as follows:

1. There will be a significant positive correlation between the frequency of stressful events and the frequency of headache.

2. The relationship between the frequency of stressful events and the frequency of headache will be moderated by the level of self-control-efficacy such that, the correlation between the frequency of stressful events and the frequency of headache will be greater for subjects low in self-control-efficacy than for those high in self-control-efficacy.

3. Subjects with higher self-control-efficacy will engage in higher levels of affective regulation in response to stressful events than those lower in self-control-efficacy.
4. Subjects with higher self-control-efficacy will appraise stressful events as less threatening and more amenable to change than subjects lower in self-control-efficacy.

5. When events are appraised as more amenable to change, subjects will engage in more direct coping and less avoidance coping than when events are appraised as less amenable to change.

6. Events appraised as less threatening or as more amenable to change will be associated with lower levels of emotional upset.

7. Lower levels of direct coping, lower levels of affective regulation and higher levels of avoidance coping will be associated with higher levels of emotional upset in response to stressful events.

8. A greater number of stressful events will be reported in headache cycles (prior to headache onset) than in headache-free cycles.

9. Stressful events occurring within headache cycles (prior to headache onset) will be appraised as more threatening and as less amenable to change than those occurring within headache-free cycles.

10. In response to stressful events occurring within headache cycles (prior to headache onset) subjects will report lower levels of affective regulation, lower levels of direct coping and higher levels of avoidance coping than they will in response to stressful events occurring within headache-free cycles.

11. In response to stressful events occurring within headache cycles (prior to headache onset) subjects will report higher levels of emotional upset than they will in response to stressful events occurring within headache-free cycles.
7.4. ASSESSMENT OF THEORETICAL CONSTRUCTS.

7.4.1. Stressful Events.

As mentioned previously in this chapter, the present research is concerned with the assessment of relatively minor stressful events which are commonly termed hassles (Kanner et al., 1981; Lazarus, 1984b).

It was noted in Chapter 3 that cognitive conceptualisations of stress (e.g., Folkman, Lazarus, Dunkel-Schetter et al., 1986, Lazarus, 1966, Lazarus and Folkman, 1984a, b) conceive of stress as a relation between the individual and the environment, mediated by cognitive appraisal processes. This raises the problem of defining a stressful event independently of cognitive appraisal. Clearly, once cognitive appraisal processes are recognised as fundamental components of stress, it is not possible to speak of these processes as being fully independent of the events upon which they are focused. The confounding of stressful events with appraisal cannot be eliminated completely. For example, the most common approach to the assessment of stressful events is to present subjects with lists of events pre-judged by researchers to be associated with some degree of emotional distress. Subjects are asked to select from these lists those events which have occurred over the last year (Holmes and Rahe, 1967), past week (Kanner et al., 1981) or preceding few hours (Stone and Neale, 1982). Such tasks require that subjects remember stressful events. The strength of the memory trace would be a function of the personal significance attached to any event at the time it occurred and, thus, responses to such questionnaires are necessarily confounded with appraisal processes. The degree of confounding is increased further by requiring subjects to rate the severity of any stressful event they endorse on the questionnaire and deriving an index of the severity of stressful events from these values (e.g., Kanner et al., 1981; Stone and Neale, 1982).

The method of asking subjects to report the occurrence of stressful events from the ground of their own experience (e.g., Clark and Watson 1988, Folkman and Lazarus, 1980) involves cognitive appraisal insofar as subjects can only select events on the basis of the extent to which they perceived those events as stressful. This method, however, does have the advantage of allowing subjects greater flexibility in their reporting of stressful events. For the purposes of recording daily stressful events it is far less cumbersome than Stone and Neale's (1982) method which requires subjects to select from a list of 80 stressful events.

Levor et al (1986) employed a combination of the above procedures by requiring subjects to select from a list of "common stressors" such as an "encounter with family or
friend" and "time pressure", supplemented with a list of items which the subject selected from the Hassles Scale as being "personally upsetting". However, it can hardly be assumed that such a combination of items would be representative of all the various types of stressful events that an individual might experience in the four week period over which the study of Levor et al (1986) was conducted.

If theoretical formulations of stress are to attach central roles to concepts such as cognitive appraisal, some confounding of stressful events with appraisal must be tolerated. In the present research it was decided to minimise this confounding by allowing subjects, at the end of each day, to decide whether or not any irritating or upsetting event had occurred during the course of that day, but to avoid the practice of requiring subjects to rate the severity of those events. Thus, a diary approach to assessment was employed with subjects indicating whether or not an irritating or upsetting event had occurred. If more than one event occurred subjects reported the one that was most upsetting.

7.4.2. Self-Control-Efficacy.

As discussed in Section 7.1.2, for the purposes of the present study, self-control-efficacy was conceptualised as the individuals' perceived capacity to regulate his or her behavioural, cognitive and affective responses. In order to assess this construct, a nine item scale (three items pertaining to each of the response categories listed above) was developed. Six of the items were drawn from Rosenbaum's (1980) Self-Control Scale, with three additional items being written by the author. Each item required subjects to indicate the extent to which they agreed or disagreed with the item on a 4-point scale. The Self-Control-Efficacy Scale is presented in Appendix H.

Rosenbaum's (1980) Self-Control Schedule was not employed as the measure of self-control-efficacy because it contains many items which refer to coping strategies. Since the present study was concerned with examining relationships between self-control-efficacy and coping strategies, the use of the Self-Control Schedule would have resulted in a confounding of these two variables. Indeed, the term "self-control-efficacy" was preferred to the term "self-control", for the latter is commonly defined in terms of the individual's propensity to employ coping strategies (e.g., Rosenbaum, 1980; Thoresen and Mahoney, 1974), rather than in terms of the individual's judged capacity to control his/her responses.
7.4.3. Appraisal.

Primary and secondary appraisals of stressful events, as defined by Lazarus and his co-workers (Lazarus, 1966; Lazarus and Folkman, 1984a, b), and as discussed earlier in this chapter, were assessed by requiring subjects to indicate the extent to which the stressful event mattered to them at the time (primary appraisal) and the extent to which they believed that they could change this event (secondary appraisal). Similar measures of these concepts were employed by Parkes (1984). Each of these judgements was made on a 4-point scale. The two rating scales were included in the Self-Monitoring Diary presented in Appendix I.

7.4.4. Coping.

The dimensions of event coping employed in the present study included direct coping, affective regulation and avoidance coping. In constructing scales to assess these dimensions of coping, the following definitions, derived from the stress and coping literature reviewed earlier, served as guides:

**Direct Coping:** Any action or thought focused upon confronting or resolving the problem. The items comprising this scale were as follows:

- Doubled my efforts and tried harder to make things work out.
- Talked to someone who could do something about the problem.
- Tried to think of a number of ways to sort out the problem.
- Tried to find out more about the situation.
- Changed something to try and make things work out.
- Went over in my mind what I would say or do.

**Affective Regulation:** Cognitive or behavioural attempts to self-regulate the intensity of the emotional response to the situation. This scale consisted of the following items:

- Tried to relax myself.
Reassured myself that things would work out all right.

Tried to view the situation as a challenge.

Reminded myself how much worse things could be.

Concentrated on something good that could come out of the whole thing.

Avoidance Coping: Cognitive or behavioural attempts to avoid the problem including engaging in wishful fantasy or evasive manoeuvres. The items comprising this scale were as follows:

Postponed dealing with the problem.

Avoided discussion of the issue.

Wished that I could change what had happened.

Avoided things that reminded me of the problem.

Wished the situation would go away or somehow be over with.

Fantasized about how things might turn out.

Wished I was a stronger person - more optimistic and forceful.

Even though the problem had not been sorted out, I tried to forget about the whole thing.

The 19 items listed above comprised the Event Coping Scale and were drawn from the Ways of Coping Scale (Lazarus and Folkman, 1984a, b) or constructed by the author on the basis of the stress and coping literature. All items were answered "yes" or "no" (see Appendix I).

7.4.5. Emotional Response.

The present study required that subjects indicate the magnitude of their aversive emotional response to any situation that they recorded in the Self-Monitoring Diary. Given that
subjects were required to respond to a number of questionnaire items on the diaries it was desirable that the assessment of their emotional response be as brief and simple as possible.

Visual analogue scales have been observed to offer reliable measures of such emotions as depression (Folstein and Luria, 1973) and well-being (Clark and Spear, 1964). Scores on visual analogue scales assessing depression have been found to correlate with scores on the Zung (1965) Depression Scale (Folstein and Luria, 1973), and to be sensitive to reductions in depression following treatment with anti-depressant medications (Zealley and Aitken, 1969). Thus, some measure of validity can be attached to these instruments. Accordingly, it was decided to employ a visual analogue scale in the assessment of each subject's emotional response to stressful events. The scale was 95 mm in length. The left hand anchor point was labelled "not at all upset"; the right hand anchor point was labelled "as upset as I have ever been". Subjects were required to place a small vertical line across the scale at the point representing how upset they felt during the stressful event (see Appendix I).

7.4.6. Headache Activity.

Headache activity was assessed by means of hourly diary recordings. The method was similar to that described in Chapter 6, except that, instead of rating headache intensity on a visual analogue scale, subjects were required to indicate their level of pain on a 6-point category scale ranging from 0 (no headache) to 5 (extremely intense headache). This procedure is very similar to that originally described by Budzynski et al (1970, 1973). The headache recording component of the Self-Monitoring Diary is presented in Appendix I.

A category scale was preferred to the visual analogue scale, employed in Chapter 6, because the bit-pad equipment, essential to the rapid and efficient measurement of thousands of visual analogue scales, was not available. Furthermore, it was considered that the increased sensitivity of visual analogue scales to reductions in pain intensity (Scott and Huskisson, 1976) was not necessary for the purposes of the present study.

7.5. METHOD.

7.5.1. Subjects.

The sample of subjects participating in the study comprised 120 headache sufferers aged between 17 and 67 years, with a mean age of 38.7 years (S.D. = 12.8 years). Their years of
full-time education ranged from 5 to 24 years, with a mean of 13.5 years (S.D. = 3.5 years). There were 88 females and 32 males. Seventeen subjects were recruited from the Neurology Outpatients Clinic at the Prince Of Wales Hospital, Sydney. Another 103 subjects were recruited from the Sydney Community, by means of a newspaper article and advertisement.

The study was not confined to any single headache type, but sought to determine whether or not the hypothesised relationships between variables, existed across the spectrum of headache activity, from tension to migraine headache.

All prospective subjects were contacted by telephone. They were accepted into the study if they satisfied the following criteria:

1. They had been suffering from headaches for at least one year.

2. They estimated their headaches to occur on an average of at least one day in each week.

3. Their headaches were not constant. This requirement was important because the study was concerned with the factors that precipitate headache attacks.

4. The onset of their headaches was not associated with any accident or disease.

5. For females taking the contraceptive pill, the onset or worsening of headache was not associated with their commencement of this method of birth control.

6. They were not receiving any form of psychotherapy, stress-management, or relaxation training.

7. They had made no change to their usual medication for headache in the previous month.

The criteria outlined above resulted in the selection of the 120 headache sufferers described at the beginning of this section.

7.5.2. Procedure.

Following the initial telephone contact, all subjects were interviewed individually, and in person, by the experimenter. They were told that they would be participating in an
investigation of the relationship between stress and headache. The procedures were outlined briefly, and subjects signed a statement giving their informed consent to participate. All subjects were informed that although the results of the study might be published, their identity would be fully confidential.

Subjects were interviewed and asked to indicate the site of their headaches and to estimate the proportion of headaches (none, less than half, more than half) accompanied by any visual disturbance (loss of vision or scintillating scotoma), nausea, vomiting or sensitivity to light. They then completed the Self-Control-Efficacy Scale and a shortened version of the Marlowe-Crowne Social Desirability Scale (Strahan and Gerbasi, 1972).

The Social Desirability Scale was administered in view of Krause's (1985) observations concerning the behaviour of interaction terms in multiple regression analyses. Krause (1985) studied the moderating effects of locus of control on the relationship between life events and depression. This involved testing the significance of the coefficient pertaining to the locus of control x life events interaction term obtained through a multiple regression analysis (Cohen and Cohen, 1975; Finney, Mitchell, Cronkite and Moos, 1984). He found that the magnitude (and hence significance) of this interaction term was reduced when a measure of social desirability was not included in the equation. Krause (1985) concluded that this reduction resulted from the effects of a need for approval upon the remaining variables under study and suggested that these effects should be controlled by including social desirability as a variable within the regression equation. Accordingly, it was decided to employ the Social Desirability Scale in the present study. The shortened 20-item version of the Marlowe-Crowne Scale (Strahan and Gerbasi, 1972) was preferred to the original 33-item scale (Crowne and Marlowe, 1960) because it is shorter, correlates highly with the original scale (r > 0.9) and has a high level of internal consistency (Strahan and Gerbasi, 1972). The scale requires subjects to respond "true" or "false" to each item. It was termed the "Personal Reaction Inventory" so that subjects might be unaware that it was assessing social desirability.

After completing the questionnaires, subjects were instructed in the use of the Self-Monitoring Diary. The diary consisted of 7 double sided sheets (one for each day of the week) each diary being prefaced by an instructions sheet (see Appendix I). Each of the 7 recording sheets was divided into "Headache Recording" and "Event Recording" sections. As outlined earlier, subjects rated the intensity of their headaches for every hour of the day from 6.00 am to 5.00 am the following day on a 6-point category scale. They were required to commence recording as soon as a headache began and to continue recording until they made a zero rating, indicating that the headache had gone. They re-commenced recording if and when headache resumed. Subjects were also instructed to detail the type and quantity of medication consumed
on any particular day. Measures of headache frequency, intensity and duration were derived in the manner described in Chapter 6.

At the end of each day, subjects were required to complete the Event Recording Section of the form. They were first required to respond "yes" or "no" to the question:

"Was there anything today that you found at all irritating or upsetting? This can be an event that actually occurred today, or one that you recalled from the past or some future event that you thought about."

If they responded "no" to this first question they were not required to complete the remaining items. If they responded "yes" they were required to give a brief description of the event and to indicate the time at which it occurred to the nearest hour. They then completed the appraisal scales and Event Coping Scale described previously. The Event Coping Scale was presented as a list of some of the ways in which people react to events, rather than as a coping scale, in order to avoid the favourable connotations associated with the term "coping". Subjects then indicated how upset they felt during the event on the visual analogue scale described previously. Finally, subjects were asked to respond "yes" or "no" to the question: "Did you have a headache today?" If they answered "yes" they were required to indicate whether the event occurred before, during or after the headache. This last question was included in order to account for occasions when events occurred within one hour of headache onset. On such occasions the hourly calibration on the Headache Recording Section of the form would have made it impossible to determine the temporal relationship between the two variables solely on the basis of these recordings.

All of the items described above were included on the Self-Monitoring Diary, which is presented in Appendix I.

In order to illustrate the way in which the Event Recording Section of the diary was to be used, each subject was asked to describe a recent stressful event. Employing this event as an example, the experimenter guided the subject through completion of the form. Each subject was provided with a set of written instructions which they could take home and use for their own reference. These same instructions were also read aloud to subjects by the experimenter, and were as follows:

Irritating or upsetting events include common day to day hassles, minor annoyances, or frustrations, as well as any major pressures, problems or difficulties that may occur.

At the end of each day, REGARDLESS OF WHETHER OR NOT A HEADACHE HAS OCCURRED, please complete the Event Recording Section of the form. If an irritating or upsetting event did occur on that day, circle YES in reply to the first question, and be sure to complete both sides of the form.
Irritating or upsetting events may occur on days when you don't have headaches as well as on days when you do have headaches. So, in reply to the first question, you will circle YES or NO REGARDLESS OF THE PRESENCE OR ABSENCE OF HEADACHE.

If an event that irritated or upset you occurred on any day, circle YES and fill out both sides of the form WHETHER OR NOT YOU HAD A HEADACHE ON THAT SAME DAY.

All subjects were required to monitor their headaches, stressful events and reactions to those events for 28 days (Levor et al., 1986). Accordingly, each subject was provided with four Self-Monitoring Diaries, containing a total of 28 separate recording sheets. The diaries were designed to be small enough to be carried in a pocket or handbag. Subjects were asked to keep one diary with them at all times, and to record headaches as they occurred, rather than recording them retrospectively. All subjects were instructed to telephone the experimenter if they had any queries concerning the procedures. They were also asked not to make any alterations to their usual medication for headache during the course of the study.

After each subject had completed one week's recording, they were telephoned by the experimenter in order to ensure that they were complying with the procedures. Each subject was re-contacted towards the end of the recording period, to make arrangements for them to attend the laboratory on a second occasion, so as to return their Self-Monitoring Diaries, be briefed fully by the experimenter on the aims of the study, and be allowed the opportunity of discussing their completed records.

7.6. RESULTS.

Of the 120 subjects who entered the study, six withdrew during the self-monitoring period. One had to be admitted to hospital for a lengthy period and was unable to continue the recordings. The remaining five subjects who withdrew indicated that they could not find the time to maintain accurate records. Thus, diary recordings of headache, stressful events and reactions to those events were available from 114 subjects.

7.6.1. Identification of Cycles.

Each subjects' continuous records of headache activity were inspected for the identification of headache and headache-free cycles. As stated previously, a headache cycle was defined as a headache-free day followed by a day with headache, whilst a headache-free cycle was defined as two consecutive headache-free days. These cycles were defined independently of one another. That is, the second day of the headache-free cycle was not
counted simultaneously as the first day of a second headache-free cycle or as the first day of a headache cycle. Such a practice was avoided because it could have produced artifactual results arising from counting the one stressful event twice: First within one cycle and then within another. Thus, the two days comprising any given cycle were specific to that cycle alone.

The manner in which cycles were identified was an important consideration, for the number of each cycle type identified for any given subject would vary depending on the method employed. Thus, it was important to use one method consistently and one which, as far as possible, balanced the number of headache and headache-free cycles identified. With these considerations in mind, the following method of identifying cycles was employed:

Given that continuous series of headache-days would be excluded from cycles by virtue of the cycle definitions given earlier, whilst consecutive headache-free days would be included as headache-free cycles, it was decided to select cycles such that the number of headache cycles (and thus headache days) included would be maximised. A second advantage of such a procedure was that by identifying headache cycles first, headache-free cycles would be identified such that the second day of the headache-free cycle did not immediately precede a headache day. Such a headache-free day would have already been included as the first day of a headache cycle. Thus, this method of identifying headache cycles first ensured that any two headache-free days, included in a headache-free cycle, would be separated from any subsequent headache day by at least another headache-free day (the first day of the headache cycle). Accordingly, headache-free cycles were separated from any subsequent headache day by at least one headache-free day. Such a separation of cycle types was considered desirable from the point of view of minimising any lagged effect of stressful events occurring in headache-free cycles on headache activity in subsequent headache cycles.

After identifying headache cycles, all consecutive pairs of headache-free days were included in headache-free cycles. Where an odd number of headache-free days clustered together, these were paired into cycles such that one non-cycle headache-free day would precede the start of a headache cycle, thereby, once again, maximising the period between any headache-free cycle and subsequent headache cycle.

The method outlined above resulted in the identification of 432 headache cycles and 347 headache-free cycles amongst the 114 subjects who completed the self-monitoring diaries. It was possible to identify at least one of each cycle type for 82 of these 114 subjects.
7.6.2. Internal Analysis of Scales.

The internal consistency of the Self-Control-Efficacy Scale was evaluated by means of coefficient alpha (Cronbach, 1951). An alpha value of 0.76 was obtained (see Appendix H for the full reliability analysis).

The Event Coping Scale required dichotomous responses, and thus the internal consistency of its subscales was evaluated by calculating coefficient KR-20 (Kuder and Richardson, 1937).

No reliability analysis was conducted on the Social Desirability Scale in view of the fact that results attesting to a high level of internal consistency for this scale have already been provided by previous researchers (Strahan and Gerbasi, 1972).

The 114 subjects who completed the self-monitoring phase of the study filled out the Event Coping Scale between one and 28 times. A total of 935 completed coping scales were available for analysis. The KR-20 coefficients were 0.72 for the Direct and Avoidance Coping Scales and 0.61 for the Affective Regulation Scale (see Appendix I).

In order to determine whether or not the three subscales of the Event Coping Scale represented separate dimensions of coping, the 19 items comprising the Scale were subjected to a principal components analysis (PA 1) with varimax rotation (Nie, Hull, Jenkins, Steinbrenner and Bent, 1975). All 935 completed records were employed in this analysis. In line with expectations, three factors were extracted and accounted for 38.7% of the variance. Accepting rotated factor item loadings of 0.3 or greater as significant (Comrey, 1973; Gorsuch, 1983), all of the Direct Coping items loaded significantly on the first factor; all of the Avoidance Coping items loaded significantly on the second factor, and all of the Affective Regulation items loaded significantly on the third factor. Each items' factor loadings were significant for only a single factor, except for item 1 (Doubled my efforts and tried harder to make things work out) which loaded as expected on the Direct Coping factor but also on the Affective Regulation factor; and item 8 (Fantasized about how things might turn out), which loaded as expected on the Avoidance Coping factor, but also on the Direct Coping factor (see Appendix I).

The results of the principal components analysis corresponded very closely to the hypothesized structure of the Event Coping Scale, suggesting that the scale may be regarded as assessing three different ways of coping, although inspection of the correlation matrix in Appendix I suggests that the scales cannot be regarded as independent.
7.6.3. Planned Analyses.

In each of the following analyses the Type I error rate was set at 0.05 unless stated otherwise.

Hypotheses 1 and 2.

The first two hypotheses concerned the relationship between stressful events and headache frequency, and the proposed moderating effect of self-control-efficacy. These hypotheses were evaluated by means of a hierarchical multiple regression analysis (Cohen and Cohen, 1975). Within such an analysis, the moderator effect is assessed by entering all variables into the equation, and then entering the interaction term. In this instance, the interaction term was stressful events x self-control-efficacy. The values of this term were obtained by simply multiplying the scores for stressful events and self-control-efficacy for each subject (Cohen and Cohen, 1975). The expectation was that as the level of self-control-efficacy increases the correlation between stressful events and headache frequency would become weaker. Such a relationship would be indicated if the standardised regression coefficient for the interaction term were found to be significantly less than zero (Finney et al., 1984).

Given that the absence of any control for social desirability has been found to attenuate moderator effects, as assessed through multiple regression analysis (Krause, 1985), the social desirability scores were entered into the equation prior to the entry of the interaction term. The results of this analysis are presented in Table 7-1.
### Table 7-1

Hierarchical Regression Analysis Relating Stressful Events, Self-Control-Efficacy and Social Desirability to Headache Frequency (N = 114).

<table>
<thead>
<tr>
<th>Predictor Variables</th>
<th>r</th>
<th>Step 1</th>
<th>Step 2</th>
<th>Step 3</th>
<th>Step 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Events</td>
<td>0.21*</td>
<td>0.21*</td>
<td>0.22*</td>
<td>0.22*</td>
<td>1.31*</td>
</tr>
<tr>
<td>Self-Control-Efficacy</td>
<td>0.06</td>
<td>0.08</td>
<td>0.06</td>
<td>0.41*</td>
<td></td>
</tr>
<tr>
<td>Social Desirability</td>
<td>0.11</td>
<td>0.11</td>
<td>0.11</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Events x Self-Control-Efficacy (interaction)</td>
<td></td>
<td>0.19*</td>
<td>-1.13*</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| Change R²           | 0.043 | 0.007 | 0.010 | 0.033 |
| F-ratio for change R² | 5.037* | 0.774 | 1.269 | 3.934* |
| Total R²            | 0.043* | 0.050* | 0.061* | 0.093* |

*Indicates p < 0.05 (2-tailed significance test).

The significant positive correlation between stressful events and headache frequency provides support for Hypothesis 1. The significant negative standardised regression coefficient of the interaction term (events x self-control-efficacy) is consistent with the hypothesis that self-control-efficacy operates as a moderator of the relationship between stressful events and headache frequency, this relationship becoming weaker as the level of self-control-efficacy increases (Hypothesis 2).

A second way to illustrate the moderating effect of self-control-efficacy is to compare the correlation between stressful events and headache frequency for subjects high in self-control-efficacy with that for subjects scoring low on this variable. "High" and "low" groups were established with cut-off points placed one standard deviation above and below the mean Self-Control-Efficacy Scale score for the 114 subjects who recorded stressful events and headaches (Mean = 25.90; S.D. = 5.25). For the 23 subjects high in self-control-efficacy, the
correlation between the frequency of stressful events and the frequency of headache was not significant \( (r = 0.13; 2\text{-tailed } p > 0.05) \). For the 21 subjects low in self-control-efficacy, the correlation between these variables was significant \( (r = 0.70; 2\text{-tailed } p < 0.001) \). Fisher's z-test for the difference between two independent correlations (Cohen and Cohen, 1975) yielded a z-value of 2.27 \( (1\text{-tailed } p = 0.012) \), indicating that the correlation was significantly greater for the low self-control-efficacy subjects.

The results indicate that the frequency of stressful events is positively and significantly correlated with the frequency of headache, and that this relationship is buffered or moderated by the level of self-control-efficacy.

**Hypotheses 3 and 4.**

In Hypotheses 3 and 4 it was predicted that higher levels of self-control-efficacy would be associated with higher levels of affective regulation, lower event mattered ratings and higher event change ratings. The results did not support the hypothesis that self-control-efficacy would be associated positively with affective regulation (Hypothesis 3), nor the hypothesis that self-control-efficacy would be correlated positively with event change or event mattered ratings (Hypothesis 4) (see Table 7-2). High levels of self-control-efficacy were associated significantly with low levels of avoidance coping, but were not correlated significantly with any other aspect of coping or with the strength of the emotional response to the stressor (see Table 7-2).

**Table 7-2**

<table>
<thead>
<tr>
<th>Variable</th>
<th>r</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Event Mattered Rating</td>
<td>0.04</td>
<td>N.S.(^+)</td>
</tr>
<tr>
<td>Event Change Rating</td>
<td>0.03</td>
<td>N.S.(^+)</td>
</tr>
<tr>
<td>Direct Coping</td>
<td>-0.05</td>
<td>N.S.(^*)</td>
</tr>
<tr>
<td>Avoidance Coping</td>
<td>-0.35</td>
<td>N.S.(^*)</td>
</tr>
<tr>
<td>Affective Regulation</td>
<td>0.08</td>
<td>N.S.(^+)</td>
</tr>
<tr>
<td>Upset Rating</td>
<td>-0.16</td>
<td>N.S.(^*)</td>
</tr>
</tbody>
</table>

\(^*\)Indicates a 2-tailed test (unplanned analysis).
\(^+\)Indicates a 1-tailed test (planned analysis).
Hypothesis 5.

In Hypothesis 5 it was predicted that, when events are appraised as amenable to change, subjects will engage in more direct coping and less avoidance coping. Change ratings correlated significantly and in the expected direction with direct coping ($r = 0.43; 1$-tailed $p < 0.001$), thereby supporting the hypothesis. However, contrary to the hypothesis, avoidance coping also correlated significantly and positively with change ratings ($r = 0.17; 1$-tailed $p = 0.038$).

In view of the fact that change ratings were positively correlated with both direct and avoidance coping, it was decided to examine whether or not avoidance coping accounted for a significant proportion of the variance in change ratings, independent of the variance accounted for by direct coping. This was accomplished by means of a stepwise multiple regression analysis, relating direct and avoidance coping to change ratings. The results indicated that the relationship between direct coping and change ratings remains significant despite the entry of avoidance coping into the equation, and that avoidance coping accounts for no significant proportion of the variance in change ratings, beyond that already accounted for by direct coping (see Table 7-3).

Table 7-3
Stepwise Regression Analysis Relating Direct and Avoidance Coping to Change Ratings ($N = 114$).

<table>
<thead>
<tr>
<th>Predictor Variables</th>
<th>$r$</th>
<th>Standardised Regression Coefficients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Step 1</td>
</tr>
<tr>
<td>Direct Coping</td>
<td>$0.43^{**}$</td>
<td>$0.43^{**}$</td>
</tr>
<tr>
<td>Avoidance Coping</td>
<td>$0.17^*$</td>
<td></td>
</tr>
<tr>
<td>Change $R^2$</td>
<td></td>
<td>$0.184^{**}$</td>
</tr>
<tr>
<td>F-ratio for change $R^2$</td>
<td>$25.188^{**}$</td>
<td>$0.067$</td>
</tr>
<tr>
<td>Total $R^2$</td>
<td></td>
<td>$0.184^{**}$</td>
</tr>
</tbody>
</table>

*Indicates $p < 0.05$ (2-tailed significance test).

**Indicates $p < 0.001$ (2-tailed significance test).
The results supported the hypothesis that change ratings would be positively correlated with direct coping but not the hypothesis that change ratings would be negatively correlated with avoidance coping.

Hypotheses 6 and 7.

In Hypothesis 6 it was predicted that events appraised as less threatening and as more amenable to change would be associated with lower levels of emotional upset. In Hypothesis 7 it was predicted that lower levels of direct coping, lower levels of affective regulation and higher levels of avoidance coping would be associated with higher levels of emotional upset.

The results supported Hypothesis 6 with respect to the relationship between event mattered ratings and emotional upset, but no significant relationship between change ratings and emotional upset was observed (see Table 7-4).

Hypothesis 7 was supported with respect to avoidance coping which correlated positively and significantly with upset ratings (see Table 7-4). Against the hypothesis, however, affective regulation was not correlated with upset ratings and direct coping correlated positively (not negatively as expected) with upset ratings (see Table 7-4).

Table 7-4
Correlations: Diary Recordings of Appraisal and Coping
Variables with Upset Ratings (N = 114).

<table>
<thead>
<tr>
<th>Variable</th>
<th>r</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Event Mattered Rating</td>
<td>0.37</td>
<td>0.001</td>
</tr>
<tr>
<td>Event Change Rating</td>
<td>0.01</td>
<td>N.S.</td>
</tr>
<tr>
<td>Direct Coping</td>
<td>0.22</td>
<td>0.01</td>
</tr>
<tr>
<td>Avoidance Coping</td>
<td>0.27</td>
<td>0.002</td>
</tr>
<tr>
<td>Affective Regulation</td>
<td>0.06</td>
<td>N.S.</td>
</tr>
</tbody>
</table>

In view of the unexpected positive correlation between direct coping and upset ratings, it was decided to examine whether or not this variable accounted for a significant proportion of the variance in upset ratings beyond the variance accounted for by avoidance coping. This was accomplished by means of a step-wise multiple regression analysis relating direct and avoidance coping to upset ratings. The results indicated that the relationship between
avoidance coping and upset ratings remains significant despite the entry of direct coping into the equation, and that direct coping accounts for no significant proportion of the variance in upset ratings beyond that already accounted for by avoidance coping (see Table 7-5). Thus, the results supported Hypothesis 7 with respect to avoidance coping, but not with respect to direct coping.

### Table 7-5

Regression Analysis Relating Direct and Avoidance Coping to Upset Ratings (N = 114).

<table>
<thead>
<tr>
<th>Predictor Variables</th>
<th>r</th>
<th>Standardised Regression Coefficients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Step 1</td>
</tr>
<tr>
<td>Avoidance Coping</td>
<td>0.27</td>
<td>0.27*</td>
</tr>
<tr>
<td>Direct Coping</td>
<td>0.22*</td>
<td></td>
</tr>
<tr>
<td>Change R²</td>
<td>0.071</td>
<td></td>
</tr>
<tr>
<td>F-ratio for change R²</td>
<td>8.567*</td>
<td>2.223</td>
</tr>
<tr>
<td>Total R²</td>
<td>0.071*</td>
<td>0.089*</td>
</tr>
</tbody>
</table>

* Indicates p < 0.05 (2-tailed significance test).

**Hypothesis 8.**

In Hypothesis 8 it was predicted that a greater number of stressful events would occur in headache cycles, prior to headache onset, than in headache-free cycles. Eighty two subjects reported at least one of each cycle type. These data were employed in a within subjects analysis conducted by means of a one-tailed paired t-test. A significant difference between cycles was observed ($t (81) = 2.29; p = 0.013$) with a greater number of stressful events occurring in headache (Mean = 0.243; S.D. = 0.192) than in headache-free cycles (Mean = 0.188; S.D. = 0.226). Thus, the results were supportive of the hypothesis.
Hypotheses 9, 10 and 11.

These hypotheses pertain to the behaviour of the appraisal, coping, and upset data across headache and headache-free cycles. The within-subjects analysis of these data required that subjects report at least one stressful event within each cycle type (the appraisal coping and upset measures were employed by subjects only when they reported a stressful event). Forty-two subjects met this criterion and were included in the paired t-test analysis.

Counter to Hypothesis 9, events occurring in headache-free cycles were assigned higher mattered ratings than those occurring in headache cycles. However, the hypothesis that events occurring in headache-free cycles would be appraised as being more amendable to change than those occurring in headache cycles, was supported (see Table 7-6).

Hypothesis 10 was supported with respect to affective regulation and direct coping, each of which was higher in headache-free than in headache cycles. However, the result for avoidance coping ran counter to the hypothesis that there would be higher levels of this type of coping in the headache than in the headache-free cycles (see Table 7-6).

Table 7-6

<table>
<thead>
<tr>
<th>Variable</th>
<th>Headache Cycle</th>
<th>Headache-Free Cycle</th>
<th>Paired t-value</th>
<th>1-tailed p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mattered Rating</td>
<td>2.714 (0.808)</td>
<td>3.015 (0.733)</td>
<td>-2.20</td>
<td>0.017</td>
</tr>
<tr>
<td>Change Rating</td>
<td>0.661 (0.725)</td>
<td>0.967 (0.879)</td>
<td>-1.92</td>
<td>0.030</td>
</tr>
<tr>
<td>Direct Coping</td>
<td>2.682 (1.615)</td>
<td>3.116 (1.651)</td>
<td>-1.69</td>
<td>0.049</td>
</tr>
<tr>
<td>Avoidance Coping</td>
<td>3.229 (1.540)</td>
<td>3.585 (1.823)</td>
<td>-1.75</td>
<td>0.044</td>
</tr>
<tr>
<td>Affective Regulation</td>
<td>2.225 (1.317)</td>
<td>2.672 (1.380)</td>
<td>-2.42</td>
<td>0.010</td>
</tr>
<tr>
<td>Upset Rating</td>
<td>39.531 (18.942)</td>
<td>46.843 (19.904)</td>
<td>-2.40</td>
<td>0.011</td>
</tr>
</tbody>
</table>

In the above table the standard deviations are entered in brackets and degrees of freedom = 41.
Contrary to Hypothesis 11, upset ratings were significantly lower in headache than in headache-free cycles (see Table 7-6).

7.6.4. Supplementary Analyses.

Analysis 1.

Ninety subjects reported at least one stressful event occurring within a period of headache, thereby making it possible to determine whether stressful events preceded increases in headache intensity. This hypothesis was explored by taking each individual headache that included a stressful event and calculating the mean headache intensity prior to the event, and the mean intensity following the event. For those subjects reporting a stressful event during headache on more than one day, thus providing more than a single mean score for each intensity measure, the average of these means served as the unit of analysis. Thus, for each of the 90 subjects, there was a pre-event mean intensity and a post-event mean intensity. The significance of the difference between these means was evaluated with a 2-tailed paired t-test. Ratings of headache intensity were found to be significantly greater (t (89) = 2.39; p < 0.02) after stressful events (Mean = 2.15; S.D. = 0.75) than before stressful events (Mean = 1.93; S.D. = 0.84).

Analysis 2.

In view of the result of Analysis 1, it was decided to explore the relationship between changes in headache intensity following the stressful event and appraisal, coping and emotional responses, as recorded on the Self-Monitoring Diary. Eighty nine of the 90 subjects examined in Analysis 1 recorded data on the above variables. A stepwise multiple regression analysis was conducted on the mean headache intensity ratings made after the event. The appraisal, coping and emotional responses to the event served as predictor variables. Since a correlational analysis showed that post-event headache intensity ratings were correlated significantly with pre-event intensity ratings, and with the number of headache hours following the event (post-event duration) (see Table 7-7), these variables were entered into the equation on the first step of the analysis. Once again, for those subjects reporting a stressful event during headache on more than a single day, the mean for each of the variables served as the unit of analysis.
The results indicated that increases in avoidance coping in response to stressful events occurring within headache attacks, were associated significantly with increases in headache intensity following the event. Increases in direct coping in response to these stressful events were associated significantly with reductions in headache intensity (see Table 7-7).

Table 7-7

Stepwise Regression Analysis Relating Appraisal, Coping and Emotional Upset Variables to Post-Event Headache Intensity (N = 89).

<table>
<thead>
<tr>
<th>Predictor Variables</th>
<th>Standardised Regression Coefficients</th>
<th>Step 1</th>
<th>Step 2</th>
<th>Step 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Post-Event Duration</td>
<td>0.54*</td>
<td>0.46**</td>
<td>0.46**</td>
<td>0.44**</td>
</tr>
<tr>
<td>Pre-Event Intensity</td>
<td>0.39**</td>
<td>0.24**</td>
<td>0.25**</td>
<td>0.19**</td>
</tr>
<tr>
<td>Avoidance Coping</td>
<td>0.22*</td>
<td>0.21*</td>
<td>0.26**</td>
<td></td>
</tr>
<tr>
<td>Direct Coping</td>
<td>-0.30**</td>
<td>-0.24**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Change R²</td>
<td>0.343</td>
<td>0.045</td>
<td>0.051</td>
<td></td>
</tr>
<tr>
<td>F-ratio change R²</td>
<td>22.439**</td>
<td>6.296**</td>
<td>7.633**</td>
<td></td>
</tr>
<tr>
<td>Total R²</td>
<td>0.343**</td>
<td>0.388**</td>
<td>0.439**</td>
<td></td>
</tr>
</tbody>
</table>

*Indicates p < 0.05 (2-tailed significance test).
**Indicates p < 0.001 (2-tailed significance test).

After the third step of the analysis, presented above, none of the remaining self-monitoring variables accounted for a significant proportion of the variance in the post-event headache intensity scores.

Analysis 3.

Since primary appraisal has been thought to be a significant determinant of coping behaviour (Lazarus and Folkman, 1984a, b) it was decided to correlate the event mattered
ratings with the coping measures. For the 114 subjects who completed the study, the
correlations of event mattered ratings with direct, avoidance and affective regulation coping
were 0.17, 0.09 and 0.00 respectively. None of these correlations were significant on a 2-tailed
test.

**Analysis 4.**

It was decided to divide the sample of headache subjects into migraine, tension-vascular
and tension headache types to explore the possibility that these groups might score differently
on the variables under investigation.

Employing the continuum model of headache severity (Bakal and Kaganov, 1979;
Holroyd and Andrasik, 1982a) as a basis for classification, subjects were divided into
headache types, according to the frequency with which they reported the following migrainous
features in association with their headaches:

1. Visual disturbance (loss of vision or scintillating scotoma).
2. Nausea.
3. Vomiting.

These features are widely recognised as defining characteristics of migraine (e.g., Ad

Subjects were classified as migraineurs if they reported two or more features occurring
in association with more than 50% of their headaches; as tension-vascular headache sufferers
if they reported at least two features occurring with less than 50% of their headaches, and / or a
single feature occurring with more than 50% of their headaches; or as tension headache
sufferers if they reported no more than a single feature occurring with less than 50% of their
headaches.

The above classification resulted in 33 migraineurs, 65 tension-vascular headache
sufferers, and 22 tension headache sufferers. Differences between these groups on each
variable studied were examined for significance by means of one-way analyses of variance. No
significant results were obtained (see Appendix J).
7.7. DISCUSSION.

Study II was concerned with an examination of the theoretical model outlined in Section 7.1.4. This model was concerned with an explanation of the occurrence of headache in terms of stressful events and the processes of cognitive appraisal and coping which are thought to determine the impact of these events on the individual's psychological and somatic functioning (Aldwin and Revensen, 1987; Folkman and Lazarus, 1986; Folkman, Lazarus, Gruen et al., 1986; Manne and Sandler, 1984, Pearlin, Menaghan, Lieberman and Mullen, 1981).

Consistent with the view that stressful events are the most common precipitants of headache attacks (e.g., Friedman et al., 1954; Howarth, 1965; Selby and Lance, 1960), the frequency of stressful events was found to be greater in periods preceding headache (i.e., in headache cycles prior to headache onset), than in periods of headache freedom (i.e., in headache-free cycles). A similar finding was reported by Levor et al, (1986). The study conducted by Levor et al (1986) was concerned only with migraine headache attacks. Thus, the results of the present study extend the observed relationship between stressful events and headache onset to the spectrum of headache encompassing both the tension and migraine types.

Other studies seeking relationships between stressful events and headache onset, have merely relied upon headache sufferers' own retrospective reports (e.g., Friedman et al., 1954; Henryk-Gutt and Rees, 1973; Howarth, 1965), or have attempted to show that headache sufferers report a greater frequency of stressful events than headache-free control subjects (e.g., Kearney et al., 1987; Holm et al., 1986). However, these studies cast no light on the question of whether or not there exists any causal relationship between stressful events and headache onset. This issue is addressed best by longitudinal studies of the kind reported in this chapter and by Levor et al (1986). The results of these latter types of study indicate that stressful events do tend to precede periods of headache, more often than they do periods of headache freedom, thereby suggesting that stressful events may be causally related to headache onset. The present study provides stronger evidence for the existence of such a relationship than that reported by Levor et al (1986), who counted stressful events occurring during headache cycles regardless of whether they occurred before, during or after headache attacks. In the present study, only those events in headache cycles occurring prior to headache onset were counted and compared with those occurring in headache-free cycles. This refinement of methodology allows greater confidence to be attached to the conclusion that stressful events precede headache onset more often than periods of headache freedom.

The present study also went beyond that of Levor et al (1986) by requiring subjects to note the times at which stressful events occurred, thus making it possible to examine the
relationship between the occurrence of stressful events during headache attacks and the intensity of subsequent headache activity. The results indicated that headaches became significantly more intense after stressful events than they were prior to the occurrence of those events. Once again, the longitudinal nature of the study makes for the suggestion that stressful events occurring during headache attacks may be causally related to subsequent increases in headache intensity.

The results of the present study suggest that common stressful events may be causally related to headache onset and to increases in headache intensity amongst recurrent headache sufferers. The possibility that subjects may have suspected that the experimenter was seeking a relationship between the occurrence of stressful events and pandered to this expectation, by making a greater effort to identify stressful events just prior to headache onset and just prior to increases in headache intensity, cannot be discounted. However, the higher upset ratings associated with stressful events occurring in headache-free than in headache cycles is inconsistent with the view that the data were affected by expectancy effects.

In addition to the findings discussed above, a significant and positive correlation was observed between the frequency of stressful events and the frequency of headache (see Table 7-1). Consistent with the model proposed in Section 7.1.4, the extent to which individuals believed that they possessed the capacity to regulate their cognitive, emotional and behavioural responses (self-control-efficacy) moderated the strength of the relationship between stressful events and headache frequency. That is, subjects with low levels of self-control-efficacy demonstrated a positive and significant relationship between the frequency of stressful events and headache frequency, whilst those with high levels of self-control-efficacy demonstrated a weak non-significant relationship.

These results suggest that self-control-efficacy may be an important psychological resource functioning to protect headache sufferers from the deleterious effects of stressful events on headache frequency, in a manner similar to that by which locus of control has been found to buffer the relationship between life events and psychological symptoms (e.g., Johnson and Sarason, 1978; Krause, 1985).

The buffering effect observed for self-control-efficacy suggests that psychological interventions for headache, such as cognitive therapy, relaxation training, and biofeedback training, might exert their therapeutic effects, at least in part, by increasing subjects' self-control-efficacy regarding their cognitive, emotional and behavioural responses to stressful events.
The analysis of subjects' appraisal, coping and emotional responses to stressful events, occurring in headache and headache-free cycles (see Table 7-6) indicated that, in headache-free cycles, subjects appraised events as being more amenable to change, employed higher levels of affective regulation coping and higher levels of direct coping in response to those events than they did in response to events occurring prior to headache in the headache cycles. These observations are consistent with the model proposed in Section 7.1.4 and with the literature suggesting that direct and affective regulation coping are associated with better adaptational outcomes (e.g., Billings and Moos, 1984; Felton and Revenson, 1984).

However, the analysis presented in Table 7-6 also produced some unexpected results.

Firstly the level of avoidance coping was greater in headache-free than in headache cycles. This observation runs counter to previous studies indicating that avoidance coping tends to be associated with poorer adaptational outcomes (e.g., Holahan and Moos, 1986; Holm et al., 1986; Manne and Sandler, 1984) and suggests that the higher level of avoidance coping observed for tension headache subjects relative to headache-free controls by Holm et al (1986), may not be related to the occurrence of headache attacks. The present findings suggest that the relationship between avoidance coping and headache onset may be quite different from the relationship between avoidance coping and emotional distress noted in the stress and coping literature. In the present study higher levels of avoidance coping were associated significantly with higher levels of emotional upset in response to stressful events, but mitigated against the onset of headache in the face of those events (see Table 7-6). Given that higher levels of direct, affective regulation and avoidance coping were found to precede periods of headache freedom, the number or diversity of coping strategies employed in response to a stressful event, rather than the actual nature of those strategies, may be an important determinant of whether or not a headache develops.

Secondly, the event mattered ratings and emotional upset ratings were greater in headache-free than in headache cycles. This pattern of findings suggests that the events most relevant to headache onset may be those which the headache sufferer perceives as being of relatively minor significance to his or her well-being. This interpretation of the results suggests the hypothesis that headache sufferers may be less sensitive to their emotional and physiological reactions, to many stressful events, than headache-free control subjects and that this may contribute to the occurrence of their attacks. Indeed, it has been emphasised that teaching headache sufferers to become more sensitive to their own cognitive, emotional and physiological reactions to stressors may be critical to the successful psychological treatment of this condition (Bakal et al., 1981).
It was found that avoidance coping in response to stressful events occurring during headache episodes was associated with an increase in headache intensity following the event, whilst direct coping was associated with a decrease in subsequent headache activity. These findings are consistent with the view that avoidance behaviour may play a significant role in sustaining chronic pain (Letham, Slade, Troup and Bentley, 1983; Philips, 1987a; Slade, Troup, Letham and Bentley, 1983). Although the longitudinal nature of the data makes it tempting to suggest that a causal relationship may exist between these two methods of coping and headache intensity following the stressful event, the fact that coping strategies are processes which develop over time and which cannot, therefore, be regarded as having occurred only in the hour at which the stressful event was recorded, would lead one to be sceptical of such a conclusion. An alternative interpretation of the findings, in terms of intense headache incapacitating the person to the extent that he or she is able to engage only in passive avoidance strategies, rather than in more active direct coping strategies, cannot be ruled out. An exploration of the direction of any causal relation between coping and headache intensity would be approached best by treatment studies seeking to alter coping and to track the relationship between such alterations and headache intensity over time.

Self-control-efficacy did demonstrate a moderating effect on the relationship between stressful events and headache frequency. Given that events appraised as more amenable to change, and coped with by means of higher levels of affective regulation, direct coping, and avoidance coping, were found to be less likely to give rise to subsequent headache activity, it was surprising that these variables were not observed to constitute the psychological processes by which self-control-efficacy exerted its buffering effect on headache frequency. The correlations of change ratings, affective regulation and direct coping with self-control-efficacy were not significant (see Table 7-2). Although event mattered ratings were correlated positively with ratings of emotional upset, this measure of primary appraisal was unrelated to self-control-efficacy.

Avoidance coping was found to correlate negatively with self-control-efficacy. This is consistent with the view that avoidance coping strategies preclude mastery experiences, and therefore stifle the growth of self-efficacy (Bandura, 1977, 1982, 1986). However, the fact that lower, rather than higher, levels of avoidance coping were associated with headache onset (see Table 7-6) suggests that this type of coping is unlikely to mediate the buffering effect of self-control-efficacy on headache frequency. Although avoidance coping was associated with higher ratings of emotional upset in response to stressors, lower rather than higher upset ratings preceded headache onset.

Thus, despite the demonstration of a significant buffering effect for self-control-efficacy, and despite the study of appraisal, coping and emotional responses, it was not
possible to elucidate the psychological mechanisms which underlie the buffering process. A similar state of affairs was reported by Folkman, Lazarus, Gruen et al (1986), who found that, although mastery correlated negatively with measures of psychological and health status, no significant relationships between mastery and appraisal or coping processes could be identified. More research is required into the buffering effect of self-control-efficacy on the relationship between stressful events and headache frequency in order to uncover significant mediational processes.

Lazarus and his colleagues (Lazarus, 1977; Lazarus and Folkman, 1984a, b) have argued that appraisal processes determine both coping responses and adaptational outcomes. In the present study, event mattered ratings (primary appraisals) did not correlate significantly with any event coping variable (see Analysis 3), but did correlate significantly and positively with upset ratings (see Table 7-4). Event change ratings (secondary appraisals) did not correlate significantly with upset ratings, but did correlate positively and significantly with direct coping scores (see Table 7-3). This latter finding is a replication of the result obtained by Parkes (1984) who used a similar measure of direct coping. The finding is also consistent with the observation that when events are appraised as amenable to change, subjects tend to engage in more problem-focused coping (Folkman and Lazarus, 1980). In the present study the appraisal of events as amenable to change mitigated against the occurrence of headache.

In view of the above findings, one could speculate that appraising stressful events occurring during headache attacks as amenable to change, might be important in facilitating the use of direct coping strategies, which were found to be associated with lower ratings of headache intensity subsequent to stressful events occurring during headache attacks (see Table 7-7). Thus, through the action of secondary appraisal processes on direct coping, the increase in headache intensity, that was observed to occur following stressful events, may be minimised.

Consistent with the stress and coping literature indicating that avoidance coping strategies tend to be associated with poorer adaptational outcomes (e.g., Aldwin and Revenson, 1987; Cronkite and Moos, 1984; Holahan and Moos, 1986; Manne and Sandler, 1984), avoidance coping was found to be associated with greater emotional upset in response to stressful events (see Table 7-4). Surprisingly a similar relationship was observed for direct coping. However, a multiple regression analysis revealed that direct coping did not account for a significant proportion of the variance in upset ratings, beyond that explained by avoidance coping (see Table 7-5). The significant correlation for direct coping with emotional upset may have been a spurious result, attributable to the finding that the Avoidance and Direct Coping Scales were not fully independent (see the factor analysis presented Appendix I).
The model proposed in Section 7.1.4. was not fully supported by the data collected in the present study. The major areas in which it was not supported concern the proposed linkages between self-control-efficacy, appraisal, coping, and the strength of the emotional response to stressful events occurring prior to headache onset. However, the data did support a role for stressful events in the onset and exacerbation of headache attacks and also supported a role for self-control-efficacy as a buffer of the relationship between the frequency of stressful events and the frequency of headache. A greater frequency of coping responses, in relation to stressful events, was associated with a decreased probability of subsequent headache onset, as was the tendency to appraise stressful events as amenable to change.

The results are consistent with the thesis that cognitive processes may play an important role in determining the severity of recurrent headache.

The results of the study reported in this chapter suggest that psychological interventions for recurrent headache may exert their therapeutic effects by increasing the level of perceived self-control-efficacy. Future studies could examine this hypothesis, along with the role of appraisal, coping, and emotional upset in the process of treatment, by means of studies tracking the behaviour of these variables over the course of psychological treatment. This would be particularly important from the point of view of specifying a maximally effective focus for psychological interventions.

A second line of inquiry would be to try and clarify the rather multi-faceted measure of self-control-efficacy employed in the present study. The Self-Control-Efficacy Scale assessed perceived efficacy with respect to cognitive, behavioural and emotional control. The development of a factored measure, assessing each of the three components separately, might yield more specific information on the nature of the buffering process observed in the present study.

A third avenue of investigation is suggested by the intriguing finding that stressful events are less likely to result in a subsequent headache when they are appraised as highly important and generate a substantial emotional response. The question of whether or not some suppression of cognitive or emotional responses is a characteristic of headache sufferers (Grothgar and Scholz, 1987), disposing them to react to stressful events with headache, is worthy of further investigation. Some light might be cast on this issue by obtaining similar data on headache-free control subjects, and comparing their reactions to stressful events with those of headache subjects.

It is clear that the results of the present study suggest a number of directions for further research. However, having demonstrated that there may be an important role for cognitive
processes, such as self-control-efficacy and the appraisal of events as amenable to change, in the linkage between stressful events and headache onset, the question of the psychophysiological processes through which these variables could conceivably contribute to headache would seem of paramount importance to the construction of theoretical models linking cognitive processes to headache.

As discussed in Chapter 2, a number of attempts have been made to relate psychological variables to headache through muscular and vascular processes, but these have not produced consistent results (e.g., Anderson and Franks 1981; Cohen et al., 1982; Morley, 1985). The central theory of headache (Sicuteri, 1982) suggests that stress may contribute to headache through an action on central pain control mechanisms involving serotonin and endogenous opioids. Bandura et al (1987) taught subjects cognitive strategies for the management of cold pressor pain and observed that this increased their perceived efficacy to withstand and reduce pain, and also increased their pain tolerance times. These subjects then received an injection of saline or naloxone (an opiate antagonist). Those subjects receiving naloxone became less able to tolerate pain than their counterparts receiving saline. Moreover, the extent to which pain tolerance times were reduced by naloxone was associated with the level of perceived efficacy to reduce pain following training in cognitive strategies. These results suggest that efficacy expectations may be related to pain through central pain regulatory mechanisms. Thus, it is conceivable that the concept of self-control-efficacy, studied in this chapter, could be related to headache through the process of sensory modulation as outlined in the central theory of headache.

With the aim of exploring the prospects for linking cognitive constructs with the central theory, the next chapter is concerned with an empirical evaluation of the premise that headache sufferers possess weakened sensory modulation systems.

7.8. SUMMARY.

The frequency of stressful events was found to be correlated with headache frequency and to be greater in periods preceding headache attacks than in periods of headache freedom. Stressful events occurring during headache attacks were observed to be followed by increases in headache intensity.

The relationship between stressful events and headache frequency was observed to be buffered by the level of self-efficacy regarding control over behavioural, cognitive and affective responses (self-control-efficacy). It was proposed that this buffering effect of self-control-efficacy would be mediated by appraisals of the importance of stressful events and of
the extent to which those events are amenable to change; by affective regulation, direct and avoidance coping responses to stressful events and by the emotional response to stressors. The appraisal processes were found to be unrelated to self-control-efficacy. Greater frequencies of each type of coping studied mitigated against the onset of headache in response to stressful events, as did the appraisal of events as amenable to change. Avoidance coping was correlated negatively with self-control-efficacy but since lower, rather than higher, frequencies of this type of coping preceded periods of headache, avoidance coping was considered unlikely to mediate the buffering action of self-control-efficacy. Affective regulation and direct coping were unrelated to self-control-efficacy. Ratings of emotional upset were found to be lower in periods leading up to headache than in periods of headache freedom as were appraisals of the importance of stressful events. These two variables correlated significantly with each other, but neither correlated significantly with self-control-efficacy.

It was concluded that the cognitive processes of self-control-efficacy and appraisal of stressful events as amenable to change, as well as the frequency of coping behaviours, may serve to protect headache sufferers from the deleterious effects of stressful events on headache. The mechanisms by which self-control-efficacy exerted its buffering effect remained unclear. It was decided to extend the inquiry into the role of cognitive processes in recurrent headache by examining the prospects for linking these with the central theory.
CHAPTER 8

STUDY III

TOWARDS A MECHANISM LINKING COGNITIVE PROCESSES WITH HEADACHE: AN INVESTIGATION OF PAIN SENSITIVITY AND SENSORY MODULATION IN HEADACHE SUFFERERS.

8.1. INTRODUCTION.


The central theory of headache was outlined in Chapter 2. To reiterate, the theory proposes that headache is, at least in part, the result of some disorder in CNS pain regulatory mechanisms (Sicuteri, 1976, 1981, 1982). This theory is not without its problems. Firstly, there is the problem of accounting for the localisation of headache. That is, why does the headache sufferer often experience pain in a specific region of the head, rather than elsewhere in the body? To account for this observation the theory must be supplemented by postulating a heightened sensitivity to pain at the site of headache, arising from a local disturbance of pain processing in the trigeminal nerve (e.g., Drummond, 1987) or from local chemical action and/or vasodilative processes (e.g., Lance, 1982). Secondly, it is not clear whether the hypothesised deficiency in sensory modulation is a consistent feature of the headache sufferer's pain control system, or whether it is a feature which becomes apparent only when headache is present, or when the pain control system has been over-taxed, as is postulated to occur when the headache sufferer is under stress (Sicuteri, 1982). In spite of these problems, however, there is evidence to suggest that alterations in the function of endogenous pain inhibitory substances may be involved in the pathophysiology of headache (e.g., Anthony and Lance, 1985; Genazzani et al., 1984; Nappi, Facchinetti, Martignoni, Petraglia, Bono et al., 1985; Nappi, Facchinetti, Martignoni, Petraglia, Manzoni et al., 1985), and that stress may act to disrupt central pain inhibitory processes (e.g., Rossier et al., 1980; Palkovits et al., 1976). These points were discussed in Chapter 2.
In the last chapter, self-efficacy expectations regarding control over cognitive, emotional and behavioural responses, were shown to buffer the relationship between stressful events and headache frequency. The central theory of headache asserts that the central pain control system performs a similar buffering function such that, in the face of stress, the individual is less likely to experience headache if this system is "... efficient, stable and adaptable ...", but more likely to experience headache if it is "... phenotypically or genotypically fragile ..." (Sicuteri, 1982, p. 72). As discussed in Chapter 8, there is some evidence to suggest that self-efficacy to manage pain may be related to the functioning of endogenous pain inhibitory substances (Bandura et al., 1987). Accordingly, it is conceivable that the aspects of self-efficacy studied in Chapter 8, could have exerted their effects on headache frequency through some action on central pain regulatory processes.

This chapter is concerned with the prospects for articulating concepts such as self-efficacy to the mechanisms of central pain regulation specified in the central theory of headache. If psychological constructs are to be related to headache in this manner, it is necessary to explore and clarify the extent to which CNS pain control processes play a part in the pathophysiology of headache. If, as Sicuteri (1982) contends, it is the interaction of stress with a weakened pain control system that contributes substantially to the onset of headache attacks, it follows that headache sufferers should have some deficiency in their capacity for central pain inhibition, setting them apart from headache-free persons. The aim of this chapter is to conduct an evaluation of this proposition.

As noted in Chapter 2, less research has been conducted into the role of pain inhibitory processes in headache than into the involvement of peripheral mechanisms, such as vasodilatation and muscle contraction. Some researchers have examined the sensitivity of headache sufferers and headache-free persons to experimental pain, but the findings are equivocal (see Chapter 2). The majority of these studies have not matched subjects on variables such as age and sex, which are known to affect sensitivity to this kind of pain (Woodrow, Friedman, Siegelaub and Collen, 1972), and many have not controlled for the presence or absence of headache (e.g., Langemark and Oleson, 1987). The study reported in this chapter seeks to redress these methodological shortcomings, and to explore the hypothesis that there may exist a neurophysiological basis to any heightened pain sensitivity in headache sufferers, as suggested by the central theory.

8.1.2. The Study of Central Pain Control Functions.

The capacity of CNS mechanisms to modulate the impact of sensory input on the cortical structures subserving the experience of pain, has been studied by measuring pain
sensitivity (e.g., Lieberman, Corkin, Spring, Growdon and Wurtman, 1983; Sicuteri, Anselmi and Del Bianco, 1978); sampling pain inhibitory substances such as the endogenous opioids from the cerebrospinal fluid (CSF) (e.g., Genazzani et al., 1984; Nappi, Facchinetti, Martignoni, Petraglia, Bono et al., 1985; Nappi, Facchinetti, Martignoni, Manzoni et al., 1985); manipulating the level of pain inhibitory substances in the CNS by the administration of substances such as naloxone (e.g., Adams, 1976; Buchsbaum, Davis and Bunney, 1977) or parachlorophenylalanine (Sicuteri et al., 1973); or by studying the amplitude of the average evoked potential (the electrical response of the brain to sensory input) (e.g., Buchsbaum and Davis, 1979; von Knorring, 1974).

The assessment of pain sensitivity is discussed in Appendix A. A wide variety of pain producing stimuli have been employed, including the cold pressor test, radiant heat, pressure algometer and electrical stimulation, with the most common pain measures derived from these procedures being pain threshold and pain tolerance (Wolff, 1978, 1984). These measures have been found to correlate with CSF endorphin levels (von Knorring, Almay, Johansson and Terenius, 1978), and to be responsive to opiate antagonists (Buchsbaum et al., 1977; Buchsbaum, Davis, Coppola and Naber, 1981a). Thus, measures of pain sensitivity would appear to be related to physiological aspects of pain regulation, as well as responsive to psychological influences (see Appendix A).

As discussed in Chapter 2, the manipulation of brain levels of serotonin and endogenous opioids has been found to affect the response of animals to stimulation (e.g., Proudfit and Anderson, 1975; Yaksh and Rudy, 1978). However, assessing the levels of these substances in humans is made difficult by the fact that invasive procedures are required, particularly in respect of CSF sampling (e.g., von Knorring et al., 1978). A less invasive method of assessing physiological correlates of pain sensitivity is afforded by the average evoked potential (AEP) generated by the brain in response to stimulation.

8.1.3. AEP Amplitudes and Pain Modulation.

When the senses of a human or animal are stimulated, the input generates an electrical response from the cortex which may be detected in electroencephalographic (EEG) recordings. This response is termed the evoked response or evoked potential (Bartley, 1934; Dawson, 1954). These responses are very small and are easily lost in the background EEG rhythms generated by the brain (Buchsbaum, 1976; Callaway, 1966). Since the evoked potential (measured in microvolts) is a series of peaks and troughs with a reproducible form, it is possible to obtain an image of this wave by exposing the organism to repeated stimulation and averaging the response obtained. In this manner the average evoked potential becomes
apparent as the background random "noise" in the EEG averages towards zero (Buchsbaum, 1976; Callaway, 1966). The AEP develops as a series of peaks and troughs after a latent period (measured in milliseconds from the time at which stimulation was delivered). These components have been the subject of much research in psychiatry and neurology, proving most useful in the diagnosis of multiple sclerosis (Halliday, McDonald and Mushin, 1973).

Those components of the AEP studied most commonly in pain research occur over the first 300 milliseconds after stimulus onset, there being three components that have been prominent in the literature on pain modulation. For the purposes of this chapter these peaks in the AEP shall be referred to as the \( P_1 \), \( N_1 \) and \( P_2 \) peaks. Positive peaks are indicated by the letter "P"; negative ones by the letter "N". There has been some confusion over whether positive peaks should be illustrated as deflecting downwards or upwards (Callaway, 1966). The choice of direction is of course purely arbitrary. The \( P_1 \), \( N_1 \) and \( P_2 \) peaks are illustrated in Figure 8-1, with positive peaks deflecting downwards. The \( P_1 \), \( N_1 \) and \( P_2 \) peaks have been identified by reference to the time bands in which they are considered to occur. For example, Buchsbaum and his colleagues (Buchsbaum and Davis, 1979; Buchsbaum, Davis, Coppola and Naber, 1981a, b; Lavine, Buchsbaum and Poncy, 1976) have referred to the \( P_1 \), \( N_1 \) and \( P_2 \) peaks as \( P_{100} \), \( N_{120} \) and \( P_{200} \), on the basis that these components fall within the time bands 76-112 msec, 116-152 msec and 168-248 msec respectively. However, Connolly and Gruzelier (1982) found that on many occasions the three major peaks, generated in response to visual stimulation, did not fall within the respective time bands. Accordingly, rather than refer to the AEP peaks in terms of their latency, it was decided to follow Connolly and Gruzelier (1982) and employ the \( P_1 \), \( N_1 \) and \( P_2 \) terminology.

**FIGURE 8-1**

![Average Evoked Potential Peaks](image)
AEP's may be obtained by means of visual (Connolly and Gruzelier, 1982), auditory (Prescott, Connolly and Gruzelier, 1984) or somatosensory (Chen, Chapman and Harkins, 1979) stimulation. However, little is known about the cortical structures responsible for the P1, N1 and P2 components (Chundler and Dong, 1983). Thus, the relationship between AEP's and pain modulation is based almost entirely upon the association of these components with measures of pain sensitivity (e.g., Lavine et al., 1976; Ashton, Golding, Marsh and Thompson, 1984), or with measures of the level of pain inhibitory substances in the CSF (e.g., von Knorring, Almay, Johansson and Terenius, 1979).

The rationale for the application of AEP's to the study of pain modulation dates back to the work of Petrie (1967). She was concerned with assessing a dimension of personality which she termed "perceptual reactance" or "augmenting-reducing". People at the opposite extremities of this dimension were termed "augmenters" and "reducers" (Petrie, 1967). Petrie (1967) postulated that the nervous system of augmenters is such that they tend to amplify the intensity of stimulation, whilst that of reducers functions to attenuate stimulation. According to Petrie (1967), augmenters are much more likely to experience noxious stimuli as painful than reducers.

Petrie conceived of the augmenting-reducing dichotomy as having a neurophysiological basis:

"... perhaps the most helpful model of these processes is to think of them as contrasted examples of central modulation of sensory perception. Such central modulation may be dependent on the presence and nature of reverberating circuits in the nervous system... biochemical factors undoubtedly will be found to play an important part ..." (Petrie, 1967, p. 104).

Thus, the concept of augmenting-reducing, as defined by Petrie (1967), is similar to the gate control concept of pain modulation introduced by Melzack and Wall (1965).

Petrie (1967) assessed augmenting-reducing by means of the kinaesthetic figural after-effect (KFA). This after-effect is assessed by blindfolding subjects and instructing them to rest their hands with the palms upwards for 45 minutes. They are then given a wooden block (the standard) which they handle for a short period. The standard is then taken away, and subjects are presented with a wedge which becomes progressively wider from the bottom to the top. On the wedge is a movable marker which subjects move to the point which they consider to correspond to the width of the standard block. This procedure is repeated over a number of trials. Those subjects who over-estimate the size of the standard block are called augmenters; those who under-estimate its size are called reducers. Employing this methodology, augmenters have been found to be less pain tolerant and to be less disturbed by sensory deprivation than reducers (Petrie, 1967; Petrie, Collins and Solomon, 1960; Sweeney, 1966).
Buchsbaum and Silverman (1968) were the first to employ the AEP as an alternative measure of augmenting-reducing. These researches reasoned that if augmenters amplify the impact of stimulation on the nervous system, and reducers attenuate this impact, the AEP amplitudes of augmenters should increase with the level of stimulation at a greater rate than those of reducers. Employing a modification of Petrie's (1967) KFA task, Buchsbaum and Silverman (1968) calculated perceptual reactance scores. They then recorded the AEP's generated in response to four different light intensities. Each intensity was presented 120 times. The visual average evoked potentials (VEPs) were recorded from an electrode placed at C4 (see Figure 8-2). The amplitude intensity slopes for the P1, N1 and P2 components were calculated by means of least squares regression for each subject. It was found that the amplitude / intensity slope for the negative peak occurring at about 140 msec (N1), correlated significantly, and in the expected direction, with scores on the KFA.

Since Buchsbaum and Silverman's (1968) findings, researchers have been concerned with the relationship between AEP components and measures of pain sensitivity, with the most popular recording site being the vertex (Cz; see Figure 8-2) (e.g., Ashton et al, 1984; Chapman, Chen, Colpitts and Martin, 1981; Haier, 1983). Several studies have reported significant relationships between AEP components (in visual and somatosensory modalities) and tolerance for experimental pain. (e.g., Buchsbaum, 1976; von Knorring, 1978; von Knorring Espvall and Perris, 1974). No such studies have been conducted on the auditory AEP.

**FIGURE 8-2**

Buchsbaum and his colleagues have found the amplitude / intensity slope for various components of the somatosensory average evoked potential (SSEP) to be correlated with ratings of pain intensity generated by electric shock (e.g., Buchsbaum and Davis, 1979; Buchsbaum et al., 1977). These researchers have reported that opiate antagonists, such as naloxone, increase amplitude / intensity slopes in the SSEP recorded from vertex and C4 (the area of the scalp over the somatosensory cortex; see Figure 8.2) for the N1 component (Buchsbaum et al., 1977, 1981; Buchsbaum et al., 1977). However, although Buchsbaum et al (1981b) observed this effect to be present over all 29 subjects studied, Buchsbaum et al (1977) found it to occur only for those subjects with high pain tolerance levels, as assessed on a previous electrical pain test.

In a study of the analgesic effects of aspirin, this drug was found to reduce the size of the SSEP amplitude / intensity slope for the N1 component (Buchsbaum and Davis, 1979). The P1 and P2 amplitude / intensity slopes have also been related to pain sensitivity (Lavine et al., 1976), with the former variable having been found to increase with naloxone (Davis, Buchsbaum and Bunney, 1978). In each of these studies, somatosensory stimulation was employed and recordings were made from the vertex.

In many studies differences between the P1 and N1 peaks (P1-N1) (e.g., Birchall and Claridge, 1979; Buchsbaum and Pfefferbaum, 1971; Stelmack, Achorn and Michaud, 1977) and between the N1 and P2 peaks (N1-P2) (e.g., Buchsbaum, 1976; Kaskey, Salzman, Klorman and Pass, 1980) have been said to reflect augmenting-reducing. Indeed, von Knorring and his colleagues have reported on relationships between pain sensitivity and the amplitude / intensity slope of a VEP component defined as the largest peak to peak amplitude occurring over the first 150 msec of the VEP wave recorded from the occiput (von Knorring 1978; von Knorring et al., 1974).

In their conceptualisation of AEP amplitude / intensity slopes as measures of sensory inhibition, Buchsbaum and Pfefferbaum (1971) contended that augmenting-reducing on this variable represents the operation of a non-specific inhibitory process. However, the absence of significant cross-modal correlations for P1-N1 and N1-P2 peak to peak amplitude / intensity slopes, recorded from vertex in response to visual and auditory stimulation (Kaskey et al., 1980; Raine, Mitchell and Venables, 1981), suggests that augmenting-reducing in the AEP may be modality specific and, therefore, not indicative of a generalised mechanism of sensory inhibition. As Raine et al., (1981) point out, it is possible that cross-modal relationships might emerge if recordings are made over the specific cortical areas corresponding to the stimulus modalities employed.
Although each of the $P_1$, $N_1$ and $P_2$ AEP amplitudes have been related to the process of pain modulation, there is confusion over which components are of most relevance. Furthermore, drawing any conclusion from this field is made difficult by the range of methodologies employed by researchers, and by the methodological problems which characterise many of the AEP studies reviewed above. These methodological problems include the selection of a recording site, the number and intensity of stimuli, the identification and measurement of AEP component amplitudes, the contribution of eye movement artifacts and the role of attentional and arousal factors. Each of these issues is considered in the following sections.

8.1.3.1. The Selection of a Recording Site.

The phenomenon of augmenting-reducing has been found to vary markedly with the placement of the recording electrode (Connolly and Gruzelier, 1982; Prescott et al., 1984). However, in the literature on augmenting-reducing a plethora of recording sites have been employed, with little rationale for their selection being offered. In cases where the researcher is concerned with somatosensory stimulation, there is evidence to suggest that the vertex may be the site at which AEP components are observed most readily. (Goff, Matsumiya, Allison, and Goff, 1977). In topographic studies of SSEP components, these researchers observed that at vertex the amplitudes of the $P_1$, $N_1$ and $P_2$ components are at least 90% of the maximum size observed at other cranial locations. Otherwise, however, there exists little to guide researchers in their choice of a recording site.

8.1.3.2. The Number and Intensity of Stimuli.

There exist no guidelines for the choice of the number or intensity of stimuli to be employed in AEP studies of augmenting-reducing. Most studies have employed four different stimulus intensities (e.g., Buchsbaum et al., 1977; Davis et al., 1978; Stark and Norton, 1974), but others have used six (e.g., Connolly and Gruzelier, 1982; Connolly, Gawel and Rose, 1982) or three (e.g., von Knorring, 1978; von Knorring et al., 1974).

In VEP studies, the stimulus intensity has been described in terms of photostimulator settings (e.g., Buchsbaum and Pfefferbaum, 1971; von Knorring, 1977; von Knorring et al., 1974), in terms of its intensity at the source of stimulation (e.g., Haier, Robinson, Braden and Williams, 1984), or in terms of its intensity at the subject (e.g., Connolly and Gruzelier, 1982; Connolly et al., 1982). In SSEP studies, the intensity of the electrical stimulus is usually given in milli-amps, with the duration of the pulse also being specified (e.g., Ashton et al., 1984;
Buchsbaum et al., 1977, 1981b). Occasionally, the shocks are delivered according to the intensity required to generate responses from "barely noticeable" to "painful" in each subject (e.g., Lavine et al., 1976). These variations make it difficult to compare results obtained from different laboratories.

8.1.3.3. The Identification and Measurement of AEP Peak Amplitudes.

Although the $P_1$, $N_1$ and $P_2$ peaks have been said to fall within specific latency ranges (e.g., Buchsbaum, 1976; Buchsbaum and Silverman, 1968), it has been noted that the form of the AEP is sometimes poorly defined, making it difficult to identify these triphasic components (Connolly and Gruzelier, 1982). It has also been observed that the frequency with which VEP peaks occur within the time bands specified by Buchsbaum and his associates (e.g., Buchsbaum and Pfefferbaum, 1971; Buchsbaum and Silverman, 1968) is low and tends to vary with recording electrode placement (Connolly and Gruzelier, 1982). Furthermore, in augmenting-reducing studies, where stimuli of varying intensities are employed, the rigid specification of time bands is bound to produce difficulties in peak identification, as the latencies of VEP peaks have been found to be inversely related to stimulus intensity (e.g., Creutzfeldt and Kuhnt, 1967). Indeed, the classification of subjects as augmenters or reducers varies markedly depending on the AEP components studied, and on the method of peak identification employed (Connolly and Gruzelier, 1982; Prescott et al., 1984).

A more flexible approach to peak identification, which is less dependent on the specification of time bands, has been proposed by Connolly and Gruzelier (1982). These researchers noted that the $N_1$ peak of the VEP was the most readily identified in recordings from vertex, occipital and temporal electrode placements. Thus, these researchers' approach to peak identification was to first identify the $N_1$ component, defined as the most negative point in the latency range of 80-180 msec. The positive peaks were then defined relative to the $N_1$ peak, with $P_1$ being the most positive peak within the first 60 msec before $N_1$ and $P_2$ being the most positive peak occurring between $N_1$ and 280 msec (Connolly and Gruzelier, 1982).

Three different methods of measuring peak amplitudes are common in the literature. Peak to peak amplitudes are measured as the vertical distance from the $P_1$ to the $N_1$ peaks ($P_1- N_1$) or from the $N_1$ to the $P_2$ peaks ($N_1- P_2$) (e.g., Buchsbaum and Pfefferbaum, 1971; Chen et al., 1985; Haier et al., 1984). Once the peaks have been identified, no measurement problems are involved in the assessment of peak to peak amplitudes. However, some evidence has been presented suggesting that the individual peaks involved in peak to peak measures may be differentially affected by increasing stimulus intensities (e.g., Connolly and Gruzelier, 1982; Prescott et al., 1984). The advantage of the peak to peak amplitude measure is that it does not
require the specification of a baseline (Donchin et al., 1977). In order to measure individual peaks, it is necessary to specify a baseline. Two methods have been employed for determining baselines. Some researchers have employed the mean of the entire AEP epoch as the baseline from which individual peak values are subtracted (e.g., Buchsbaum et al., 1981b; Lavine et al., 1976). The problem with this procedure is that since the amplitude of all peaks contributes to the baseline, measures of each individual peak relative to this baseline cannot be regarded as independent (Connolly and Gruzeller, 1982). Other studies report the use of a pre-stimulus baseline in the measurement of individual peak amplitudes (e.g., Buchsbaum and Davis, 1979; Connolly and Gruzeller, 1982; Davis et al., 1978). The pre-stimulus baseline is obtained by sampling the EEG, from the recording site, for a very short time just prior to stimulus onset. In this manner, independent measures of individual peak amplitudes may be obtained.

The amplitudes of AEP components identified and assessed in various manners, have been reported to be reliable across testing sessions (e.g., Buchsbaum, 1976; Soskis and Shagass, 1974; Stark and Norton, 1974). In particular, the amplitude / intensity slope has been reported to be most reliable (Stark and Norton 1974). However, this measure has been criticised on the grounds that the correlation between amplitude and intensity, for individual subjects, is often close to zero and that, therefore, to describe this relationship as a monotonically increasing or decreasing function is misleading (Connolly and Gruzeller, 1982; Iacono, Gabbay and Lykken, 1982).

8.1.3.4. The Role of Eye Blinks and Eye Movements.

A serious methodological deficiency which characterises much of the work on augmenting-reducing in the AEP is the failure to control for recording artifacts produced by eye blinks and eye movements (e.g., Buchsbaum et al., 1977, 1981b; Buchsbaum and Pfefferbaum, 1971; Buchsbaum and Silverman, 1968; Davis et al., 1978; von Knorring, 1978; von Knorring et al., 1974). This source of AEP contamination is particularly important in studies of the VEP, where eye blinks will alter the level of stimulation at the retina. Some researchers have attempted to overcome the problem by testing subjects with their eyes closed (e.g., von Knorring, 1978; von Knorring et al., 1974), but such a method has been criticised because requiring subjects to close their eyes, often generates slow-wave activity in the EEG, which can affect the AEP in an idiosyncratic manner (e.g., Cooper, Osselton and Shaw, 1974). It has also been pointed out that contaminating eye movements can still occur even when the eyes are closed, and that factors, such as the thickness of the eyelids and skin pigmentation, would affect the intensity of light at the retina (O'Toole and Iacono, 1987).
Iacono et al (1982) studied vertex P_j-N_j amplitude / intensity slopes calculated from VEP records contaminated with eye blinks, and compared these with the slopes calculated from records with eye blinks removed. Contaminated records were found to reveal smaller slopes than uncontaminated records, indicating that eye blinks could contribute to the phenomenon of reducing. It is, therefore, essential to control for eye blink artifacts in VEP augmenting-reducing studies.

Many AEP augmenting-reducing studies have employed somatosensory stimulation (e.g., Buchsbaum et al., 1977, 1981; Davis et al., 1978; Lavine et al., 1976) and have not employed any control for eye movements or eye blinks. However, Donchin et al (1977) have pointed out that any eye movement alters the orientation of the evoked potential field, and exerts an effect on scalp electrodes proportional to the distance of those electrodes from the eyes. Donchin et al (1977) note that vertex electrodes would certainly be affected by these artifacts. Accordingly, many researchers now monitor eye movements and eye blinks with the electro-oculogram (EOG), irrespective of stimulus modality, and exclude contaminated evoked potential recordings from the AEP (e.g., Connolly and Gruzelier, 1982; Prescott et al., 1984; Raine et al., 1981).

8.1.3.5. The Role of Attentional and Arousal Factors.

It has been suggested that factors such as the level of attention paid to the stimulus, and the subject's level of arousal may influence AEP components (e.g., Callaway, 1966; Hillyard and Picton, 1979). Indeed, it has been observed that when the VEP is studied by testing subjects repeatedly within a single testing session the amplitude / intensity slope for the P_j-N_j component is inversely related to increases in skin conductance (Birchall and Claridge, 1979). These authors concluded that augmenting-reducing in the VEP may be an inverse function of arousal levels. Nevertheless, researchers concerned with the phenomenon of augmenting-reducing, or with relating AEP components to pain mechanisms, have not attempted to consider the role of attentional or arousal factors in the interpretation of their results.

While the AEP has shown promise as a measure of sensory modulation, the wide range of methodologies employed and the absence of any clear convention in the identification and measurement of peak amplitudes, have resulted in this field becoming a rather confused area of psychophysiology. Nevertheless, since a deficiency in sensory modulation has been postulated to play a central role in the pathophysiology of headache (Sicuteri, 1981, 1982), it was considered worthwhile to explore the prospects for applying an AEP methodology to the study of this disorder.
8.1.4. AEP Amplitudes and Headache.

Very few studies of AEP parameters in headache sufferers have been conducted. Those reported in the literature have dealt almost exclusively with the response of migraineurs to visual stimulation.

VEP components studied in unilateral migraine sufferers during attacks have been reported to be smaller on the headache than on the headache-free side (Regan and Heron, 1969, 1970). These researchers attributed their findings to the presence of cerebral ischemia under the electrode.

Lehtonen (1974) compared VEP responses in migrainous and headache-free control subjects. None of the migraineurs had a headache at the time of testing. Subjects were exposed to 200 stimuli, presented either as single flashes or as flickering flashes at a variety of different rates. All stimuli were of a single intensity. Subjects were tested with eyes open and with eyes closed. The VEP was recorded from the left and right occipital regions and from points on either side of the vertex. Allowing for the number of different recording sites and experimental conditions employed, very few significant differences between groups were observed. In the case of single flashes with eyes open, only the amplitude of the positive component occurring at 170-260 msec (P_2), recorded from the right occipital electrode, was greater for migraineurs than for controls. For single flashes with eyes closed, the only significant difference to emerge was for the positive component occurring at 50-70 msec (P_1), recorded from the left occipital electrode. Again, migraineurs demonstrated larger amplitudes than controls. In the case of flickering stimulation, when subjects had their eyes closed, no significant differences between groups were apparent; when they had their eyes open, larger response amplitudes were observed for migraineurs at both occipital electrodes, but this occurred at only one of eight stimulus repetition rates (22 flicks per second). Unfortunately, Lehtonen (1974) did not state which VEP components demonstrated this response. The results are complicated further by the fact that Lehtonen measured peak to peak amplitudes, but reported separate amplitude measures for each peak. Peak to peak measurements incorporate information about both peaks. It is difficult to appreciate how information about individual peaks can be obtained from this method of measurement.

In a subsequent study, VEP amplitudes recorded from right and left occipital sites were pooled and compared across migraine and control subjects (Lehtonen, Hyyppa, Kaihola, Kangasniemi and Lang, 1979). Subjects were tested, with eyes open and with light flickering at 22 flicks per second. On this occasion, the negative component at 120-175 msec (N_1), and the positive component, at 170-260 msec (P_2) were smaller for migraine than for control subjects. These differences were consistent over four separate assessment sessions.
MacLean, Appenzeller, Cordaro and Rhodes (1975) studied the VEP recorded from the parietal and occipital regions. They did not say on which side of the head the parietal electrodes were placed. VEP's generated from 100 flashes of light were obtained for eight migraineurs and five headache-free control subjects. All subjects were tested with eyes closed; seven of the migraineurs were assessed during and between headaches. The authors reported that independent raters were unable to distinguish migraineurs, with or without headache, from the control subjects on the basis of VEP patterns.

Employing a reversing checkerboard pattern of light as the evoking stimulus, and recording the VEP from electrodes placed at the right, left and midline points in the occipital region, Kennard, Gawel, Rudolph and Rose (1978) tested subjects with eyes open and observed no differences on any VEP amplitude between migraineurs (assessed in the headache-free interval) and control subjects. For migraineurs reporting left sided headaches, the peak to peak amplitudes for a negative component occurring at 68-83 msec, and for a positive component occurring at 88-111 msec, were found to be greater on the left side of the head than on the right. The authors were unable to explain this finding.

Brinciotti, Guidetti, Matricardi and Cortesi (1986) studied the VEP in headache-free children and in children with common or classical migraine. Visual checkerboard stimulation was employed and recordings were made from $F_z$ (see Figure 8-2). The study was restricted to the $P_2$ amplitude which was found to be greater for the headache sufferers than for the controls. There was no significant difference between the classical and common migraineurs on this amplitude. The authors state that the $P_2$ amplitude was measured peak to peak, but they do not specify the reference peak (presumably it was $N_1$).

It is difficult to draw any conclusion from the studies reviewed above other than to say that the evidence for the involvement of VEP abnormalities in migraine is equivocal. Despite the fact that all of these studies employed visual stimulation, none controlled for EOG artifacts and the results obtained are, therefore, susceptible to the spurious influences described previously. As is the case with studies attempting to relate AEP components to pain perception, the variability in the methods applied to the study of the VEP in headache and headache-free control subjects is likely to be contributing to the absence of consistent findings.

The most thorough examination of the VEP in migraineurs and headache-free controls to date is that reported by Connolly, Gawel and Rose (1982). These researchers sought to explore the possibility that some deficiency in sensory modulation may characterise migraineurs by studying VEP components. They exposed 16 migraineurs and 22 headache-free control subjects to 6 different light intensities, each intensity being presented 60 times in blocks of 10. The blocks were presented in a randomised order. Migraineurs were assessed in
the headache-free state. Subjects were instructed to keep their eyes open, the EOG was monitored and recordings contaminated by eye movement artifacts were rejected. The VEP was recorded from the vertex and from bilateral temporal sites.

Connolly et al (1982) employed the method of Connolly and Gruzelier (1982), outlined in the previous section, in identifying the P1, N1 and P2 peaks. They measured the P1-N1 and N1-P2 peak to peak amplitudes, as well as the amplitude of each peak relative to a pre-stimulus baseline. They pooled their results across stimulus intensities and found that, for vertex recordings, the P1-N1 peak to peak amplitude and the amplitude of the individual N1 peak were greater for migraineurs than for controls. No other significant differences were observed for vertex recordings. Pooling the data for the temporal electrodes across both sides of the head and across all six intensities, the N1-P2 peak to peak amplitude was found to be greater for migraineurs than for controls. The P1 amplitude was smaller for the migraineurs whilst the N1 amplitude was larger. Temporal P1-N1 peak to peak amplitudes were greater for subjects with right sided headaches than for those with bilateral headaches. Those migraineurs with right sided headaches demonstrated larger P1-N1 amplitudes, at left temporal sites, than those with bilateral headaches.

It is unfortunate that Connolly et al (1982) did not present a between groups analysis for linear or quadratic trend across intensities (Kirk 1968), as this would have allowed for some evaluation of any differential rates of amplitude increase over stimulus intensity and, thus, afforded some integration of their findings with the literature on augmenting-reducing in the AEP.

Connolly et al (1982) were not able to articulate their results to a theory of headache couched in terms of a deficit in sensory modulation (e.g., Sicuteri, 1982; Sicuteri Anselmi and Del Bianco, 1978) because they focused specifically on migraineurs, many of whom are known to be particularly sensitive to light when they have a headache (e.g., Dalessio, 1980; Lance, 1982); and because they employed only photic stimulation. Thus, an interpretation in terms of some specific hypersensitivity in the visual system of migraineurs, rather than in terms of a general failure of sensory modulation, may account for their findings. In attempting to separate these two alternative interpretations, it would be important to examine the AEP response of headache sufferers and control subjects to stimuli in other modalities. Moreover, from the point of view of applying the AEP methodology to an examination of the central theory of headache (Sicuteri, 1982; Sicuteri, Anselmi and Del Bianco, 1978), it would be important to determine the extent to which any AEP abnormalities, identified in headache sufferers, could be related to measures of pain sensitivity. The identification of significant relationships between these variables would suggest that AEP abnormalities, such as those
reported by Connolly et al (1982), may reflect some neurophysiological deficiency in the CNS pain regulatory system.

8.1.5. Aims of Study III.

Study III sought to explore the prospects for extending the findings of Connolly et al (1982) so that they might have greater implications for the central theory of headache. Towards this end, it was proposed to study the SSEP in headache and headache-free control subjects; to assess pain sensitivity in each of these groups, so as to explore the premise that headache sufferers lack the capacity to modulate pain (Sicuteri et al., 1978); and to determine the strength of any relationship between AEP amplitudes and pain sensitivity.

As argued at the beginning of this chapter, some demonstration of the involvement of CNS mechanisms in the pathophysiology of headache would raise the possibility that the cognitive factors described in Chapter 8 may be related to headache through these processes and would, thus, have implications for the development of theoretical models linking cognitive factors with headache activity.

8.2. HYPOTHESES.

The experiment described in subsequent sections of this chapter was designed to test the following hypotheses:

1. Headache subjects tested between headaches will have lower pain thresholds and demonstrate reduced pain tolerances relative to headache-free control subjects. This hypothesis was derived from the central theory of headache (Sicuteri, Anselmi and Del Bianco, 1978), outlined in Chapter 2. If it is true that headache sufferers are deficient in their capacity to modulate their experience of pain (Sicuteri, Anselmi and Del Bianco, 1978), they should be found to be more pain sensitive than headache-free persons.

2. Headache sufferers will demonstrate lower pain thresholds and reduced pain tolerances during headache attacks, as compared with headache-free periods. This hypothesis follows from the view that any failure of pain modulation may not become evident until headache occurs (Sicuteri, 1982).
3. The $P_1-N_1$ amplitude of the SSEP, recorded from vertex, will be larger for headache sufferers, tested between attacks, than for headache-free controls. Furthermore, the rate at which the $P_1-N_1$ amplitude increases with stimulus intensity will be greater for headache subjects tested between attacks than for headache-free subjects. This hypothesis follows from the view that headache sufferers may be unable to modulate the impact of sensory input on their nervous systems because of some neurophysiological deficiency in the sensory modulating mechanism (Sicuteri, Anselmi and Del Bianco, 1978). If this theory were true, the amplitude of the SSEP would be expected to be larger for headache sufferers, and to increase with stimulus intensity at a rate greater for headache sufferers than for headache-free controls. Such patterns of responding have been observed for pain sensitive persons (e.g., Buchsbaum and Davis, 1979; Buchsbaum et al., 1977; Harkins and Chapman, 1978; von Knorring, 1978) and for persons given the opiate antagonist naloxone (e.g., Buchsbaum et al., 1977, 1981b). The choice of the $P_1-N_1$ component of the SSEP was based on the findings of Connolly et al (1982) with respect to the VEP.

4. The $P_1-N_1$ amplitude of the SSEP, recorded from vertex, will be greater and will increase at a greater rate with stimulus intensity for headache sufferers tested during headache attacks than for those same subjects tested between attacks. This hypothesis follows from the view that any disruption to the sensory modulating system may be specific to episodes of headache (Sicuteri, 1982).

5. The $P_1-N_1$ amplitude of the SSEP will be related to pain sensitivity such that, for both headache and control subjects, this component will be larger, and will increase at a greater rate with stimulus intensity, for pain sensitive than for pain insensitive subjects. This hypothesis follows from the assumption that augmenting in the $P_1-N_1$ amplitude of the AEP is indicative of a deficiency in central pain inhibitory functions (e.g., von Knorring, 1978; von Knorring et al., 1974, 1979).

8.3. METHOD.

8.3.1. Subjects.

The sample of subjects participating in the study comprised 36 headache sufferers and 36 headache-free control subjects, matched for age and sex with each of the headache
sufferers. Pairs of subjects were considered to be matched on age provided that each member of the pair was aged within five years of the other.

Headache sufferers were recruited from the Neurology Outpatients Clinic at the Prince Of Wales Hospital, Sydney (n = 21), from the staff of the Prince Of Wales and Prince Henry Hospitals and from the general community (n = 15). The latter group of subjects were recruited by means of advertisements placed on hospital and community notice boards.

As was the case in the Study II (see Chapter 8), the present study was not confined to any single headache type. The intention was to discover if the hypothesised differences between headache sufferers and control subjects would hold across the spectrum of headache from tension to migraine.

All prospective headache subjects were contacted by telephone and were accepted into the study if they met the criteria outlined in Chapter 7. In addition, they were required not to have taken any regular prophylactic medication for headache in the month preceding the study. It was considered that prophylactic medication could affect pain sensitivity and SSEP measures in unspecified ways. The sample of 36 headache sufferers satisfied the selection criteria, and consisted of 22 females and 14 males, aged between 21 and 63 years, with a mean age of 41.6 years (S.D. = 10.7 years), all of whom reported suffering with headache on at least one day in each week.

In view of the wide prevalence of Headache (see Chapter 1), it is difficult to find people who have not suffered a headache at some time or another. Accordingly, headache-free persons were defined operationally, as those who reported suffering no more than one mild headache per month and who denied the occurrence of any migrainous features (as set out in Section 7.6.4 of Chapter 7) in association with any such headaches.

Headache-free control subjects were recruited from the general community and from the staff of the Prince of Wales and Prince Henry Hospitals by means of notice board advertisements. All control subjects were required not to be taking any regular medication. Thirty six control subjects were selected such that they could be matched with at least one member of the headache group. The control group consisted of 22 females and 14 males aged between 18 and 65 years with a mean age of 41.0 years (S.D. = 13.6 years).

All subjects signed a statement giving their informed consent to take part in the study. They were assured that although the results of the study may be published, their identity would remain confidential.
In view of the fact that there exists, within the central theory of headache, some confusion over whether the postulated deficit in pain modulation is specific to the head (Sicuteri, 1982), or is a generalised condition affecting all regions of the body (Sicuteri, Anselmi and Del Bianco, 1978), it was decided to assess pain sensitivity in both the head and the index finger of the non-dominant hand.

Various procedures exist for inducing pain in the fingers, including the cold pressor test, the Forgione-Barber Pain Stimulator and electrical stimulation. These methods are reviewed in Appendix A. In the present study electrical stimulation was preferred because the stimuli can be delivered over a number of trials, thus making for a high level of reliability (e.g., Tursky and O’Connell, 1972; Wolff, 1978). Furthermore, pain induced in the human by electrical stimulation has been shown to be responsive to analgesics (e.g., Wolff, Kantor and Cohen, 1976; Wolff, Kantor, Jarvik and Laska, 1966), allowing some validity to be attached to the method.

Wolff and his colleagues (Wolff, 1978, 1984; Wolff et al., 1966, 1976) reported a method by which the subject receives electrical shocks by placing each of two fingers into one of two beakers, each filled with saline solution in which electrodes are submerged. This procedure has been reported to be highly reliable (Wolff, 1978). However, it was decided not to adopt this method in the present study, in view of the risk posed to subjects by the possibility of them inadvertently knocking the beakers over and spreading saline solution across the laboratory floor. Instead, copper wires fleeced with absorbent cotton material served as electrodes. The area around the first and second phalanx of the index finger on the subject’s non-dominant hand was rubbed with lime stone to remove any dirt or loose tissue and laced with conductive jelly. The indifferent electrode was wrapped around the first phalanx of the prepared finger; the active electrode was wrapped around the second phalanx. The experimenter ensured that the electrode placements were comfortable for all subjects. The electrodes were connected to a constant current stimulator (Medelec ST10 sensor), and then soaked with a 10% saline solution. The resultant impedance of the electrode-skin circuit was below 5000 Ohms. It has been noted that ratings of pain intensity are related consistently with the amperage of the stimulus when the impedance is maintained below 5000 Ohms (Tursky and Watson, 1964).
Shocks were delivered to subjects at the rate of one every two seconds. These stimuli were delivered as square wave pulses, measured in milli-amps (mA), with each pulse having a duration of one millisecond (msec).

The pain measures studied were the pain threshold and the pain tolerance, which have been widely employed in studies of experimental pain and which have also been found to be responsive to the effects of analgesics (e.g., Wolff et al., 1966, 1976; see also Appendix A). These measures were obtained by the method of limits (Wolff, 1978; Wolff and Horland, 1967). The experimenter increased the intensity of stimulation in one mA steps and noted the point at which the subject indicated that some sensation was present (ascending sensation threshold), the level at which the subject indicated that the sensation was painful (ascending pain threshold) and the point at which the subject stated that the pain was unbearable (pain tolerance). At this point the experimenter immediately reduced the shock intensity to a level half-way between the obtained tolerance and threshold levels. The intensity of shock was reduced in one mA steps and the experimenter noted the point at which the subject indicated that the sensation was no longer painful (descending pain threshold). Shock intensity continued to be reduced in this manner until the subject indicated the absence of any sensation (descending sensation threshold). At this point, the experimenter embarked on a second ascending trial. Five ascending and descending trials were conducted for each subject. The pain threshold and tolerance levels were defined as the mean of the shock intensities corresponding to each of these parameters over all ascending and descending trials (Wolff, 1978). The maximum intensity of shock delivered to any subject was set at 65 mA.

The instructions given to subjects were similar to those employed by Wolff and Horland (1967), and were as follows:

This test involves electrical stimulation of the fingers. There is no danger associated with the electricity because everything is carefully controlled and perfectly safe. I am going to take a number of measurements and repeat them five times. I would like you to give me five verbal responses which I shall now explain. When I first turn on the current, you will not feel anything. I shall slowly increase the current, and I want you to say "now" as soon as you begin to feel the slightest sensation, such as a faint touch or tickling sensation. As the current continues to increase, say "pain" as soon as the first sensation changes to pain. Concentrate carefully on the sensation in your finger and as soon as this changes into any kind of pain, ache or hurting sensation, say "pain" straight away. As the current continues to increase, I want you to say "stop" when the pain becomes unbearable. As soon as you say "stop", I will turn down the current. As the current decreases, I want you to say "pain gone" as soon as the sensation in your finger is no longer painful. Finally, say "all gone" as soon as all sensations of any kind have disappeared. I shall then begin to increase the current again and we will repeat the procedure. Remember to concentrate carefully on the sensation in your finger so that you can identify the changes as soon as they occur. You are free to terminate your involvement in this procedure at any stage if you so wish. Do you have any questions?
During the electrical pain procedure, each subject lay on a bed with his or her head supported by a pillow. Their non-dominant hand, with the electrodes attached to the index finger, was placed on a small plastic grid. At the commencement of each of the five pain measurement trials, the electrodes were soaked with saline solution with a syringe. Any excess saline solution was absorbed into a towel placed between the grid and the bed.

In a pilot study designed to determine the test-retest reliability of the electrical pain induction procedure 24 headache-free subjects (17 females and 7 males), aged between 21 and 57 years (mean age = 31.8 years; S.D. = 10.8 years), were tested on two occasions, each separated by a period of seven to fourteen days. The subjects were recruited from amongst the staff of the Prince Henry Hospital. Reliability was assessed by means of Pearson's correlation coefficient. For pain threshold, the reliability was 0.91 (2-tailed p < 0.01). For pain tolerance, the reliability was 0.95 (2-tailed p < 0.01). Thus, the electrical pain procedure employed in the present study demonstrated a high level of reliability for each of the pain measures. The reliability coefficients obtained were of a similar magnitude to those reported by previous researchers employing electrical stimulation (e.g., Tursky, 1974; Wolff, 1978).

8.3.2.2 Inducing Pain in the Head.

Few methods have been developed for inducing pain in the head. The most commonly employed stimulus is radiant heat. Pain is induced in the forehead by focusing a high intensity light beam onto a blackened surface of the skin (Hardy, Wolff and Goodell, 1952; Mor and Carmon, 1977). However, this procedure was not employed, as it carries the risk of blistering the flesh (Wolff, 1978). A second method of pain induction is offered by the pressure algometer (Keele, 1954). In this method, a flat tipped plunger attached to a pressure gauge is applied to the surface of the skin, until pain develops. This method was not selected because it has been reported to have a rather low level of reliability in comparison with other pain induction procedures (Wolff, 1977). Langemark and Oleson (1987) induced pain in headache and control subjects by palpating the various muscles of the cranium. However, the rate and pressure of palpation was uncontrolled, and the authors provided no data concerning the reliability of their method.

In view of the fact that existing methods for inducing pain in the head were deemed unsuitable, it was decided to develop another procedure. Immersing the vertex in cold water at a temperature below 18°C, has been reported to produce pain in the head (Wolf and Hardy, 1941). In a modification of this procedure, the experimenter and a colleague found that head pain could be induced to unbearable levels by holding a piece of ice against the temporal
region. Furthermore, it seemed that the level of pain and its rate of increase were independent of the degree of pressure with which the piece of ice was held against the skin.

Pieces of ice were produced by filling plastic satchels with distilled water and placing these on a perspex shelf located within the freezer compartment of a refrigerator. Distilled water was preferred to ordinary tap water as the latter is known to contain impurities that might have affected the rate at which the water was cooled. The temperature of the freezer was maintained at -17°C. Freezing distilled water in plastic satchels allowed the size and shape of the ice pieces to be standardised. The pieces of ice were rounded with one flat side (the side sitting on the shelf). The flat side had a diameter of 4.5 cm and each piece of ice consisted of 15 millilitres of distilled water.

From the point of view of applying stimuli of a consistent temperature to the temporal region, it was important to determine the length of time for which pieces of ice had to be kept in the freezer before their temperature became stable. Accordingly, a pilot study was conducted, whereby a thermometer was inserted into one of the water-filled satchels and placed inside the freezer. After periods of 6 and 24 hours, the temperature of the ice was noted to be -14°C. After 30 hours, the temperature of the ice was observed to be -14.5°C. A further inspection, 55 hours after incubation again revealed this temperature to be unchanged. Thus, the temperature of the ice was considered to have stabilised after 30 hours in the freezer. Only pieces of ice which had been placed in the freezer for at least 30 hours served as stimuli in the main experiment.

Subjects were seated in front of a chart recorder, through which the paper moved at 0.25 cm/sec. A steel rod was fitted across the chart recorder, and a moveable perspex frame, holding a pen perpendicular to the paper, was fitted onto the rod. The chart recorder was activated as soon as the flattened side of the piece of ice was placed against the subject's temple. The ice was held in place by the experimenter, and a towel was placed around the subject's neck so as to absorb droplets of water. The subject indicated the presence of pain (pain threshold) by making a movement of the pen towards the top of the paper. When the pain became unbearable (pain tolerance) the subject pushed the pen to the very top of the paper, and the experimenter removed the ice. Pain threshold and tolerance were measured in terms of the time taken (in seconds) to reach each of these points. The experimenter first assessed pain sensitivity in the right temple and then in the left temple. The procedure for each temple was discontinued either when subjects reached pain tolerance, or after 180 seconds had elapsed since the placement of the ice. Prior to the measurement of temporal pain sensitivity, subjects were issued with the following instructions:

In a moment I will hold a small piece of ice against your temple. Because of the cold, the resulting sensation may be unpleasant, but by no means harmful.
Initially, your temple will feel cold, but after a time a dull aching pain will develop. Concentrate carefully on the sensations in your temple, and notice when these change from cold to pain. As soon as this happens, begin moving the pen up the paper. As the pain develops, move the pen further up the paper. If the pain becomes unbearable, push the pen to the very top of the paper and I will remove the ice. Remember, do not start moving the pen up the paper until the sensation in your temple changes from cold to pain. Also remember that as soon as you push the pen to the very top of the paper, I shall remove the ice. You are free to terminate your involvement in this procedure at any stage if you so wish. Do you have any questions?

A pilot study was conducted to determine the test-retest reliability of the temporal ice pain measures and also to evaluate the effect on pain measures of varying the degree of pressure with which the ice was held against the temple. Twenty one headache-free subjects (12 females and 9 males) recruited from amongst the staff of Prince Henry Hospital, served as subjects. They were aged between 20 and 57 years (mean age = 35.6 years; S.D. = 13.0 years). These subjects were tested on two occasions, each separated by a period of seven to fourteen days. The level of pressure applied to the skin through the piece of ice was varied in a counter-balanced fashion, with 10 subjects receiving a low level of pressure on the first occasion and a higher level on the second occasion, and 11 subjects receiving these respective levels of pressure in the reverse order. As it was not possible to standardise the degree of pressure, this was simply varied by the experimenter in an ad hoc fashion, with the ice being held "gently" against the skin on one occasion, and "firmly" on another. The pain measures obtained in each of the "gentle" and "firm" conditions were compared separately for the right and left temples by means of the paired t-statistic (see Table 8-1). The test-retest reliability of the temporal ice pain measures was assessed by means of the Pearson correlation coefficient (see Table 8-2).
Table 8-1

Mean Threshold and Tolerance Scores for the "Gentle" and "Firm" Applications of the Temporal Ice Pain Test (N = 21).

<table>
<thead>
<tr>
<th>Pressure</th>
<th>Left Temple</th>
<th>Right Temple</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pressure Threshold</td>
<td>Tolerance</td>
</tr>
<tr>
<td>Firm</td>
<td>29.5 (40.8)</td>
<td>129.6 (65.8)</td>
</tr>
<tr>
<td>Gentle</td>
<td>20.0 (18.8)</td>
<td>126.6 (68.4)</td>
</tr>
<tr>
<td>Paired t-value</td>
<td>1.48</td>
<td>0.92</td>
</tr>
<tr>
<td>2-tailed p-value</td>
<td>N.S.</td>
<td>N.S.</td>
</tr>
</tbody>
</table>

Note: The standard deviations are entered in brackets. N.S. Indicates a result which fell short of the p < 0.05 significance level.

The results indicated that the degree of pressure with which the pieces of ice were applied to the temple, had no significant effect on the obtained threshold and tolerance measures.

Table 8-2

Reliability Coefficients for each Temporal Ice Pain Measure (N = 21).

<table>
<thead>
<tr>
<th>Pain Measure</th>
<th>r</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain Threshold:</td>
<td></td>
</tr>
<tr>
<td>Left Temple</td>
<td>0.63</td>
</tr>
<tr>
<td>Right Temple</td>
<td>0.91</td>
</tr>
<tr>
<td>Pain Tolerance:</td>
<td></td>
</tr>
<tr>
<td>Left Temple</td>
<td>0.98</td>
</tr>
<tr>
<td>Right Temple</td>
<td>0.94</td>
</tr>
</tbody>
</table>

All correlations in the above table were significant beyond the 2-tailed p < 0.01 level.
The results indicated that the temporal ice pain test demonstrated good reliability for both pain threshold and pain tolerance measures, despite the fact that a deliberate effort was made to vary the level of pressure applied over the two occasions.

8.3.3. SSEP Apparatus and Procedure.

8.3.3.1. Stimuli.

As noted previously in this chapter, there is little in the literature on augmenting-reducing in the AEP to guide the researcher in the choice of the number and intensity of stimuli to be employed.

In augmenting-reducing paradigms, where the intention is to calculate amplitude/intensity slopes, more than two stimulus intensities must be employed for obvious reasons. However, beyond this requirement, no rationale has been offered concerning the optimum number of stimulus intensities. Practical considerations restrict the number of stimulus intensities that can be employed. For example, if blocks of stimuli are to be presented in a randomized fashion and if the number of intensities exceeds the number of recording channels available, some of the averaged traces cannot be stored within the recording machine, and thus have to be stored on computer so that more channels are free to record evoked potentials at other stimulus intensities. As the number of different stimulus intensities increases the procedure of switching stored traces between the recording machine and the computer becomes progressively more cumbersome and time consuming. In the present study three channels were available for recording evoked potentials. It was considered that switching the trace for one intensity between the recording apparatus and the computer was not so demanding as to interfere with the conduct of the experiment. Thus, it was decided to employ four different stimulus intensities, this being the number used most commonly in SSEP studies of augmenting-reducing (e.g., Buchsbaum et al., 1977, 1981b; Davis et al., 1978; Lavine et al., 1976; Prescott et al., 1984).

It was considered necessary to define stimulus intensities in terms of stimulus parameters (e.g., Buchsbaum et al., 1977; Davis et al., 1978) rather than in terms of subjective intensity ratings (e.g., Bromm, 1984; Lavine et al., 1976), as the central theory of headache predicts that the latter measures would differ between the headache and control groups. If the parameters of the stimuli delivered to subjects differed consistently between the two groups, this could artificially produce differences in the SSEP. Furthermore, it was necessary to select stimuli such that the minimum intensity would be perceived by all subjects, and such that the
maximum intensity would be no more than slightly above the pain threshold for most subjects, thus avoiding the jumpy motor reactions which can contaminate SSEP recordings (Bromm, 1984).

A pilot study was conducted with the aim of identifying a stimulus intensity range that would meet the above criteria. Eighteen headache-free persons (11 females; 7 males), recruited from amongst the staff of the Prince Henry Hospital, served as subjects in a study designed to determine sensory threshold, pain threshold and pain tolerance levels for electrical stimulation. The age of the subjects ranged from 21 to 60 years (mean age = 28.1 years; S.D. = 9.6 years). The method employed was the same as that described in Section 8.3.2.1, except that the electrical pulse duration was 0.1 msec and the maximum shock intensity was 100 mA (the limit of the Medelec ST10 Sensor constant current stimulator).

The results obtained indicated that on each of the 5 ascending and descending trials, no subject was insensitive to stimulation at or above an intensity of 5 mA. Averaging over each of the five ascending and descending trials, pain threshold levels ranged from 17.6 mA to 43.8 mA (Mean = 28.3 mA; S.D. = 7.5 mA). Averaging over the five pain tolerance measures gave a range of 35.0 mA to 100.0 mA (Mean = 69.8 mA; S.D. = 20.6 mA).

In view of the findings listed above, it was decided to set the minimum stimulus intensity at 6 mA and to increase the intensity in steps of 6 mA's, up to a maximum of 24 mA. Thus, the four stimulus intensities employed were 6 mA, 12 mA, 18 mA and 24 mA. This ensured that the stimulus intensities were equally spaced and that, in the main experiment, there would be a very high probability that the minimum shock would be perceived by all subjects and that the maximum shock would be well below the pain tolerance level for most subjects.

It has been recommended that in AEP studies, the delivery of stimulus intensities should be randomized so as to maintain the subject's arousal at a constant level (Bromm, 1984). Accordingly, investigators have either completely randomized the stimuli (e.g., Buchsbaum et al., 1981b; Davis et al., 1978), or have presented stimuli in blocks of one intensity at a time, and randomized the order of blocks, there being several blocks of each intensity (e.g., Birchall and Claridge, 1979; Connolly et al., 1982).

The number of evoked potentials employed in the generation of an AEP wave varies from one research group to another. Ashton et al (1984) used 30 evoked potentials, while Birchall and Claridge (1979) used 128 separate waves. Callaway (1966) suggested that, in the interests of obtaining an AEP with clearly defined components, 36 individual evoked potentials should be averaged. Most studies have derived the AEP from approximately 60
separate wave forms (e.g., Buchsbaum et al., 1981b; Connolly et al. 1982; Haier, 1983; Prescott et al., 1984). This latter procedure was followed in the present study with a sufficient number of each stimulus intensity being presented to generate 64 corresponding evoked potentials, these being averaged by computer. Since trials contaminated by large muscle movements, eye blinks and eye and eye movements were rejected, the actual number of stimulus presentations varied slightly from subject to subject.

In the present study, each of the four stimulus intensities was divided into four blocks, there being a minimum of 16 stimuli within each block (additional stimuli were added as required when trials were rejected) and a total of 16 blocks. The order of presentation of the blocks was randomized, the only restriction being that no two blocks of the same intensity were permitted to be presented consecutively (Birchall and Claridge, 1979). The inter-stimulus interval was set at one second (Birchall and Claridge, 1979; Buchsbaum et al., 1981b; Haier, 1983) and the interval between blocks ranged from 5 to 15 seconds (Connolly et al., 1982; Connolly and Gruzelier, 1982; Prescott et al., 1984).

Stimuli were delivered to the index finger of the non-dominant hand in the manner described in Section 8.3.2.1. The stimulus electrodes were soaked with saline solution prior to stimulation and they were soaked again at intervals of four stimulus blocks. In a post-experiment interview all subjects indicated that they felt the 6 mA stimulus.

8.3.3.2. SSEP Recording.

In view of the fact that Connolly et al (1982) observed differences between migraineurs and headache-free control subjects on the P\textsubscript{j}-N\textsubscript{j} component of the VEP recorded from vertex, and since the SSEP has been reported to be generally larger at this site than at other scalp locations (Goff et al., 1977), it was decided to record the SSEP from vertex (C\textsubscript{z}; see Figure 8-2).

It has been noted that chlorided silver electrodes are more sensitive to changes in electrical potential and have a lower level of impedance than platinum alloy or stainless steel electrodes (Cooper, Ossleton and Shaw, 1980). Thus, it was decided to record the SSEP with chlorided silver cup electrodes. As the subject lay down on a bed, with head supported by a pillow, the experimenter located the vertex, rubbed the area with lime stone to remove loose skin and cleaned this region with antiseptic. Conductive jelly was placed inside the cup electrode and this electrode was fitted onto the scalp by means of collodion glue. The reference electrode was fitted to the ear lobe on the side contralateral to the finger being stimulated. After cleaning the ear lobe with antiseptic, the reference electrode was fitted to this region in
the manner described for the vertex electrode. The ear lobe has been regarded as an indifferent site by many researchers (e.g., Buchsbaum et al., 1981b; Buchsbaum and Silverman, 1968; Connolly et al., 1982; Haier, 1983; Prescott et al., 1984).

As discussed previously in this chapter, eye blinks and eye movements are a potential source of artifact in AEP research (Cooper et al., 1980; Donchin et al., 1977). Accordingly, it was decided to monitor eye blinks and eye movements by means of the electro-oculogram (EOG). Chlorided silver cup electrodes were placed supraorbitally and over the outer canthus of the left eye to monitor both vertical and horizontal eye movements (Connolly and Kleinman, 1978). These electrodes were fitted in the manner described for the vertex electrode. Thus, one channel of the recording machine was used to monitor the EOG while the remaining three channels were available for recording evoked potentials from vertex.

Trials on which muscle movements generated potentials greater than 100 micro-volts (uV) at vertex were rejected online by the recording machine, as were trials on which eye blinks or eye movements generated potentials greater than 100 micro-volts in the EOG. (Connolly et al., 1982; Connolly and Gruzelier, 1982; Prescott et al., 1984). So as to minimise the occurrence of these artifacts, every effort was made to ensure that subjects were comfortable; they were asked not blink during periods when their finger was being stimulated and they were required to focus their vision on a small black spot on a white screen placed at a distance of 800 cm from their eyes, adjusted to a comfortable height. These precautions are recommended by Cooper et al (1980).

Prior to the commencement of SSEP recording, the experimenter checked the impedance of all electrodes. Those electrodes with an impedance greater than 5000 Ohms were removed and re-applied until the impedance of all electrodes was below 5000 Ohms (Cooper et al., 1980).

The four electrodes employed during recording were attached to a four channel Medelec connector box and, from there, electrical potentials were relayed to a Medelec four channel recorder where they were amplified, averaged and stored.

In order to reduce the noise present in AEP averages to the point where specific components can be readily identified, it is necessary to filter each individual evoked potential prior to on-line averaging (Kaskey et al., 1980). In the interest of obtaining an AEP wave with clearly defined components, it has been pointed out that the sampling rate should be greater than the frequency range allowed to pass through the filter (Cooper et al., 1980). In the present study, the EEG recorded from vertex was sampled at an interval of one msec (500 Hz). Thus, the EEG was filtered within the range 1-125 Hz. This range is referred to as the band width
(Cooper et al., 1980). The band width chosen in the present study was similar to that employed by previous researchers (e.g., Chapman et al., 1981; Gortelmeyer and Zimmerman, 1984).

The recording machine was time-locked to the Medelec ST10 constant current stimulator so that the recording of each evoked potential commenced immediately after the stimulus presentation and continued for 300 msec. Thus, all SSEP’s were 300 msec in length, this length being necessary for the identification of all SSEP components through to $P_2$.

The recording machine was connected to an Apple 2C Duo Disk Drive Computer, on which all SSEP’s were stored for analysis. During the recording procedure itself, the SSEP’s obtained after each 6 mA stimulus block were stored on the computer, so as to leave three channels available for the remaining stimulus intensities. The 12, 18 or 24 mA stimulus block SSEP’s recorded immediately prior to the presentation of the second, third and fourth 6 mA blocks were transferred to the computer before recalling the 6 mA SSEP’s to the recording machine.

All subjects were earthed by means of a lead electrode fleeced with cotton and soaked in saline solution which was strapped to the forearm, contralateral to the finger being stimulated. This electrode was then attached to the Medelec connector box.

The SSEP recordings were conducted in the Evoked Potential Laboratory in the Department of Neurology at Prince Henry Hospital, Sydney. The illumination within the room was maintained at a constant level. Blinds were pulled down over the windows and a 60 watt electric light was placed behind the subject.

All subjects were told that they could terminate the procedure at any stage if they so wished.

8.3.4. General Procedure.

Prior to the experiment, subjects were told that the study was concerned with attempting to discover whether there exists any physiological cause for headaches. They were briefed on the procedures involved and required to sign a form giving their informed consent to participate. Headache and control subjects were interviewed about their headache activity, in the manner described in Section 7.5.2.

All subjects were assessed individually. Headache subjects (in the headache-free state) and control subjects first took part in the temporal ice pain test described in Section 8.3.2.2.
These subjects were then taken to the Evoked Potential Laboratory and took part in the electrical finger pain test. The experimenter then explained the SSEP procedure and administered the State Anxiety Scale (Spielberger et al., 1970). This questionnaire was administered in order to check on the possibility that differential levels of arousal existed between the headache and control subjects, as this factor has been found to influence AEP amplitudes (Birchall and Claridge, 1979). The SSEP procedure was carried out in the manner described in Sections 8.3.3.1 and 8.3.3.2. Subjects were then disconnected from the SSEP apparatus and thanked for their co-operation. Headache sufferers were asked to contact the experimenter at the start of a headache and to make arrangements to attend the laboratory during that attack. They were asked not to take any medication for the headache until the experimental procedures had been completed.

Sixteen headache subjects presented in a headache state. The procedures outlined above were repeated for these subjects, except that the temporal ice pain test was not administered. It was considered that subjects with headache may find it difficult to discriminate the pain induced by the ice test from the headache which they were already experiencing.

8.3.5. Assessment of SSEP Component Amplitudes.

The SSEP recording apparatus employed in the present study did not have the facility for recording a pre-stimulus EEG. Thus, it was not possible to measure individual peak amplitudes. The recording machine arbitrarily set the baseline to zero micro-volts one micro-second prior to the recording of each evoked potential. Since there was no way of determining the pre-stimulus baseline EEG for each evoked potential, it would have been meaningless to attempt to measure individual peak amplitudes relative to the arbitrary zero level. Accordingly, peak amplitudes were measured (in micro-volts) peak to peak for the $P_j-N_j$ and $N_1-P_2$ components.

The SSEP components, $P_1$, $N_1$ and $P_2$, were identified by means of the procedure described by Connolly and Gruzelier (1982) and employed by Connolly et al (1982) in their study of the VEP in migraineurs and control subjects. This method was discussed in Section 8.1.3.3., and was preferred to the time band method (Buchsbaum and Pfefferbaum, 1971; Buchsbaum et al., 1981b), as AEP components are frequently observed to fall outside the time bands stipulated (e.g., Connolly and Gruzelier, 1982; Prescott et al., 1984). Following Connolly and Gruzelier (1982), the $N_1$ component was identified first and defined as the most negative point in the latency range 80-180 msec; $P_1$ was defined as the most positive point occurring between 60 msec and $N_1$ and $P_2$ as the most positive point occurring between $N_1$ and 280 msec.
When each completed SSEP (64 trials) was recalled from the computer, it was presented on an oscilloscope. The peak to peak amplitudes (in micro-volts) were displayed on the oscilloscope after placement of one of two moveable cursors at each peak. Thus, peak to peak amplitudes were determined by placing one cursor at $N_1$ and the other at either $P_1$ or $P_2$, and observing the vertical distance between these cursors as displayed on the oscilloscope. Separate measurements were taken for each of the four SSEP's obtained for each subject.

The test-retest reliability of the $P_1-N_1$ and $N_1-P_2$ SSEP amplitudes was determined by means of a pilot study. Twelve headache-free subjects (7 females and 5 males), recruited from amongst the staff of Prince Henry Hospital, served as subjects. They were aged between 22 and 57 years (mean age = 40.7 years; S.D. = 13.3 years). These subjects were tested on two occasions, each separated by a period of seven to fourteen days. Reliability was assessed by calculating the Pearson correlation coefficient between the values obtained on the two occasions for each SSEP measure. The results indicated that the component amplitude measures, derived from the SSEP procedure, are reliable when an individual is tested on two separate occasions (see Table 8-3).

Table 8-3

<table>
<thead>
<tr>
<th>Stimulus Intensity (mA)</th>
<th>SSEP Components</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$P_1-N_1$</td>
</tr>
<tr>
<td>6</td>
<td>0.64</td>
</tr>
<tr>
<td>12</td>
<td>0.61</td>
</tr>
<tr>
<td>18</td>
<td>0.78</td>
</tr>
<tr>
<td>24</td>
<td>0.80</td>
</tr>
</tbody>
</table>

The reliability coefficients were of a magnitude similar to that reported by previous researchers (e.g., Buchsbaum et al., 1981b; Stark and Norton, 1974) and were all significant beyond the 2-tailed $p < 0.05$ level.
8.4. RESULTS.

8.4.1. Planned Analyses.

The Type I error rate for the following statistical analyses was set at 0.05, unless stated otherwise.

Hypothesis 1.

In this hypothesis it was predicted that headache subjects, tested between attacks, would have lower pain threshold and tolerance levels than headache-free control subjects.

The dependent variables were electrical finger pain threshold and tolerance, and temporal ice pain threshold and tolerance. Temporal ice pain threshold was taken as the mean of the threshold measures observed for each side of the head. Similarly, temporal ice pain tolerance was taken as the mean of the tolerance measures observed for each temple.

Since headache and control subjects were matched for age and sex it was intended to examine the hypothesis by conducting a paired t-test on each of the four pain measures. However, inspection of the data obtained for the temporal ice pain tolerance measure indicated that 28 control subjects and 13 headache sufferers reached the 180 second tolerance limit imposed on the test. Given data with such a skewed distribution, it was decided to examine differences between groups on their tolerance for ice pain by means of the distribution-free Wilcoxon matched-pairs signed ranks test (Welkowitz et al., 1976). After subtracting the tolerance level for each headache subject from that of their respective matched control subject, and assigning ranks to the difference scores, the positive ranks totalled 323 whilst the negative ranks totalled only 27. The obtained z-value was 3.75, indicating that control subjects had significantly greater tolerance levels on the temporal ice pain test than their headache counterparts (one-tailed p = 0.0002).

The significance of differences between groups on the remaining pain measures was evaluated with one-tailed paired t-tests. The results indicated that headache subjects were more pain sensitive than controls on all pain measures, except the threshold measure obtained from the temporal ice pain test, for which there was no significant difference between groups (see Table 8-4).
Table 8-4

Paired T-Test Analysis of Pain Measures (Number of Matched Pairs = 36).

<table>
<thead>
<tr>
<th>Pain Measure</th>
<th>Control Mean</th>
<th>Headache Mean</th>
<th>t-value (df=35)</th>
<th>1-tailed p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ice Threshold</td>
<td>28.5 (29.8)</td>
<td>23.9 (37.6)</td>
<td>0.54</td>
<td>N.S.</td>
</tr>
<tr>
<td>Electrical Threshold</td>
<td>17.2 (7.4)</td>
<td>12.8 (5.6)</td>
<td>3.21</td>
<td>0.002</td>
</tr>
<tr>
<td>Electrical Tolerance</td>
<td>35.4 (15.4)</td>
<td>25.8 (11.3)</td>
<td>3.52</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Note: In the above table and subsequent tables, N.S. indicates a result which fell short of the p < 0.05 level and the standard deviations are entered in brackets.

Hypothesis 2.

It was hypothesised that headache sufferers would be more sensitive to electrical finger pain during than between attacks. Sixteen headache subjects were assessed in headache and headache-free states. One-tailed paired t-tests indicated no significant differences between conditions on electrical pain threshold or tolerance. Thus, the results did not support Hypothesis 2 (see Table 8-5).


TABLE 8-5

Paired T-Test Analysis of Electrical Pain Measures Obtained During and Between Headache Attacks (N = 16).

<table>
<thead>
<tr>
<th>Pain Measure</th>
<th>Headache Absent Mean</th>
<th>Headache Present Mean</th>
<th>Paired t-value (df=15)</th>
<th>1-tailed p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Electrical Threshold</td>
<td>13.8 (6.5)</td>
<td>14.0 (6.5)</td>
<td>0.34</td>
<td>N.S.</td>
</tr>
<tr>
<td>Electrical Tolerance</td>
<td>27.0 (10.0)</td>
<td>25.7 (11.2)</td>
<td>0.95</td>
<td>N.S.</td>
</tr>
</tbody>
</table>

Hypothesis 3.

It was hypothesised that the $P_1-N_1$ amplitude of the SSEP would be larger for headache sufferers than control subjects and that the size of this amplitude would increase with stimulus intensity at a rate greater for headache subjects than controls.

The analysis of SSEP amplitudes involved a repeated measures factor (Stimulus Intensity with four levels). It has been pointed out that the use of pooled error univariate F-tests to test the significance of the within subjects main effect and the interaction of between and within subjects factors assumes that the correlations between pairs of levels, on the repeated measures factor, are equal and that the variances are equal across the levels of this factor (Jaccard and Ackerman, 1985; Vasey and Thayer, 1987). Repeated measures designs rarely meet these assumptions (Rogan, Keselman and Mendoza, 1979) with the typical consequence of using the pooled error term being an inflation of Type I errors (Jaccard and Ackerman, 1985; Vasey and Thayer, 1987). Accordingly, it has been proposed that within subjects effects and interactions should be assessed by specifying particular contrasts for each and testing these for significance with separate (rather than pooled) error terms (Boik, 1981; Rogan et al., 1979). Thus, given that the present study was concerned with differences between groups on the rate at which SSEP amplitudes increase with stimulus intensity, it was decided to examine the Intensity effect and Group x Intensity interactions on linear trend, with separate error terms. The main effect of Intensity was not of major theoretical interest, so these results are confined to Appendix K. Group x Intensity interactions and main effects of Intensity on quadratic and cubic trend (each determined with separate error terms) are also presented in Appendix K.
The experimental design for the analysis of the $P_1-N_1$ amplitude conformed to a 2 x 4 split-plot design (Kirk, 1968). The two factors were as follows:

Factor A:

- Group with 2 levels:
  - Headache
  - Control

Factor B:

- Stimulus Intensity with 4 levels:
  - 6 mA
  - 12 mA
  - 18 mA
  - 24 mA

Headache and control subjects were matched in pairs, thus imposing a block structure on the design and minimising any effect of subject heterogeneity. Each subject received all levels of the stimulus Intensity Factor.

The design was analysed by means of a two-way analysis of variance (ANOVA) with repeated measures on the Stimulus Intensity Factor. The ANOVA revealed a significant main effect for Group ($F(1, 35) = 7.82; p = 0.008$), with $P_1-N_1$ amplitudes being larger for headache (Mean = 5.53; S.D. = 2.93) than for control subjects (Mean = 4.10; S.D. = 1.50). The ANOVA also revealed a significant Group x Intensity interaction on linear trend ($F(1, 35) = 8.59; p = 0.006$). Inspection of Figure 8-3 indicates that $P_1-N_1$ amplitudes increased more rapidly with stimulus intensity for headache than for control subjects. Thus, the results supported Hypothesis 3. The ANOVA summary table is presented in Appendix K-1.

A between groups comparison of State-Anxiety scores with a paired t-test revealed that the headache (Mean = 38.92; S.D. = 9.80) and control (Mean = 36.33; S.D. = 9.84) groups did not differ significantly on this variable ($t(35) = 1.30; 2$-tailed $p > 0.05$) and argues against an interpretation of the significant $P_1-N_1$ difference between groups in terms of factors such as differential arousal levels (Birchall and Claridge, 1979).

**Hypothesis 4.**

It was hypothesised that the $P_1-N_1$ amplitude of the SSEP would be larger, and would increase at a greater rate with stimulus intensity for headache subjects, assessed during headache attacks, than for those same subjects assessed when headache-free. Sixteen headache subjects were assessed in headache and headache-free states.
FIGURE 8-3

$P_1-N_1$ Mean Amplitudes for Headache Sufferers and Control Subjects.

- □ Headache
- △ Control
The experimental design for the analysis of the $P_1-N_1$ amplitude conformed to a 2 x 4 split plot design (Kirk, 1968). The two factors were as follows:

Factor A:

Status with 2 levels:
- Headache
- Headache-free

Factor B:

Stimulus Intensity with 4 levels:
- 6 mA
- 12 mA
- 18 mA
- 24 mA

Each subject was tested in a headache and headache-free condition. Within each of these conditions all four stimulus intensity levels were presented.

The design was analysed by means of a two-way ANOVA, with repeated measures on the Stimulus Intensity factor. The analysis revealed no significant main effect for Status ($F(1, 15) = 0.18; p > 0.05$) and no significant Status x Intensity interaction on linear trend ($F(1, 15) = 0.04; p > 0.05$). The ANOVA summary table is presented in Appendix K-2.

It was concluded that there were no significant differences between the headache (Mean = 6.22; S.D. = 2.38) and headache-free (Mean = 6.42; S.D. = 3.14) conditions on $P_1-N_1$ amplitudes, or on the rate at which these amplitudes increased with stimulus intensity (see Figure 8-4). Hence, the results obtained did not support Hypothesis 4.

**Hypothesis 5.**

It was hypothesised that the larger the $P_1-N_1$ amplitude, and the greater the rate at which this component increased with stimulus intensity, the more pain sensitive subjects would be. This relationship could have been examined by calculating the $P_1-N_1$ amplitude / intensity slope for each subject and correlating these values with pain sensitivity measures. Previous researchers have reported significant correlations between amplitude / intensity slopes and measures of pain sensitivity (e.g., Buchsbaum and Davis, 1979; Buchsbaum et al., 1977; von Knorring, 1978). However, it was decided to avoid calculating amplitude / intensity slopes in view of the fact that, for individual subjects, these measures often give a very misleading impression of the behaviour of the AEP amplitude over the range of stimulus intensities (Connolly and Gruzelier, 1982; Iacono et al., 1982). Instead, it was decided to divide the headache and control subjects into pain sensitive and pain insensitive groups and to...
FIGURE 8-4

P1-N1 Mean Amplitudes for Headache Subjects Tested During and Between Attacks.

- With Headache
- Without Headache
test for differences between these latter two groups on the mean $P_1-N_1$ amplitude and for differences on linear trend for this component over increasing stimulus intensities. Separate analyses were conducted for the headache and control subjects.

Within the headache and control groups, subjects were classified as having a "high" or "low" pain threshold or pain tolerance level on each of the two pain tests. For the electrical finger pain measures and for the temporal ice pain threshold measure, this was achieved by means of a median split of the obtained values. In view of the fact that, on the temporal ice pain tolerance measure, a large number of subjects in both the headache and control groups reached the tolerance time limit of the test, thus imposing a ceiling effect on the values, it was decided to classify all subjects reaching this limit as having a "high" tolerance, and all those who failed to reach the limit as having a "low" tolerance. Within the headache and control groups, subjects scoring "high" on at least three of the four measures were classified as pain insensitive, whilst those scoring "low" on at least three of the four measures were classified as pain sensitive. Unclassified subjects were not included in the analysis. For the headache subjects, this procedure resulted in there being 13 pain sensitive subjects and 10 pain insensitive subjects, 11 subjects being unclassified. For the control subjects, there were 13 pain sensitive subjects and 15 pain insensitive subjects, 11 subjects being unclassified.

The experimental design for the analysis of the $P_1-N_1$ amplitude conformed to a 2 x 4 split-plot design (Kirk, 1968). The two factors were as follows:

Factor A:
- Pain Sensitivity with 2 levels:
  - Pain Sensitive
  - Pain Insensitive

Factor B:
- Stimulus Intensity with 4 levels:
  - 6 mA
  - 12 mA
  - 18 mA
  - 24 mA

The design was analysed by means of a two-way ANOVA with repeated measures on the Stimulus Intensity factor. For the headache subjects ($n = 23$) the ANOVA revealed no significant main effect for pain sensitivity ($F (1, 21) = 0.04; p > 0.05$), there being no significant difference between pain sensitive (Mean = 5.58; S.D. = 2.19) and pain insensitive headache subjects (Mean = 5.83; S.D. = 3.40) on the size of $P_1-N_1$ amplitudes. The ANOVA also revealed no significant Pain Sensitivity x Intensity interaction on linear trend ($F (1, 21) = 0.15; p > 0.05$) (see Figure 8-5). The ANOVA summary table is presented in Appendix K-3.
FIGURE 8-5

$P_1-N_1$ Mean Amplitudes for Pain Sensitive and Pain Insensitive Headache Subjects.

- ■ Pain Sensitive
- △ Pain Insensitive
Similar results were obtained for control subjects (n = 28). There was no significant main effect for Pain Sensitivity (F (1, 26) = 0.01; p > 0.05), there being no significant difference between pain sensitive (Mean = 3.81; S.D. = 1.22) and pain insensitive control subjects (Mean = 3.86; S.D. = 1.29) on the size of P1-N1 amplitudes. The ANOVA also revealed no significant Pain Sensitivity x Intensity interaction on linear trend (F (1, 26) = 0.16; p > 0.05) (see Figure 8-6). The ANOVA summary table is presented in Appendix K-3.

The results did not support the hypothesis that P1-N1 amplitudes would be larger and increase more rapidly with stimulus intensity for pain sensitive than for pain insensitive subjects (Hypothesis 5).

8.4.2. Supplementary Analyses.

The Type I error rate for the following statistical analyses was set at 0.05, unless stated otherwise.

**Analysis 1.**

In order to investigate whether or not the sensitivity of headache subjects to ice pain varied with the site of headache, it was decided to identify subjects reporting unilateral headaches affecting the temporal region, and compare pain sensitivity on the side affected habitually by headache with that unaffected by headache. Thirteen subjects reported headaches affecting only one side of the head, including the temporal region. A paired t-test revealed that temporal ice pain thresholds obtained from the side affected habitually by headache (Mean = 12.1; S.D. = 14.9) were not significantly different from those obtained from the unaffected side (Mean = 10.7; S.D. = 7.9) (t (12) = 0.47; 2-tailed p > 0.05).

The data for the temporal ice pain tolerance measure were found to be very skewed. Since parametric tests, such as the paired t-test, assume a normal distribution, it was decided to employ the distribution-free Wilcoxon matched pairs signed ranks test to test for differences between the two sides of the head on pain tolerance. After subtracting the ice pain tolerance score for the temporal region on the unaffected side of the head from that obtained for the side affected habitually by headache and assigning ranks to the difference scores (Welkowitz et al., 1976), the positive ranks totalled 20 and the negative ranks totalled 18. The z-value obtained was 0.28 which was not significant (2-tailed p > 0.05).
FIGURE 8-6

$P_1-N_1$ Mean Amplitudes for Pain Sensitive and Pain Insensitive Control Subjects.
The above results indicate that there was no significant differential influence of the reported site of headache on pain sensitivity in unilateral headache subjects, as assessed by the temporal ice pain test.

**Analysis 2.**

Any effect of headache type on the pain measures was investigated by identifying migraine and tension headache subjects in the manner described in Chapter 7. As a result of this procedure, 13 headache subjects fell into the migraine category and 15 fell into the tension headache category. Only eight subjects fell into the tension-vascular category. The latter falls between the tension and migraine extremities of the headache severity continuum (Bakal and Kaganov, 1979; Holroyd and Andrasik, 1982a), and would, thus, not be expected to contribute substantially to the identification of differences in psychophysiological responses along this continuum. Accordingly, it was decided not to include the small number of subjects falling into the tension-vascular category in the analysis of the effects of headache type on the variables under investigation.

In view of the ceiling effect evident in the data obtained on the temporal ice pain tolerance measure, it was decided to examine differences between the migraine, tension headache, and control groups on this variable, by means of the distribution-free Kruskal Wallis analysis of variance, corrected for tied ranks (Siegel, 1956). In this procedure, each subject is ranked according to their score on the dependent variable. These ranks are then treated as the units of analysis. The analysis resulted in a chi-square value of 14.38 with two degrees of freedom (p < 0.001).

Comparisons between mean ranks for each pair of groups were carried out with Mann-Whitney U-Tests corrected for ties, as suggested by Kirk (1968). The family-wise error rate was controlled by means of Dunn's procedure, whereby the Type I error rate is divided by the number of comparisons made (Kirk, 1968). Thus, the Type I error rate was set at 0.017 (ie., 0.05 / 3). The Mann-Whitney U-Tests revealed the mean pain tolerance rank for controls (39.36) to be significantly greater than that for both the tension (25.13) (z = 2.96; 2-tailed p = 0.003) and migraine (22.00) (z = 3.39; 2-tailed p = 0.0007) groups. No significant difference between the tension and migraine groups was observed (z = 0.45; p = 0.65).

The significance of differences between the tension, migraine and control groups on the remaining pain measures was evaluated by means of one-way analyses of variance. When a significant F-value was obtained, _a posteriori_ comparisons between all pairs of means were conducted with Scheffe's procedure (Kirk, 1968). Significant group effects were observed on
electrical pain threshold ($F(2, 61) = 4.11; p = 0.021$) and on electrical pain tolerance ($F(2, 61) = 6.51; p = 0.003$) but not on temporal ice pain threshold ($F(2, 61) = 1.43; p > 0.05$) (see Appendix L for the ANOVA summary tables). Scheffé's procedure revealed the control subjects to have significantly higher electrical pain thresholds and tolerances than either of the headache groups who did not differ significantly from one another (see Table 8-6).

**Table 8-6**

Means on Ice Pain Threshold and Electrical Pain Threshold and Tolerance For Tension, Migraine and Control Groups.

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>Ice Threshold</th>
<th>Electrical Threshold</th>
<th>Electrical Tolerance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tension</td>
<td>15</td>
<td>30.5 (45.3)</td>
<td>12.7 (5.6)</td>
<td>22.6 (8.4)</td>
</tr>
<tr>
<td>Migraine</td>
<td>13</td>
<td>12.4 (13.7)</td>
<td>12.0 (6.1)</td>
<td>24.7 (10.2)</td>
</tr>
<tr>
<td>Control</td>
<td>36</td>
<td>28.5 (29.8)</td>
<td>17.2* (7.4)</td>
<td>35.4* (15.4)</td>
</tr>
</tbody>
</table>

*Indicates that, for electrical pain threshold and tolerance, the control group means were significantly greater than those for the two headache groups, as revealed by Scheffé's procedure.

On electrical pain threshold, the differences between the means for the tension and migraine, tension and control, and between the migraine and control groups were required to exceed 6.43, 5.22 and 2.38 respectively, in order to reach the 0.05 level of significance as assessed by Scheffé's procedure. On electrical pain tolerance these differences were required to exceed 12.50, 10.14 and 10.67. The critical $F$-value was given by $2 F_{0.05}(2, 61) = 6.30$. In the absence of a significant $F$-value for the ice threshold measure, no pairwise comparisons were made.

**Analysis 3.**

In order to determine whether or not there was any significant effect of headache type on $P_1-N_1$ mean amplitudes, or on the rate at which these amplitudes increased with stimulus intensity, the tension, migraine and control groups were compared on this variable.
The experimental design for the analysis conformed to a 3 x 4 split plot design (Kirk, 1968). The two factors were as follows:

Factor A:

Group with 3 levels: 
- Tension Headache
- Migraine Headache
- Control

Factor B:

Stimulus Intensity with the four levels given previously.

There were 13 migraineurs, 15 tension headache subjects and 36 control subjects. The design was analysed by means of a two-way ANOVA, with repeated measures on the Stimulus Intensity factor. The ANOVA revealed a significant main effect for Group (F (2, 61) = 5.71; p = 0.005) and a significant Group x Intensity interaction on linear (F (2, 61) = 3.51; p < 0.05) and quadratic trend (F (2, 61) = 5.30; p < 0.01) (see Appendix K-4 for the ANOVA summary table).

The significant Group effect was explored by testing for differences between means with Scheffe's procedure. The tension headache subjects were observed to have significantly larger P1-N1 amplitudes (Mean = 6.43; S.D. = 2.92) than the controls (Mean = 4.10; S.D. = 1.50) (F (2, 61) = 7.54; critical F0.05(1, 61) = 6.30), but the migraineurs (Mean = 4.98; S.D. = 3.05) did not differ significantly from either of these groups. The differences between the means for the tension and migraine, tension and control, and between the migraine and control groups were required to exceed 2.14, 1.73 and 1.83 respectively, in order to reach the 0.05 level of significance as assessed by Scheffe's procedure. The critical F-value was given by 2 F0.05(2, 61) = 6.30.

The significant Group x Intensity interactions on linear and quadratic trend were explored by comparing pairs of groups within each trend component and controlling the Type I error rate by adjusting the significance level for the number of comparisons made (Kirk, 1968). Since it was intended to make all three comparisons between groups for each trend component, the Type I error rate was set at 0.017 (i.e., 0.05 / 3).

The a posteriori F-tests revealed significant differences between tension headache and control subjects on linear trend (F (1, 49) = 6.38; p = 0.015) and between the migraine and control subjects on quadratic trend (F (1, 47) = 10.86; p = 0.002). The difference between the migraine and tension headache subjects on quadratic trend fell just short of the 0.017 significance level (F (1, 26) = 6.22; p = 0.019). No other significant differences between
groups on linear or quadratic trend were noted. The results are illustrated in Figure 8-7 and the ANOVA summary tables are presented in Appendix K-4.

Inspection of Figure 8-7 indicates that the significant difference between the tension and control groups on linear trend, was due to the fact that the $P_1-N_1$ amplitude increased more rapidly with stimulus intensity for the tension headache subjects than for the controls. The significant difference between control and migraine subjects, on quadratic trend, appears to have been due to the fact that, between the two highest stimulus intensities, the $P_1-N_1$ amplitude for the control group continued to rise, whilst for the migraineurs, this amplitude tended to reduce slightly.

Analysis 4.

The tension, migraine and control groups were compared on the $N_j-P_2$ amplitude by the method described in Analysis 3. The ANOVA revealed a significant main effect for Group ($F(2, 61) = 3.17; p < 0.05$) but no significant Group x Intensity interaction on linear ($F(2, 61) = 0.77; p > 0.05$) or quadratic trend ($F(2, 61) = 2.36; p > 0.05$) (see Appendix K-5 for the ANOVA summary table). It was concluded that there were no significant differences between the tension headache, migraine or control groups on linear or quadratic trend for the $N_j-P_2$ amplitude. The results are illustrated in Figure 8-8.

The significant Group effect was explored by testing for differences between mean $N_j-P_2$ amplitudes with Scheffe's procedure. The tension headache subjects were observed to have significantly larger $N_j-P_2$ amplitudes ($Mean = 7.69; S.D. = 4.74$) than the controls ($Mean = 5.08; S.D. = 2.52$) ($F(2, 61) = 6.33$; critical $F$-value = 2 $F_{0.05}(2, 61) = 6.30$). The migraineurs ($Mean = 5.80; S.D. = 3.63$) did not differ significantly from either of the remaining groups. The differences between the means for the tension and migraine, tension and control, and between the migraine and control groups were required to exceed 3.21, 2.60 and 2.74 respectively, in order to reach the 0.05 level of significance as assessed by Scheffe's procedure.

Analysis 5.

Regardless of headache type, the headache and control subjects were compared on $N_j-P_2$ amplitude by means of the procedure outlined under Hypothesis 3. The 2 x 4 split plot design, with subjects matched in pairs for age and sex, was analysed by means of a two-way ANOVA with repeated measures on the Stimulus Intensity factor. The ANOVA revealed a significant main effect for Group ($F(1, 35) = 4.24; p < 0.05$), with $N_j-P_2$ amplitudes being
FIGURE 8-7

P₁-N₁ Mean Amplitudes for Tension, Migraine and Control Subjects.

- ■ Tension
- + Migraine
- Δ Control

Stimulus Intensity (mA)
FIGURE 8-8

\( N_1-P_2 \) Mean Amplitudes for Tension, Migraine and Control Subjects.

- ■ Tension
- † Migraine
- Δ Control
larger for headache (Mean = 6.65; S.D. = 4.00) than for control subjects (Mean = 5.08; S.D. = 2.52). There was no significant Group x Intensity interaction on linear (F (1, 35) = 2.54; p > 0.05) or quadratic trend (F (1, 35) = 2.47; p > 0.05) (see Appendix K-6 for the ANOVA summary table). The results are represented graphically in Figure 8-9.

Analysis 6.

In order to examine whether or not the behaviour of the $N_1-P_2$ amplitude, in response to increasing stimulus intensity, was affected by the presence or absence of headache (Status), this SSEP amplitude was compared across headache and headache-free conditions for the 16 headache subjects assessed during and between attacks. The analysis followed the procedures outlined for the analysis of Hypothesis 4. The ANOVA revealed no significant main effect for Status (F (1, 15) = 0.57; p > 0.05) nor any significant Status x Intensity interaction on linear (F (1, 15) = 1.46; p > 0.05) or quadratic trend (F (1, 15) = 0.48; p > 0.05) (see Appendix K-7 for the ANOVA summary table). It was concluded that there were no significant differences between the headache (Mean = 7.04; S.D. = 2.98) and headache-free (Mean = 7.63; S.D. = 4.34) conditions on $P_1-N_1$ amplitudes or on the rate at which these amplitudes increased with stimulus intensity (see Figure 8-10).

Analysis 7.

In order to investigate any relationship between the $N_1-P_2$ SSEP amplitude and pain sensitivity, headache and control subjects were divided into pain sensitive and pain insensitive groups, as described previously, and the $N_1-P_2$ amplitude was analysed separately for each of the headache and control groups, as outlined in the analysis of Hypothesis 5.

For the headache subjects (n = 23), the ANOVA revealed no significant main effect for Pain Sensitivity (F (1, 21) = 0.30; p > 0.05), there being no significant difference between pain sensitive (Mean = 7.17; S.D. = 3.72) and pain insensitive headache subjects (Mean = 6.17; S.D. = 5.10) on the size of $N_1-P_2$ amplitudes. The ANOVA also revealed no significant Pain Sensitivity x Intensity interaction on linear (F (1, 21) = 0.04; p > 0.05) or quadratic trend (F (1, 21) = 0.01; p > 0.05) (see Figure 8-11). The ANOVA summary table is presented in Appendix K-8.

For the control subjects (n = 28) the main effect for Pain Sensitivity fell just short of significance (F (1, 26) = 3.62; p = 0.068), there being a trend for pain sensitive subjects (Mean = 6.12; S.D. = 3.28) to have larger $N_1-P_2$ amplitudes than pain insensitive subjects (Mean =
FIGURE 8-9

N₁-P₂ Mean Amplitudes for Headache Sufferers and Control Subjects.

Stimulus Intensity (mA)

N₁-P₂ Amplitude (uV)

- Headache
- Control
FIGURE 8-10

N\textsubscript{1}-P\textsubscript{2} Mean Amplitudes for Headache Subjects Tested During and Between Attacks.

- □ With Headache
- △ Without Headache

Stimulus Intensity (mA)

N\textsubscript{1}-P\textsubscript{2} Amplitude (µV)
FIGURE 8-11

$N_1$-$P_2$ Mean Amplitudes for Pain Sensitive and Pain Insensitive Headache Subjects.

- ■ Pain Sensitive
- △ Pain Insensitive

Stimulus Intensity (mA) vs. $N_1$-$P_2$ Amplitude (μV)
4.23; S.D. = 1.86). The ANOVA revealed a significant Pain Sensitivity x Intensity interaction on linear trend ($F(1, 26) = 4.37; p < 0.05$), indicating that $N_1-P_2$ amplitudes increased more rapidly with stimulus intensity for pain sensitive than for pain insensitive control subjects (see Figure 8-12). There was no significant Pain Sensitivity x Intensity interaction on quadratic trend ($F(1, 26) = 0.17; p < 0.05$). The ANOVA summary table is presented in Appendix K-8.

8.5. DISCUSSION.

The present study was concerned with an investigation of the central theory of headache (Sicuteri, 1982; Sicuteri Anselmi and Del Bianco, 1978). The theory asserts that headache sufferers may be characterised by a deficiency in the central pain inhibitory system, disposing it to breakdown during periods of stress (Sicuteri, 1982), and that this contributes substantially to the onset of headache attacks. The proponents of this hypothesis have been rather vague about how this deficiency becomes manifest as a headache. On the one hand, it has been suggested that there exists a generalised failure of the endogenous analgesic system causing headache sufferers to be more sensitive to pain than headache-free persons in all regions of the body (Sicuteri, Anselmi and Del Bianco, 1978). This implies that some peripheral process such as local chemical action (Chapman et al., 1960), vasodilatation (Tunis and Wolff, 1953) or muscle contraction (Ostfeld et al., 1957) must interact with the disruption of central pain control mechanisms to account for the presence of pain in cranial regions. On the other hand, however, Sicuteri (1982) has suggested that the weakness in the pain inhibitory system is such that it affects only the head, neck and shoulders. Furthermore, it is not clear from the theory whether this weakness is manifest at all times or only on occasions when headache is present. Finally, there remains the question of whether any heightened pain sensitivity observed in headache sufferers can be shown to have a neurophysiological basis. The central theory, contends that the deficiency in the pain control system of headache sufferers is constituted by a depletion of pain inhibitory substances such as serotonin, endorphins and enkephalins in the CNS (Sicuteri, 1976, 1981, 1982; Sicuteri, Anselmi and Del Bianco, 1978). The study reported in this chapter was concerned with the clarification of these issues.

Headache subjects assessed between attacks were found to be more sensitive than control subjects to electrical pain in the finger, as represented by lowered pain threshold and tolerance levels. With respect to differences between these groups on their sensitivity to pain in the head, the results were less conclusive. Headache subjects demonstrated a lower tolerance for temporal ice pain than control subjects, but there was no difference between these groups on the pain threshold measure. These results suggest that, during periods of headache freedom, headache sufferers may be more sensitive to pain than headache-free persons, and
FIGURE 8-12

$N_1$-$P_2$ Mean Amplitudes for Pain Sensitive and Pain Insensitive Control Subjects.

- ■ Pain Sensitive
- △ Pain Insensitive

Stimulus Intensity (mA)
that this heightened sensitivity may be a generalised condition affecting other regions of the body besides the head.

Subjects reporting unilateral headaches were no more sensitive to ice pain in the temporal region, habitually affected by headache, than in the corresponding region contralateral to the site of headache. This finding suggests that when headache-free, headache sufferers may be no more pain sensitive over the site of headache, than in other regions of the head. However, Drummond (1987) did find scalp tenderness to be greater over the usual site of headaches than in other regions. In the present study only 13 subjects reported unilateral headaches affecting the temporal region, whereas, Drummond (1987) studied 65 subjects reporting unilateral headaches. The difference in sample sizes could account for the discrepant results.

As noted in Chapter 2, several studies have observed no significant differences in pain sensitivity between headache and control subjects (Feurstein et al., 1982; Gannon et al., 1981; Martin and Mathews, 1978). However, in these studies, subjects were not matched for age and sex, which are known to affect pain report in experimental studies (Woodrow et al., 1972). In the study reported by Martin and Mathews (1978), pain sensitivity was assessed whilst many of the headache subjects were experiencing headache. Any difference between groups may have been masked by the presence of pain concurrent with the experimental stimulus (Chen et al., 1985; Le Bars et al., 1983). In the present study, an effort was made to rectify these methodological shortcomings and this may have contributed to the observation of a heightened pain sensitivity amongst headache subjects.

Drummond (1987) assessed the rate of increase in pressure pain induced in the fingers of headache sufferers (tested in the headache-free interval) and control subjects, but observed no difference between groups. The headache subjects tested between attacks consisted of 68 migraineurs and 10 subjects reporting episodic tension headaches. This latter group of subjects was observed to report more rapid increases in pressure pain than either controls or migraineurs. Unfortunately, the frequency of headache within the headache groups was not specified. In the study reported in this chapter, all headache subjects reported their headaches as occurring at least once per week. It may be that the headache subjects included in the present study had more frequent headaches than the migraineurs studied by Drummond (1987), and that this might be responsible for the discrepant results with respect to finger pain. In future investigations it would be useful to classify subjects on the basis of headache frequency before testing for differences between groups on pain sensitivity.

When the sample of headache sufferers assessed in the present study was divided into tension and migraine types no significant difference between groups on sensitivity to finger
pain (assessed between headache attacks) was observed, although both groups were found to be significantly more pain sensitive than the controls on all measures except temporal ice pain threshold. These observations suggest that the difference in finger pain sensitivity between Drummond's (1987) migraineurs and episodic tension headache subjects, may have been due to factors other than a difference in the number of migrainous features characterising these two groups.

In the present study no significant differences on finger pain sensitivity were observed for the 16 headache subjects tested during and between headache attacks. These results do not support the hypothesis that headache is accompanied by a generalised increase in pain sensitivity (Sicuteri, 1982). Indeed, it has been reported that headache sufferers are more sensitive to finger pain in the headache-free interval than during attacks (Drummond, 1987). This latter finding may be explicable in terms of pain in one part of the body attenuating the experience of pain in other regions (e.g., Chen et al., 1985; Le Bars et al., 1983).

The results obtained on the pain measures employed in the present study are consistent with the hypothesis that there exists, amongst headache sufferers, a generalised weakness in the endogenous pain control system, affecting various regions of the body (Sicuteri, Anselmi and Del Bianco, 1978), which is not specific to the occurrence of headache. The findings are consistent with the observation that headache sufferers, assessed in the headache-free have lower CSF levels of the pain inhibitory substance beta-endorphin than controls (e.g., Genazzani et al., 1984; Nappi, Facchinetti, Martignoni, Petraglia, Bono et al., 1985; Nappi, Facchinetti, Martignoni, Petraglia, Manzoni et al., 1985). However, since results less supportive of this hypothesis were reported by Drummond (1987), with respect to finger pressure pain, more research is required before the central theory can be regarded as having substantial empirical support. It would be important to attempt to identify groups of headache sufferers for whom heightened pain sensitivity may play a role in the pathophysiology of their attacks. The results of the present study suggest that headache sufferers, reporting at least one headache per week, may fall into this category irrespective of whether or not they report migrainous features in association with their headaches. The results reported in this chapter also suggest that if there is a deficiency in the pain inhibitory system of headache sufferers, this deficiency is not specific to the shoulders and above, as hypothesised by Sicuteri (1982), and that any such deficiency is likely to be a constant feature of the pain control system rather than a phenomenon which occurs only in association with headache episodes. However, it is possible that some additional disruption of pain regulatory mechanisms does occur during headache attacks but that, in the present study, this phenomenon was masked by subjects' concurrent experience of headache. For example, CSF enkephalin levels have been found to be lower during than between episodes of migraine headache (Anselmi et al., 1980).
Alternative interpretations of the results obtained on the pain measures, in terms of psychological differences between headache and control subjects, cannot be ruled out. Attitudes towards experimental pain have been found to exert a profound influence upon pain measures (e.g., Davison and Vallis, 1969; Spanos, et al., 1981; Vallis and Bucher, 1986; see Appendix A). Given their history of having been inconvenienced or distressed by pain repeatedly, the headache subjects may have appraised the experimental pain stimulus as more threatening than their headache-free counterparts. This fear of pain may have depressed headache subjects' pain threshold and tolerance levels (Letham, et al., 1983; Slade, et al., 1983). Future studies could address this hypothesis by assessing headache and control subjects' cognitive and affective responses during experimental pain induction procedures. Such a methodology has been described by Spanos and his colleagues (Spanos, Radtke-Bodorik, Ferguson and Jones, 1979; Spanos et al., 1981).

Sicuteri and his co-workers (Sicuteri, 1976, 1982; Sicuteri, Anselmi and Del Bianco, 1978) contend that headache sufferers are more pain sensitive than headache-free individuals, and that this heightened sensitivity has its origin in depleted levels of pain inhibitory substances in the CNS. The finding that plasma levels of serotonin are lower during than between migraine attacks (e.g., Anthony et al., 1967; Curran et al., 1965), and lower in persons with daily tension headache than in control subjects (e.g., Anthony and Lance, 1985; Rolf et al., 1981) is consistent with this hypothesis. However, it is difficult to generalise from differences in plasma levels of serotonin to differences in brain levels of this amine, particularly since serotonin is not stored in large quantities in brain structures (Fozard, 1982).

In the present study it was decided to investigate the hypothesised heightened sensitivity of the headache sufferer's nervous system to sensory input (Sicuteri, 1976, 1982; Sicuteri, Anselmi and Del Bianco, 1978), by studying the behaviour of SSEP amplitudes over increasing stimulus intensities. This line of inquiry was considered important from the standpoint of evaluating the assumption that differences in neurophysiological functioning underlie headache sufferer's heightened sensitivity to pain (Sicuteri, 1982; Sicuteri, Anselmi and Del Bianco, 1978).

The literature on augmenting-reducing in AEP amplitudes suggests that marked increases in these amplitudes, in response to increasing stimulus intensities, between 60 and 300 msec after stimulation, may be indicative of a failure of sensory modulation giving rise to a heightened sensitivity to pain (e.g., Buchsbaum et al., 1977, 1981b; Harkins and Chapman, 1978; von Knorring, 1978; von Knorring et al., 1974). Furthermore, augmenting in AEP amplitudes has been found to be associated with low levels of endorphins and low levels of the serotonin metabolite 5-hydroxyindoleacetic acid (5-HIAA) in the CSF (von Knorring and Perris, 1981) and with the administration of substances known to deplete CNS levels of
serotonin and endogenous opioids (e.g., Buchsbaum et al., 1981b; Davis et al., 1978; von Knorring and Johansson, 1980). Unfortunately, this body of research has not been successful in identifying those particular AEP amplitudes related to pain mechanisms. This may be explicable in terms of the methodological inconsistencies reviewed earlier in this chapter. Nevertheless, Connolly et al (1982) studied the VEP recorded from vertex in migraine and control subjects and observed an enlarged $P_1-N_1$ amplitude for the patient group. The present study sought to extend this finding to somatosensory stimulation and to bring any enlargement of the $P_1-N_1$ amplitude for headache sufferers into line with the literature on augmenting-reducing, by investigating whether or not this amplitude increased more rapidly with stimulus intensity for headache than for control subjects. Finally, the study was concerned with exploring the question of whether or not any abnormality observed in the $P_1-N_1$ amplitude, amongst headache sufferers, could be related to measures of pain sensitivity. This last issue was considered important from the viewpoint of articulating any differences between groups on SSEP amplitudes to the central theory of headache, cast in terms of a heightened sensitivity to pain.

As hypothesised, the $P_1-N_1$ SSEP amplitude, recorded from vertex, was found to be larger and to increase more rapidly with stimulus intensity for headache subjects than for controls. The $N_1-P_2$ amplitude was also greater for headache than control subjects, however, for this component there was no difference between groups on the rate at which the amplitude increased with stimulus intensity.

In order to determine whether or not these differences in SSEP amplitudes could be related to pain mechanisms, each of the headache and control subject samples were divided into pain sensitive and pain insensitive groups. On the $P_1-N_1$ component, no differences between pain sensitive and pain insensitive groups was observed for either the headache or control subjects on mean amplitude or on the rate at which this amplitude increased with stimulus intensity. Among control subjects the $N_1-P_2$ amplitude was observed to increase more rapidly with stimulus intensity for pain sensitive than for pain insensitive subjects. There was also a trend for the mean $N_1-P_2$ amplitude to be larger for the pain sensitive subjects. However, these observations were not replicated with the headache subjects, for whom no significant differences between pain sensitive and pain insensitive groups was observed on the $N_1-P_2$ amplitude.

Thus, although the $P_1-N_1$ SSEP amplitude increased more rapidly with stimulus intensity for headache sufferers and although both this component and the $N_1-P_2$ amplitude were larger for headache sufferers than for controls, these aspects of the SSEP were not found to be related consistently to the pain sensitivity measures which also distinguished the headache and control groups. It would appear that, although the nervous systems of headache
sufferers may be more responsive to sensory input, as suggested by the presence of enlarged SSEP component amplitudes, this heightened reactivity may not underlie the heightened pain sensitivity also observed for these subjects. This suggests that the differences observed on the SSEP and pain measures may represent separate processes, each of which could be involved in the pathophysiology of headache.

Amongst depressed psychiatric patients, augmenting in the VEP has been associated with reports of greater frequencies of life events in the year prior to admission, increased ratings of distress in the face of those life events, and with ratings of the degree of difficulty encountered in adapting to such events (von Knorring, Jacobsson, Perris and Perris, 1980). These researchers defined the VEP amplitude as the largest peak to peak difference occurring over the first 280 msec following stimulus onset. The findings raise the possibility that the augmenting response in the SSEP $P_1-N_1$, amplitude observed for headache subjects in the present study, may represent a tendency for these persons to experience the exigencies of everyday life as stressful and to be, therefore, more likely to experience headache in the face of such stress. Indeed, in the last chapter, greater frequencies of stressful events were shown to be associated significantly with more frequent headache attacks. The $P_1-N_1$ amplitude may represent some process which buffers the relationship between stressful events and headache, in a manner similar to that demonstrated for self-control-efficacy in the last chapter. The exploration of such an hypothesis might cast some light on the nature of the relationship between SSEP amplitudes and headache.

The absence of relationships between SSEP amplitudes and pain sensitivity in the present study is difficult to explain. Studies which do report significant relationships between these variables have typically employed the amplitude / intensity slope as the measure of augmenting-reducing and have found this to be correlated with pain sensitivity (e.g., Buchsbaum and Davis, 1979; Buchsbaum et al., 1977; Lavine et al., 1976). The calculation of amplitude / intensity slopes was avoided in the present study because, for many individuals, the regression equation accounts for so little of the variance in the relationship between amplitude and intensity that the slope cannot be regarded as an adequate index of this relationship (Connolly and Gruzelier, 1982; Iacono et al., 1982). However, it seems unlikely that this difference in approach to the measurement of augmenting-reducing could account for the absence of significant relationships in the present study. Indeed, it is argued that the methodology employed herein is more likely to be sensitive to such relationships than the practice of calculating amplitude / intensity slopes.

As noted earlier in this chapter, the literature reporting relationships between AEP components and pain mechanisms is difficult to interpret because of the plethora of recording methodologies and peak identification methods employed. Until the approach to AEP
recording becomes more uniform it will remain difficult to account for the occurrence of unexpected results, as observed in the present study. More research into the effects of different AEP recording methods on the relationship between amplitude and pain sensitivity are required.

Connolly et al (1982) observed a greater $P_1-N_1$ mean amplitude for migraineurs than for control subjects on the VEP whilst no difference between these groups was observed in the present study. This discrepancy may be explicable in terms of the different stimulus modalities employed. That is, the results obtained by Connolly et al (1982) could represent a heightened sensitivity specific to photic stimuli in migraineurs. Many migraine sufferers are known to be sensitive to light during headache attacks (Dalessio, 1980; Lance, 1982), and Connolly et al (1982) may have been examining a process related to this phenomenon. Furthermore, it has been suggested that augmenting-reducing in the AEP may be dependent upon the modality of stimulation (Kaskey et al., 1980; Raine et al., 1981). With respect to the present findings, the above considerations may account for the absence of enlarged SSEP amplitudes for the migraine subjects.

To the author's knowledge, all studies conducted on the AEP in headache have been confined to the response of migraineurs to photic stimulation. Given that the present study uncovered significant differences between tension headache and control subjects on both the $P_1-N_1$ and $N_1-P_2$ SSEP amplitudes, it is clear that future research needs to be concerned with the spectrum of headache from tension to migraine and ought not be confined to migraineurs. In the present study, the $P_1-N_1$ amplitude was observed to be larger for tension headache subjects than for controls, whilst the migraine subjects did not differ significantly from the controls on this component. The $P_1-N_1$ amplitude also increased more rapidly with stimulus intensity for the tension headache than for the control subjects, there being no significant difference between migraineurs and controls on this aspect. Finally, the $N_1-P_2$ amplitude was found to be larger for the tension headache subjects than for the controls whilst the migraineurs did not differ significantly from the controls on this component. Thus, a heightened responsivity of the CNS to somatosensory input may be more readily apparent in tension headache sufferers than in migraineurs.

For headache subjects assessed during and between headaches, no significant differences between conditions were evident on the mean $P_1-N_1$, mean $N_1-P_2$ or on the behaviour of these amplitudes over the range of stimulus intensities. Thus, it would appear that the larger $P_1-N_1$ and $N_1-P_2$ amplitudes, and the strong augmenting response on the $P_1-N_1$ component observed for headache subjects, are consistent features of these persons, and are not accentuated during periods of headache. This suggests that the reactivity of the nervous system of headache sufferers to somatosensory input may not be altered during headache.
Reductions in CSF levels of pain inhibitory substances have been reported to occur during attacks of migraine headache (Anselmi et al., 1980) and such processes have been thought to occur centrally (Sicuteri, 1982). Accordingly, the absence of significant differences in SSEP amplitudes between headache and headache-free intervals was surprising. However, the situation is complicated by the fact that tonic pain, induced experimentally, has been found to depress the size of the \( P_1 \) and \( N_1 \) amplitudes to an extent similar to that observed for morphine (Chen et al., 1985). Thus, it is possible that during headache, two opposing forces operated on SSEP amplitudes, namely, an increased sensitivity attributable to the biochemical changes considered to occur by Sicuteri (1982) and an inhibitory effect of headache pain itself. The latter process may have masked the effect of the former on SSEP amplitudes.

The results of the present study suggest that headache sufferers may be more pain sensitive than headache-free persons and that this increased sensitivity may not be specific to the head. These observations are consistent with the central theory of headache as proposed by Sicuteri, Anselmi and Del Bianco (1978). The enlarged \( P_1-N_1 \) and \( N_1-P_2 \) components, and the stronger augmenting response on the \( P_1-N_1 \) amplitude, observed for headache subjects relative to controls, are also supportive of the theory that some deficiency in the capacity to modulate somatosensory input may characterise headache sufferers. However, the absence of significant relationships between SSEP parameters and pain sensitivity makes it difficult to claim that these SSEP abnormalities are indicative of deficiencies in pain processing. Nevertheless, the results for both the pain measures and the SSEP do suggest that headache sufferers may be more responsive to somatosensory input than headache-free persons. This heightened responsivity could contribute to the occurrence of headache attacks in the manner outlined by the central theory. That is, headache sufferers may possess weakened sensory modulation systems which are easily disrupted by stress and which may contribute to the onset and / or severity of headache attacks under these conditions (Sicuteri, 1982).

Bandura et al (1987) observed that the extent to which cold pressor pain tolerance times could be reduced following the administration of naloxone (an opiate antagonist) was associated significantly with the level of self-efficacy to reduce pain following training in cognitive pain management strategies. These results suggest that efficacy expectations may be related to central pain regulatory mechanisms.

In a second study (Bandura, Coiffi, Taylor and Brouillard, 1988), subjects' perceived mathematical self-efficacy was manipulated by assigning them to a condition where they could control the rate at which they had to solve mental arithmetic problems (high self-efficacy condition) or to a condition where the same tasks were presented at a rapid rate exceeding their cognitive capacity (low self-efficacy condition). Cold pressor pain tolerance was assessed before the mental arithmetic tasks. Ratings of mathematical self-efficacy made before and after
completion of the arithmetic problems confirmed that the manipulations altered self-efficacy in the expected directions. Following the arithmetic tasks, subjects in each condition were injected with saline or the opiate antagonist naloxone and then re-tested for pain tolerance on the cold pressor test. No significant difference between the saline and naloxone administrations was observed on percentage change in pain tolerance for the high self-efficacy (low-stress) subjects, whereas for those in the low self-efficacy (high-stress) condition the percentage increase in pain tolerance was significantly greater for subjects receiving saline than for those receiving naloxone.

Bandura et al (1988) concluded that a lowering of self-efficacy is associated with opioid activation, this being blocked for the subjects receiving naloxone. However, since ratings of stress, time pressure and perceived impairment of task performance were observed to be greater in the low self-efficacy than in the high self-efficacy group, it is equally plausible that the increased opioid activation was attributable to stress (e.g., Akil et al., 1976).

Bandura et al (1988) also observed that, for subjects in the high self-efficacy condition, those receiving saline demonstrated a significant positive correlation between increases in mathematical self-efficacy and increases in pain tolerance. No significant correlation between the variables was observed for subjects receiving naloxone. For subjects in the low self-efficacy condition, receiving saline, reductions in mathematical self-efficacy were associated significantly with increases in pain tolerance whilst, for those receiving naloxone, reductions in self-efficacy were associated significantly with reductions in pain tolerance.

The results reported by Bandura et al (1988) suggest that exposing subjects to high levels of cognitive stress (low mathematical self-efficacy condition) may produce opioid activation (as suggested by the reduction in pain tolerance for the subjects receiving naloxone), whilst for those exposed to lower levels of stress (high self-efficacy condition), no opioid activation may occur (as suggested by the absence of any differential effect of saline and naloxone on pain tolerance). When subjects are not exposed to high levels of cognitive stress (high self-efficacy condition) increases in self-efficacy may be related to increases in pain tolerance, provided that the endogenous pain control system is unimpaired (no such correlation was observed when subjects were given naloxone). For subjects exposed to high levels of cognitive stress (low self-efficacy condition), reductions in self-efficacy may be associated with increases in pain tolerance when they receive saline, but when the opioid system is impaired by naloxone, reductions in self-efficacy may be associated with reductions in pain tolerance.

The results obtained by Bandura et al (1988) have implications for an understanding of the relationship between stress and headache. Headache sufferers have been postulated to have
impaired endogenous opioid systems (e.g., Genazzani et al., 1984; Sicuteri, 1982). The results of the present study suggest that headache sufferers may be more pain sensitive and their nervous systems more reactive to somatosensory input than headache-free persons. Furthermore, the results presented in Chapter 7 suggested that self-control-efficacy may moderate the strength of the relationship between stressful events and headache frequency: lower levels of self-control-efficacy being associated with a stronger relationship between the frequency of stressful events and the frequency of headache. If the endogenous opioid system is impaired in headache sufferers, the findings obtained by Bandura et al (1988) suggest that stressful events and lower levels of self-efficacy could lead to a heightening of pain sensitivity and an increased probability of headache occurring, this process being mediated by the endogenous opioid system. Thus, the moderating effect of self-control-efficacy on the relationship between the frequency of headache and the frequency of stressful events, reported in Chapter 7, could have been mediated by endogenous opioid functions. This hypothesis could be explored by replicating the study conducted by Bandura et al (1988) on headache sufferers and headache-free subjects. Headache subjects would be expected to show lower levels of opioid activation under cognitive stress and to demonstrate greater reductions in pain tolerance following such stress than controls. Furthermore, reductions in self-efficacy, occurring under stress, would be expected to be associated with reductions in pain tolerance for headache subjects (due to a lack of opioid activation under stress) but with increases in pain tolerance for control subjects.

Additional studies could investigate the hypothesis that cognitive processes, such as self-control-efficacy, may be related to headache through the mechanism of sensory modulation, by examining interactions of these variables with measures of pain sensitivity or AEP amplitude in the prediction of headache severity. Such lines of inquiry may contribute more to an understanding of the mechanisms through which psychological variables contribute to headache than has been gained, heretofore, from a focus upon relationships between these variables and the processes of muscle contraction and vasodilatation.

8.6. SUMMARY.

In Study III an investigation of the central theory of headache was conducted by comparing headache sufferers and control subjects on pain sensitivity and SSEP amplitudes, occurring between 60 and 300 msec after electrical stimulation of the index finger.

Headache sufferers were found to have lower pain threshold and tolerance levels for electrical stimulation of the index finger than headache-free control subjects. Headache subjects were also observed to have lower pain tolerance levels when ice was applied to the
temporal region, but no significant difference between groups was observed on temporal ice pain threshold. When headache subjects were assessed on pain threshold and tolerance for electrical finger pain, during and between headache attacks, no significant difference between conditions was observed. When temporal ice pain threshold and tolerance levels were assessed in a group of headache sufferers reporting headaches affecting only one side of the head, including the temporal region, no significant differences between the habitually affected side and the unaffected side were observed.

It was concluded that headache sufferers may be more sensitive to pain than headache-free persons; that this increased sensitivity may be a consistent feature of headache sufferers, unaffected by the presence or absence of headache; and that the heightened pain sensitivity of headache subjects may not be specific to the head or the region affected habitually by headache.

The question of whether there exists any neurophysiological basis to the increased pain sensitivity of headache subjects was addressed by assessing the behaviour of the $P_{1-N_1}$ and $N_1-P_2$ components of the SSEP over increasing levels of stimulus intensity. The $P_{1-N_1}$ amplitude of the SSEP was found to be greater and to increase more rapidly with stimulus intensity for headache sufferers than for control subjects. The $N_1-P_2$ amplitude was also found to be larger for headache than for control subjects, but no difference between groups on the rate at which this amplitude increased with stimulus intensity was observed. When the $P_{1-N_1}$ and $N_1-P_2$ amplitudes were assessed in headache subjects during and between attacks, no significant differences between conditions were observed. No significant differences between pain sensitive and pain insensitive subjects were found on $P_{1-N_1}$ or $N_1-P_2$ amplitudes for headache subjects. For control subjects, the $N_1-P_2$ amplitude increased more rapidly with stimulus intensity for pain sensitive than for pain insensitive subjects, but no other significant differences were observed.

It was concluded that the nervous systems of headache sufferers may be more responsive to sensory input than those of headache-free control subjects. Some deficiency in the mechanism of sensory modulation may represent one avenue through which cognitive processes contribute to headache.
CHAPTER 9

CONCLUSIONS AND DIRECTIONS FOR FURTHER RESEARCH.

The studies reported in this thesis aimed to identify the cognitive appraisal processes related to recurrent headache and to develop and examine a conceptual model detailing the inter-relationships between appraisal processes, stressful events, coping and headache. Furthermore, the thesis was concerned with evaluating the prospects for articulating cognitive processes to headache through the mechanism of sensory modulation as outlined in the gate control theory of pain (Melzack and Wall, 1965; 1982; see Appendix A) and in the central theory of headache (Sicuteri, 1982; Sicuteri, Anselmi and Del Bianco, 1978). In an endeavour to achieve these aims, three empirical studies were conducted. The conclusions following from each of these studies are detailed in the following sections.


In Study I (see Chapter 6) an attempt was made to identify the cognitive appraisal processes involved in headache by means of a comparative treatment study. The relationship between stressful events and headache was conceptualised in terms of the rational-emotive theory of emotional distress (Ellis, 1962, 1977a, b). According to this theory human beings experience intense aversive emotions, in response to stressful events, to the extent that their belief systems are irrational. Thus, the distinction between rational and irrational beliefs was a crucial theoretical issue which had to be addressed before applying the theory to an analysis of the relationship between stress and headache.

It was argued that an irrational belief is one which demands that particular outcomes follow from one's actions or that reality be altered. However, it was noted that the rational desire for reality to be different and the irrational demand that it should or must be different (Ellis, 1962, 1977a, b) are easily confused when this dimension is assessed by questionnaire. In order to overcome the problem of distinguishing between desires and demands it was decided to focus upon rationality, defined by Ellis (1962) as an acceptance of oneself, others and the environment. Irrationality was regarded as any departure from this position. The Rationality Scale was developed along these lines and was administered to headache sufferers before and after rational-emotive therapy or progressive relaxation training.
The results did not support the rational-emotive model of the relationship between stress and headache. Reductions in headache frequency, intensity, duration and medication intake were not reliably greater for the rational-emotive than for the relaxation treatment at the post-treatment or 12-week follow-up assessments. Furthermore, when post-treatment Rationality Scale scores were correlated with each post-treatment headache outcome measure by means of partial correlation analyses (controlling for the pre-treatment values of each pair of variables), no significant partial correlation coefficients were observed for either treatment. Thus, it was concluded that the significant reductions in headache frequency observed for the relaxation treatment, and the significant reductions in headache frequency and intensity observed for the rational-emotive treatment, were not mediated by increases in rationality, as assessed by the Rationality Scale.

It was suggested that self-efficacy expectations (Bandura, 1977, 1982, 1986) may have been strengthened in each treatment by providing subjects with a rationale for understanding the origin of their headaches, as well as with strategies by which they might master their disorder. It was suggested further that the development and examination of a model, linking self-efficacy to headache, may cast more light on the mediational processes involved in the relationship between stress and headache than has been gained through a focus upon rationality.


In the theoretical model proposed in the introduction to Study II (see Chapter 7), it was postulated that self-efficacy, regarding control over behavioural, cognitive and affective responses (self-control-efficacy) moderates the strength of the relationship between stressful events and headache onset. It was postulated further that self-control-efficacy would be related to headache through the mechanisms of appraisal, coping and emotional upset. It was proposed that self-control-efficacy determines the level of affective regulation in the face of stressful events and that, this coping strategy, reduces the strength of the emotional response to stressful events, thereby mitigating against headache onset. Furthermore, it was proposed that a high level of self-control-efficacy would lead individuals to appraise stressful events as amenable to change; to engage in direct coping to alter the stressor; reduce the intensity of the emotional response; and reduce the probability of headache occurring in the face of stressful events. Finally, a low level of self-control-efficacy was expected to be associated with a tendency to appraise stressful events as not amenable to change which would, in turn, be associated with avoidance coping, an increased emotional response to the stressor and a greater probability of headache occurring.
The frequency of stressful events was observed to account for a significant proportion of the variance in diary recordings of headache frequency. Furthermore, and as expected, the strength of this relationship was found to be moderated by the level of self-control-efficacy such that, the positive correlation between stressful events and headache was greater for subjects low in self-control-efficacy than for those high in self-control-efficacy.

Stressful events occurring within headache episodes were found to be associated with increases in headache intensity following those events. When subjects' continuous records were divided into headache and headache-free cycles (a headache cycle being defined as a headache-free day followed by a headache day; a headache-free cycle as two consecutive headache-free days), stressful events were found to precede headache onset more often than periods of headache freedom. These results suggest that stressful events may be causally related both to headache onset and to increments in headache intensity.

Consistent with the proposed theoretical model, appraisals of stressful events as amenable to change and reliance upon affective regulation and direct coping efforts were more evident in periods leading up to headache freedom (headache-free cycles) than in periods preceding headache (headache cycles). Furthermore, when stressful events occurred during headache attacks, increments in headache intensity following those events were associated with higher levels of avoidance coping and with lower levels of direct coping. However, contrary to the model, events were rated as more important in headache-free cycles and levels of avoidance coping and emotional upset were observed to be greater in headache-free than in headache cycles.

Since scores on all event coping scales were greater in the headache-free than in the headache cycles, it was suggested that it may be the number of coping strategies employed in response to a stressful event, rather than the nature of those strategies, that is the important determinant of whether or not a headache will develop. It was suggested further that when stressful events occur and are not recognised as important or upsetting (as indicated by the lower scores on the Event Mattered and Upset Rating Scales), such conditions may be conducive to headache onset. The results suggest that the events related most strongly to the occurrence of headache may be those which the headache sufferer perceives as having little relevance to his or her well-being.

Contrary to those aspects of the theoretical model linking self-control-efficacy to headache through the processes of appraisal and coping, self-control-efficacy was not observed to be correlated significantly with the extent to which events were appraised as amenable to change, the level of importance attached to those events or with the levels of affective regulation or direct coping. Consistent with the model a significant negative correlation
between self-control-efficacy and avoidance coping was noted. However, the finding that lower, rather than higher, levels of avoidance coping were associated with headache onset, suggests that this type of coping does not mediate the buffering effect of self-control-efficacy on the relationship between stressful events and headache.

Higher levels of self-control-efficacy were associated with lower levels of emotional upset in the face of stressful events. Once again, however, the fact that upset ratings were found to be greater in headache-free than in headache cycles, suggests that the level of emotional upset is unlikely to represent a mechanism by which self-control-efficacy exerts its buffering effect. Therefore, it appears that the buffering effect of self-control-efficacy was not mediated by the appraisal, coping or emotional responses under investigation.

The theoretical model linking stressful events to headache through self-control-efficacy, appraisal, coping and emotional response variables was partially supported by the data obtained. Stressful events were observed to contribute to the onset and intensity of headache attacks and the strength of the relationship between these events and headache frequency was moderated by self-control-efficacy. Furthermore, appraising events as amenable to change and engaging in efforts to cope with those events were found to mitigate against headache onset.

The main areas in which the model was not supported concern the linkages postulated to mediate the buffering effect of self-control-efficacy; the absence of any differential effect of coping on headache; and the presence of a heightened aversive emotional response and higher event-mattered ratings in periods leading to headache freedom (headache-free cycles) than in periods preceding headache onset (headache cycles). The model proposed may need to be modified so as to take account of the suggestion that headache sufferers may be most vulnerable to headache onset when they do not recognise the personal significance of stressful events. This interpretation of the data is consistent with the view that sensitising headache sufferers to the events, thoughts and feelings which precede headache attacks, may be an important ingredient in psychological treatment programmes (Bakal et al., 1981).

Although the theoretical model proposed in Chapter 7 was recognised as far from complete, it was considered that a more pressing concern than the specification of linkages between self-control-efficacy and headache, or the clarification of the relationship between emotional distress and headache, was the need to explore the prospects for linking cognitive constructs, such as self-control-efficacy, to the pathophysiological mechanisms that might be involved in headache attacks.

In view of the finding suggesting that self-efficacy to tolerate pain may be related to endogenous pain inhibitory processes (Bandura et al., 1987), and bearing in mind the lack of success that psychophysicists have had in relating psychological processes to the peripheral
mechanisms implicated in the pathophysiology of headache (see Chapter 2), it was decided to conduct an evaluation of the central theory of headache, with a view to facilitating the development of theoretical models linking cognitive constructs, such as self-control-efficacy, to the mechanisms of sensory modulation thought to be involved in the pathophysiology of headache (Sicuteri, 1982; Sicuteri, Anselmi and Del Bianco, 1978). This inquiry was undertaken in Study III.


In Study III (see Chapter 9) the central theory of headache (Sicuteri, 1982; Sicuteri, Anselmi and Del Bianco, 1978) was examined by studying differences between headache and headache-free control subjects on pain sensitivity and SSEP amplitudes. According to the central theory, headache is at least in part, the result of a depletion of pain inhibitory substances in the CNS.

It has been suggested that the pain control systems of headache sufferers are fragile (Sicuteri, 1982); that this is evident as a heightened sensitivity to pain (Sicuteri, Anselmi and Del Bianco, 1978); and that this system is prone to break-down during periods of stress (Sicuteri, 1982). Thus, an investigation of pain sensitivity in headache and control subjects was carried out. It was decided to include a study of SSEP amplitudes in order to determine whether or not there exists a neurophysiological basis for any heightened pain sensitivity observed for headache sufferers. The existence of such a neurophysiological mechanism is implied by the postulation of a depletion of pain inhibitory substances in the CNS as contributing substantially to headache (Sicuteri, 1982).

Headache sufferers assessed between attacks were observed to have lower pain threshold and tolerance levels for electrical finger pain than controls. When ice blocks were applied to the temple (temporal ice pain test), headache subjects demonstrated lower pain tolerance levels, but there was no significant difference between groups on pain threshold. It was concluded that, during periods of headache freedom, headache sufferers may be more pain sensitive than headache-free persons and that this heightened sensitivity may be a generalised condition which is not specific to the head.

Subjects reporting unilateral headaches affecting the temporal region were no more pain sensitive over the site of headache than on the contralateral side when tested between headache attacks. However, this result cannot be regarded as conclusive given that, amongst a larger sample of subjects, pain sensitivity has been reported to be higher in regions of the scalp affected habitually by headache than in unaffected regions (Drummond, 1987).
No differences in sensitivity to electrical finger pain were observed for headache subjects tested during and between headache attacks. This finding is inconsistent with the hypothesis that headache attacks are accompanied by a generalised increase in pain sensitivity (Sicuteri, 1982).

An investigation into the extent of any involvement of neurophysiological mechanisms in the heightened pain sensitivity of headache sufferers was carried out by a study of SSEP amplitudes. A review of the literature on augmenting-reducing in AEP amplitudes, occurring between 60 and 300 msec after stimulation, indicated that larger amplitudes and greater rates of increase in the size of the amplitudes, with increasing stimulus intensity, may be associated with both a heightened sensitivity to pain (e.g., Buchsbaum et al., 1977, 1981b; von Knorring, 1978), and with pharmacologically induced depletions of pain inhibitory substances (e.g., Davis et al., 1978; von Knorring and Johansson, 1980). Thus, it was hypothesised that SSEP amplitudes would be larger and would increase more rapidly with stimulus intensity for headache than for control subjects.

Since Connolly et al. (1982) observed the P1-N1 amplitude of the VEP recorded from vertex to be larger for migraine than for control subjects, it was hypothesised that differences between groups on the SSEP would be most evident on this component. It was also anticipated that SSEP amplitudes would be related to measures of pain sensitivity.

As hypothesised, the P1-N1 amplitude was observed to be larger, and to increase with stimulus intensity at a greater rate for the headache subjects than for the controls. The N1-P2 mean amplitude was larger for headache subjects, but there was no difference between groups on the rate at which this component increased with stimulus intensity. When subjects within each group were divided into pain sensitive and pain insensitive groups, on the basis of their scores on the pain tests, no consistent relationships between pain sensitivity and SSEP amplitudes were observed. It was concluded that, although the nervous systems of headache sufferers may be more responsive to sensory input, this increased reactivity may not account for their heightened pain sensitivity.

The P1-N1 and N1-P2 SSEP amplitudes and their relationships with stimulus intensity were found to be unaffected by the presence or absence of headache. It was concluded that the reactivity of the nervous systems of headache sufferers to somatosensory input may not be affected by the presence of headache.

The results obtained on the pain measures and SSEP amplitudes were interpreted as being consistent with the theory that some deficiency in the capacity to modulate sensory input may be an important factor in the pathophysiology of headache. It was suggested that such a
deficiency could represent one mechanism by which stress may contribute to headache. Specifically, it was conjectured that the buffering effect of self-control-efficacy, observed in Chapter 7, might exert its influence on the stress-headache relationship by acting upon the mechanism of sensory modulation.

9.4. General Conclusions.

The results of the empirical work reported in this thesis suggest the following general conclusions:

1. The effects of rational-emotive therapy or progressive relaxation training on recurrent headache do not appear to be mediated by increases in rationality (defined as the tendency to accept oneself, others and the environment, and assessed by the Rationality Scale). In addition, rational-emotive therapy may be no more effective than relaxation training in the treatment of headache. These findings suggest that rationality may not play a significant role in the relationship between stressful events and headache activity.

2. Frequent stressful events may be causally related to headache onset and to increases in headache intensity during attacks. The strength of the relationship between stressful events and headache frequency appears to be buffered by the level of self-efficacy regarding control over behavioural, cognitive and affective responses (self-control-efficacy). Appraisals of stressful events as amenable to change and the process of engaging in efforts to cope with those events, may mitigate against subsequent headache onset. However, any linkages between self-efficacy, appraisal, coping, emotional response and headache remain unclear, as does the issue of the type of coping most likely to be related to headache onset.

3. Avoidance coping, in response to stressful events occurring during headache attacks, may be associated with higher levels of headache intensity following the event, whilst direct coping may be associated with lower post-event headache intensity ratings. However, it is not clear whether these coping strategies could be causes, consequences or simply correlates of post-event fluctuations in headache intensity. Stressful events that are not recognised as significant for one's well-being or that are not associated with a strong aversive emotional response may be most likely to be followed by a headache.
4. Headache sufferers reporting at least one headache per week may be more sensitive to experimental pain than headache-free control subjects. This heightened sensitivity does not appear to be influenced by the presence or absence of headache and is not specific to the head. The results reported in Chapter 8 support the contention that there may exist a generalised weakness of the pain control system in headache sufferers (Sicuteri, Anselmi and Del Bianco, 1978), but do not support the hypothesis that such a deficiency is specific to the head (Sicuteri, 1982). The larger $P_1-N_1$ and $N_1-P_2$ SSEP amplitudes observed for headache subjects and the fact that the $N_1-P_1$ amplitude increased more rapidly with increasing stimulus intensity for the headache subjects, suggests that a neurophysiological basis may exist for a deficiency in sensory modulation in headache sufferers. The SSEP amplitudes under investigation were unaffected by the presence or absence of headache. Although it appears unlikely that the SSEP abnormalities observed in headache sufferers underlie their heightened pain sensitivity, the SSEP results indicate, nevertheless, that headache sufferers may be lacking in their capacity to modulate sensory input. This could represent a mechanism by which psychological processes contribute to headache attacks.

The results of the studies reported in this thesis suggest that cognitive appraisal processes may make a significant contribution to the severity of headache and, thus, strengthen the rationale for the application of cognitive therapy to this problem. In particular, it was shown that frequent stressful events precede periods of headache more often than periods of headache freedom, and that the strength of the relationship between these events and headache frequency is buffered by the level of self-control-efficacy. Thus, self-control-efficacy may operate as a psychological resource serving to reduce the frequency of headache in the face of stressful events. These results suggest that self-efficacy regarding control over behavioural, cognitive and affective responses to stressors could represent an important process variable in the successful psychological treatment of recurrent headache and may, thus, facilitate the formulation of theories of the therapeutic process which have been lacking in the literature concerning psychological treatments for headache (see Chapter 4).

The results reported in Chapter 8 are consistent with the central theory of headache and, thus, raise the possibility that the relationships between stressful events, self-control-efficacy, appraisal, coping and headache, observed in Chapter 7, may be mediated by the mechanism of sensory modulation.

Subjects with naloxone induced impairment of the endogenous opioid system, placed under conditions of cognitive stress (solving arithmetic problems under time pressure), have been shown not to generate the opioid activation demonstrated by subjects receiving injections
of inactive saline solution (Bandura et al., 1988). Furthermore, under conditions of cognitive stress, reductions in self-efficacy to manage stress have been associated with reductions in pain tolerance, for subjects administered naloxone; the direction of this relationship being reversed when subjects receive saline instead of the opioid antagonist (Bandura et al., 1988). These results suggest that if there is some impairment of the endogenous opioid system in headache sufferers (e.g., Genazzani et al., 1984; Sicuteri, 1982), the buffering effect of self-control-efficacy on the relationship between stressful events and headache may be mediated by this system. The existence of some impairment in the capacity to modulate somatosensory input in headache sufferers was suggested by the results obtained in Study III (see Chapter 8).

The formulation of theories linking psychological processes to headache through the mechanism of sensory modulation, may prove to be a more profitable line of inquiry than that offered by the emphasis of previous research upon the peripheral processes of muscle contraction and vasodilatation. However, this is not to say that peripheral processes are irrelevant to headache. The results obtained in Study III indicated that any weakness in the endogenous pain regulatory system is likely to be non-specific in its focus and, therefore, unable to give an account of the location of head pain. As suggested by Lance (1982), it seems that a complete account of the pathophysiology of headache is most likely to follow from a consideration of the interaction of peripheral processes with central pain control functions.

9.5. Directions For Further Research.

The studies reported in this thesis suggest a number of directions for further research. These are elaborated in the following paragraphs.

Much of the research into the rational-emotive theory of emotional distress has been plagued by the problem of defining the construct of rationality-irrationality in such a way that it can be regarded as conceptually distinct from the emotional distress it seeks to explain (Smith, 1982). In Chapter 6 it was argued that such a distinction may be achieved by following Ellis' (1962) definition of rationality as an acceptance of oneself, others and the environment, and by recognising irrationality as departures from this position. On the basis of this conceptualisation the Rationality Scale was constructed. The scale was found to have a high test-retest reliability and a high internal consistency. It was validated against the remainder of the items on the Irrational Beliefs Test and, as predicted by rational-emotive theory, was found to correlate moderately, and in the expected direction, with scores on the Beck Depression and Trait Anxiety Inventories. However, the claim that the instrument assesses a construct separate from that of emotional distress should be investigated further by means of a factor analytic
study, investigating whether or not the scale items load together on a factor separate from those upon which high loadings are observed for items tapping anxiety or depression.

In Study I (see Chapter 6) changes in Rationality Scale scores, over the course of rational-emotive therapy were observed to correlate significantly with reductions in anxiety and depression. No such relationships were observed for subjects receiving relaxation training. Such a pattern of results suggests that the significant correlations observed in the former treatment are unlikely to be explicable in terms of a confounding between rationality and emotional distress. Thus, the Rationality Scale may be a useful measure by which to examine hypotheses derived from rational-emotive theory. In particular, it is suggested that future research into the theory might profit from the use of this scale in treatment studies designed to examine the question of whether or not increases in rationality underlie the effects of rational-emotive therapy upon psychological disorders.

In Study II (see Chapter 7) the buffering effect of self-control-efficacy upon headache frequency was demonstrated. However, the mechanisms by which this buffering takes place remain unclear. The Self-Control-Efficacy Scale assessed efficacy regarding cognitive, behavioural and emotional control. An expansion of this scale, the development of a factored measure of each of the three components, and a replication of the buffering effect, may yield further information on the nature of this process. Particular aspects of self-control-efficacy might then be observed to be related to appraisal and coping responses. The relationship between self-control-efficacy and headache could also be explored further by tracking self-control-efficacy scores over the course of psychological treatments. In the light of the results reported in Chapter 7, significant correlations would be expected between reductions in headache frequency and increases in self-control-efficacy. It could also be hypothesised that since the relationship between stressful events and headache is not significant for headache sufferers high in self-control-efficacy, these subjects might not be good candidates for psychological treatments aimed at modifying the individual's reaction to such stressful events.

Direct and avoidance coping, in response to stressful events occurring during headache attacks, were found to be related to subsequent headache intensity. Tracking the relationship between these variables over the course of psychological treatments, would provide a method for assessing the direction of the relationship of each of these coping strategies to headache intensity following stressful events.

The question of whether particular patterns of coping are related to headache onset, subsequent to a stressful event, is also worthy of further investigation. In Study II (see Chapter 7) it was the number, rather than the type, of coping responses that was related to headache onset. The event-coping scales employed in the study were found to be moderately inter-
correlated. The development of more independent scales might facilitate the identification of particular coping methods conducive to headache onset or headache prevention.

The finding that headache attacks were less likely to follow stressful events appraised as important and generating higher levels of emotional upset, than events associated with lower scores on these variables, requires clarification. It would be important to compare headache sufferers' scores on these variables with those obtained by headache-free control subjects in order to determine whether such reactions to common stressful events are a characteristic of headache sufferers.

Future studies should employ more multifaceted measures of emotional distress in an effort to identify those types of emotional responses that may be understated by headache sufferers in periods preceding attacks. In Study II the emotional reaction to events was assessed by means of a single visual analogue scale. Future studies could assess a range of affective responses including anger, anxiety and depression. A focus upon anger would appear warranted given that migraine sufferers have been reported to express less anger than headache-free persons in competitive situations requiring subjects to solve anagrams rapidly (Grothgar and Scholz, 1987). A replication of Study II, addressing a broader range of emotional responses, might indicate that a failure to express anger, in stressful situations, increases the likelihood of headache occurring.

The headache sufferers participating in Study II were experiencing at least one headache per week. Such a level of headache frequency restricted the diary recording data to short cycles of only two days in length. Thus, it was not possible to study relationships between headache onset and psychological processes which may have occurred several days earlier. This could be accomplished by selecting headache sufferers with less frequent attacks and expanding the length of the cycles. In this manner any lagged effects of stressful events, appraisal, coping or emotional responses on the occurrence of headache attacks, could be examined.

Although the results obtained in Study III (see Chapter 8) indicated that a heightened sensitivity to pain may be a feature of headache sufferers, other studies have not reported such consistent findings (Drummond, 1987). It was suggested that the discrepancy between the results obtained in Study III and those reported by Drummond (1987) may have been due to an increased frequency of headache amongst the subjects studied in this thesis. Future studies of pain sensitivity in headache could address this hypothesis by assessing pain sensitivity across subjects with varying levels of headache frequency.

Any contribution of psychological variables to the heightened pain sensitivity observed for headache sufferers should also be explored. A methodology for assessing cognitive and
affective responses during experimental pain induction has been described by Spanos and his colleagues (Spanos et al., 1979, 1981). These researchers have shown that strategies which increase pain tolerance times are effective to the extent that they reduce catastrophising ideation (i.e., shift attention away from the aversive aspects of pain; see Appendix A). Since the central theory of headache assumes that the pain inhibitory system is deficient in headache sufferers (e.g., Sicuteri, 1982; Sicuteri, Anselmi and Del Bianco, 1978), it may be hypothesised that headache subjects would be less able than controls to increase their pain tolerance times, even if they were successful in reducing their level of catastrophising.

On the SSEP headache subjects were observed to have larger $P_1-N_1$ and $N_1-P_2$ amplitudes than controls and the $P_1-N_1$ amplitude was found to increase more rapidly with stimulus intensity for headache subjects. These results are consistent with the theory that some neurophysiological deficit in the capacity for sensory modulation may be a characteristic of headache sufferers (Sicuteri, 1982; Sicuteri, Anselmi and Del Bianco, 1978). However, the absence of consistent relationships between the SSEP components studied and measures of pain sensitivity, suggests that these two variables could represent independent processes, each of which could contribute in some way to the occurrence of headache attacks.

Persons demonstrating large increases in the size of the VEP with stimulus intensity have been observed to report greater frequencies of life events and higher levels of emotional distress in association with those events (von Knorring et al., 1980). Thus, the differences on the SSEP between the headache and control groups, observed in Study III, raise the possibility that the former group may be more reactive to stressful events and that this might contribute to headache. Such an hypothesis could be explored by observing whether or not the correlation between stressful events and headache is greater for headache sufferers with larger SSEP amplitudes than for those demonstrating smaller amplitudes. It would also be important to compare headache subjects with subjects from other chronic pain groups on the SSEP so as to determine whether the enlarged amplitudes observed for headache sufferers in Study III could be a result of repeated exposure to pain, rather than a phenomenon specific to the nervous systems of headache sufferers.

Exposing headache and headache-free control subjects to cognitive stress (such as mental arithmetic under time pressure), assessing pain tolerance pre- and post-stress and testing for opioid activation by administering naloxone or saline prior to the post-stress pain test (c.f. Bandura et al., 1988) would provide a test of the hypothesis that stress contributes to headache through deficient opioid mechanisms (Genazzani et al., 1984; Sicuteri, 1982). On the post-stress pain test, headache subjects would be expected to show smaller reductions in pain tolerance, following naloxone, than headache-free controls also receiving this drug, but to demonstrate greater reductions in pain tolerance than controls under conditions of inactive
saline administration. These results would be expected if there were some deficiency in the endogenous opioid system rendering headache sufferers susceptible to increases in pain sensitivity under stress (Sicuteri, 1982).

The release of catecholamines has been associated with the exposure of subjects to laboratory stress (Cox et al., 1983; Forsman and Lindblad, 1983) and with the onset of migraine headache (Anthony, 1981). Anthony (1981) observed increased blood levels of catecholamines to be associated with the release of free fatty acids which have been found, in turn, to release serotonin from platelets (Anthony, 1978), thereby reducing blood levels of the amine. Anthony (1981) suggested that the lower blood levels of serotonin could lead to headache by reducing the vascular tone of the cranial vessels. However, it is also possible that these processes act to reduce serotonin levels in the CNS and contribute to headache by opening the pain gate (see Chapter 2).

The contingent negative variation (CNV) is a negative potential recorded from the scalp, prior to the motor response in a reaction time task, and has been thought to be related to central catecholaminergic activity (Lance, 1987; Maertens de Noordhout, Timsit-Berthier and Shoenen, 1985). The CNV amplitude has been found to be greater for migraineurs than for tension headache or headache-free control subjects (Maertens de Noordhout et al., 1985). The enlarged SSEP components observed for headache sufferers in the present study could also be related to heightened activity within the sympathetic nervous system. This hypothesis could be explored by correlating indices of sympathetic activity, such as blood levels of noradrenaline, adrenaline or dopamine-beta-hydroxylase (Anthony, 1981) with the amplitude of the P1-N1 and N1-P2 components of the SSEP.


The main contribution of the current work has been to show that cognitive processes may be significant determinants of the frequency of headache attacks. In particular, evidence was obtained to suggest that stressful events may be causally related to headache onset and that self-control-efficacy may function to buffer this relationship. Appraising stressful events as amenable to change was also observed to mitigate against the onset of headache attacks. These observations lend some support to the widely held view that stressful events play an important part in the aetiology and / or maintenance of headache attacks (Friedman, 1979; Friedman et al., 1954; Levor et al., 1986) and also support the rationale for the application to headache of psychological treatments in general, and cognitive therapy in particular.

The current work was also concerned with exploring the prospects for linking psychological processes to the mechanism of sensory modulation outlined in the central theory
(Sicuteri, 1982). An evaluation of this theory was observed to support the hypothesis that headache sufferers may be deficient in their capacity to modulate sensory input. Although such a deficiency cannot, in itself, offer a full account of headache, there being a need to postulate the involvement of peripheral processes in order to explain the location of head pain (Lance, 1982), it was argued that it may be through an action on the mechanism of sensory modulation, rather than through an action on the mechanisms of muscle contraction or vasodilatation, that psychological processes contribute to recurrent headache. The development of theoretical formulations, postulating a mediational role for sensory modulation in the relationship between psychological processes, and headache may make for more profitable lines of inquiry than have followed from an emphasis upon peripheral mechanisms.
REFERENCES


APPENDIX A

THE ROLE OF COGNITIVE PROCESSES IN PAIN

OTHER THAN HEADACHE.

A.1. Defining Pain.

Although it is customary to open discussions of pain with definitions of the phenomenon (e.g., Beecher, 1959; Sternbach, 1968), these efforts rarely serve any useful purpose. As Lewis (1942, p. v.) noted:

"Reflection tells me that I am so far from being able to satisfactorily define pain...that the attempt would serve no useful purpose."

The International Association for the study of Pain (1979, p. 250) defined pain as:

"...an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such tissue damage."

This definition has been criticised for being vague about the dimensions that comprise the term "unpleasant" (Melzack and Wall, 1982). Furthermore, it would seem plausible for a person to experience pain which is not associated with any potential or actual tissue damage or described in terms of such damage. For example, the pain of tension headache is often described as "dull" (Hunter and Philips, 1981).

Psychology and physiology have not come to terms with the definition of pain, but then neither have physics nor chemistry come to grips with concepts such as "matter" or "energy" or "light", yet our understanding of the behaviour of these phenomena does not appear to have been hampered to any great extent by the absence of clear definitions. Pain cannot be defined satisfactorily at the present moment, but some appreciation of its vicissitudes follows from a consideration of theories of pain.

Specificity theory postulates the existence of a direct connection between specialised, pain transmitting receptors in the skin and a particular pain centre in the brain (e.g., von Frey, 1895). As Melzack (1973) has argued, such an hypothesis implies that the level of pain would always be directly proportional to the level of receptor stimulation. Often the degree of pain is not related to the degree of tissue damage (e.g., Beecher, 1959; Pavlov, 1927, 1928; see also Melzack, 1973) and this seriously undermines the specificity theory.

A.3. Pattern Theories of Pain.

The pattern theorists (e.g., Hebb, 1949; Livingston, 1943) proposed that sensory impulses enter a patterning mechanism before being relayed to the cortex. The patterning concept was introduced in order to account for individual differences in the relationship between injury and pain. It was suggested that, in pathological pain states where the level of pain exceeds the extent of any tissue damage, there is some abnormality in the patterning mechanism such that reverberatory activity within this structure summates (Livingston, 1943), or becomes synchronised (Hebb, 1949) with normally non-noxious impulses, to produce pain. Psychological factors, such as emotional disturbance, were thought to contribute to the pain syndrome by further activating abnormal neuronal activity within the patterning system, thought to exist in the spinal cord (Livingston, 1943) or in the thalamus (Hebb, 1949).

Pattern theories are challenged by the fact that spinal cord lesions often do not relieve pain (e.g., White and Sweet, 1969). Although it has been proposed that, despite surgery, there may be some "leak" in the pain transmission system (Noordenbos, 1959), it is difficult to appreciate how such small inputs could occasion severe pain (Melzack, 1973).

Pattern theories in general have been criticised for being vague about the processes involved in concepts such as summation and patterning (Uttal, 1973). The gate-control theory draws heavily from the pattern theories, but is much more specific about the processes involved in pain perception.


The gate-control theory of pain (Melzack, 1973; Melzack and Wall, 1965, 1982) was the first to recognise psychological processes as exerting an influence on afferent nerve impulses before they reach those areas of the brain subserving the experience of pain. The theory
developed out of the incapacity of the existing specificity (von Frey, 1895) and pattern (e.g., Hebb, 1949; Livingston, 1943) theories to account for such phenomena as the absence of pain despite considerable tissue damage, pain in the absence of tissue damage, and the fact that surgical lesions of the spinal cord often do not relieve pain (see Beecher, 1959; Melzack, 1973).

In order to account for the lack of concordance between injury and pain, Melzack and Wall (1965, 1982) conjectured that some neural mechanism must exist and function to modulate sensory input before it is experienced as pain. They considered the most likely site of this pain gate to be the substantia gelatinosa which runs the length of the spinal cord. According to the theory, injury activates the small-diameter (A-delta and C) fibres which enter the gating mechanism through the dorsal horns of the spinal cord and activate the second order neurons which pass those signals on to an action system. This action system was thought to consist of two subsystems:

1. The sensory-discriminative system, responsible for the localisation and sensory qualities of the pain.

2. The motivational-affective system, subserving the aversive, distressing aspects of pain experience.

The strength of the sensory impulses passing through the gate and activating the second order neurons is considered to be determined by the relative levels of activity in the large (A-beta) and small fibres such that, excitation of large fibres attenuates transmission (i.e., closes the gate), whilst small fibre activity facilitates transmission (i.e., opens the gate). Once the activity in the second order neurons exceeds a critical level, those impulses are interpreted by the brain as pain.

In addition to modulation of pain by the interaction of large and small fibres at the pain gate, Melzack and Wall (1965) postulated the existence of a central control system that could also act to open or close the pain gate and, thereby, modulate afferent input before it is processed by the brain. This process was thought to be mediated by projections from the reticular formation, limbic system and cerebral cortex to the substantia gelatinosa. Such modulation was invoked to account for the absence of pain despite injury.

In a later version of the theory (Melzack and Casey, 1968), it was proposed that central control processes also modulate pain by acting directly upon the sensory-discriminative and motivational-affective systems within the brain itself. This process was described as the cognitive-evaluative dimension of pain, involving the evaluation of the sensory and affective
experiences in terms of past experience and the meaning of the situation. All three dimensions were thought to interact in determining the experience of pain.

In more recent times, the existence of a pain inhibitory mechanism (Melzack and Wall, 1965) has been supported by the discovery of endogenous opioid substances in the brainstem and their analgesic effects (see Basbaum and Fields, 1978, 1984 for reviews). Although this research has led to some minor modifications to the gate control theory, the main tenets of the theory appear to have been well supported.

It has been suggested that psychological processes may exert an inhibitory effect on the pain gate, through the action of the endogenous opioids. For example, when naloxone (an opiate antagonist) or inactive substances are administered as placebos for clinical pain, only the inactive substance results in any improvement in pain; naloxone actually worsens the condition (e.g., Levine et al., 1978).

It was by postulating the existence of a pain modulating mechanism, and by stressing the extent of cortical involvement in the operation of that mechanism, that Melzack and Wall (1965) established the field of pain as a legitimate area for psychological investigation.

A.5. The Assessment of Pain.

Methods of assessing pain fall into one of two categories: those associated with laboratory-induced pain (often referred to as experimental pain), and those associated with clinical pain syndromes. In the former case, the experimenter is able to evaluate the subject's level of pain report against the intensity of a noxious stimulus; in the latter instance, only reports of subjective pain experience are available to the observer. These contrasting approaches to the measurement of pain are considered in the following sections.

A.5.1. Laboratory Methods for Producing Pain.

Researchers have employed a variety of noxious stimuli to produce pain, including the cold pressor test (Lovallo, 1975; Spanos et al., 1979), electrical stimulation (Bromm and Treede, 1980; Tursky, 1976), radiant heat (Hardy et al., 1952; Mor and Carmon, 1975), tourniquet ischemia (Moore, Duncan, Scott, Gregg and Ghia, 1979; Smith and Beecher, 1969), the sphygmomanometer cuff (Poser, 1962) and the pressure algometer (Keele, 1954).
Pain is induced through the sphygmomanometer cuff by placing a metal grater (Hollander, 1939), or sewing pointed projections (Poser, 1962), inside the cuff and inflating it around the subject's upper arm. However, this technique is not really very satisfactory as it leaves marks on the skin lasting for a number of days (Wolff, 1978). A similar method of pain induction is offered through tourniquet ischemia. In this method a tourniquet is tied around the subject's upper arm and the subject is required to engage in various exercises with his or her hand (Smith and Beecher, 1969). Although the technique has been claimed to be sensitive to the analgesic effects of morphine and aspirin (Smith and Beecher, 1969), other researchers have been unable to replicate these effects (Moore, Weissman, Thomas and Whitman, 1971). Furthermore, this method has been reported to demonstrate rather low reliability (Stembach, Deems, Timmermans and Huey, 1977), with the severity of pain depending very heavily upon how vigorously the subject performs the ischemic exercises (Moore et al., 1979).

The pressure algometer consists of a spring-loaded pressure gauge with a flat tipped plunger. The plunger is applied to the skin and pressure exerted until pain responses are obtained (Keele, 1954). However, this technique has been found to have lower reliability than other pain induction methods (Wolff, 1977), possibly because the rate of compression is uncontrolled (Procacci, Zoppi and Maresca, 1979).

The cold pressor test has been applied widely to the study of experimental pain (e.g., Friedman, Thompson and Rosen, 1985; McCaul and Haugtvedt, 1982). The procedure involves the subject in placing his / her hand in water maintained at a temperature of approximately 0°C. The dependent variables are time to the point where the subject indicates the sensation to be painful (pain threshold) and time to the point where the subject wishes to withdraw from the stimulus (pain tolerance). The technique has been shown to have good validity, being sensitive both to placebos and analgesics (e.g., Wolff et al., 1966, 1976). However, its reliability is limited by the fact that, in general, only one or two trials can be conducted as a period of several minutes is required before normal temperature and circulation is restored (Wolff, 1978).

In the radiant heat method, a high intensity beam of light is focused on a blackened area of the subject's skin (Hardy et al., 1952; Mor and Carmon, 1979). Although the procedure affords the experimenter a high degree of control over the level of stimulation, there is some question over its validity with equivocal results being reported in human analgesic assays (Kutscher and Kutscher, 1957; Wolff et al., 1976).

Electrical stimulation has been applied to the forearm (Tursky and O'Connell, 1972), the fingers (Bromm and Treede, 1980) and to the tooth pulp (Chapman, Chen and Bonica, 1977). The stimulus lends itself to a high degree of experimental control and several measures of pain
parameters can be obtained easily, making for excellent reliability (Tursky and O'Connell, 1972). Although electrical stimulation produces a novel sensation, very different from the sensory aspects of clinical pain (Wolff, 1978), this method has been found to be sensitive to the effects of analgesics (Wolff et al., 1966, 1976).

Electrical stimulation and the cold pressor test are the most commonly employed pain induction methods in laboratory pain studies involving human subjects. Both methods have been validated against analgesics but the ease with which electrical stimulation can be repeated makes for a higher level of reliability (Wolff, 1978).

The various parameters of pain, derived from the application of noxious stimuli, are considered in the following sections.

A.5.2. Pain Threshold.

The pain threshold is the point where the subject first perceives pain, on trials of ascending stimulus intensity or, in the case of descending intensity, the point where pain ceases. This measure has been criticised for being far removed from the anxiety and suffering associated with clinical pain (Beecher, 1959). However, pain thresholds have been found to be responsive to analgesics (Wolff et al, 1976) and to psychological manipulations such as relaxation training (Elton and Stanley, 1976) or cognitive strategies (Horan, Hackett, Buchanan, Stone and Demchik-Stone, 1977). Threshold reliabilities, for a variety of pain induction methods, have been found to vary from 0.65 for the pressure algometer to above 0.95 for electrical stimulation (Wolff, 1978).

A.5.3. Pain Tolerance.

Pain tolerance is the maximum stimulus intensity the subject is prepared to endure, or the length of time he / she will tolerate the stimulus. For this reason, it can only be assessed through an ascending series of trials. This measure has been thought to be more strongly related to clinical pain than the pain threshold (Beecher, 1966; Wolff, 1984). Measures of pain tolerance have been found to be responsive to analgesics such as aspirin, codeine and morphine (Wolff et al., 1966, 1976). The reliability of pain tolerance measures has been reported to vary from 0.81 for the pressure algometer to above 0.90 for electrical stimulation and radiant heat (Wolff, 1978).
A.5.4. Pain Sensitivity Range.

The pain sensitivity range is simply the arithmetical difference between the tolerance and threshold measures, but has not been employed as widely as the parameters from which it is derived. The pain sensitivity range has been found to be responsive to codeine and aspirin for cold pressor pain, but not for electrical pain (Wolff et al., 1976). It is yet to be determined whether or not the pain sensitivity range contributes any information on the response to experimental pain, over and above that already contained in the threshold and tolerance measures.

A.5.5. Ratings of Pain Intensity.

A number of researchers have assessed experimental pain by requiring subjects to give verbal ratings of their pain level at various intervals on 5 or 10 point scales. This method has been most commonly applied in studies utilising the cold pressor test (e.g., Farthing, Venturino and Brown, 1984; Girodo and Wood, 1979; Spanos, et al., 1979) possibly because, in this pain induction method, a sizeable proportion of subjects are able to tolerate the stimulus beyond the point where there is any further increase in pain (Davidson and McDougall, 1969).


Signal detection theory started as a technique for identifying a weak signal against background noise (Green and Swets, 1966). It was first applied to the study of human pain by Clark (1969). The appeal of the technique, as far pain researchers are concerned, is that it promises to separate the subject's sensitivity to the stimulus from his / her criterion for reporting it as painful. Each of these variables is recognised to contribute to measures of pain threshold and tolerance (Gelfand, 1964; Wolff, 1984).

In signal detection theory experiments, the signal plus noise and noise distributions are assumed to be of equal variance. The signal is selected so that it is not easily distinguished from the noise, thus, leading to a considerable overlap of the two distributions. It is assumed that, in a state of uncertainty, the observer adopts some decision rule. For example, he / she may attempt to minimise the number of false positives. This decision rule can be expressed as the ratio of the likelihood of sensation having arisen from the signal plus noise distribution to the likelihood of its having arisen from the noise distribution. In its application to the assessment of experimental pain, this measure is thought to indicate the subject's bias for reporting pain, whilst the distance between the means of the distributions is thought to indicate
how reliably he / she can discriminate between different categories of pain intensity (Chapman, Gehrig and Wilson, 1975; Clark, 1969; Clark and Goodman, 1974). The former measure is referred to as the criterion and the latter as the subject’s sensitivity or as $d'$. With the development of the signal detection methodology, it was hypothesised that only the criterion measure would be sensitive to psychological manipulations, whilst the $d$ measure would respond only to pharmacological manipulations (Clark, 1969). However, $d'$ has been found to be responsive to such psychological factors as anxiety (Schumacher and Veldon, 1984) and modelling (Craig and Cohen, 1975). The tranquillizer, diazepam, has been found to increase pain tolerance, but to have no effect on the criterion or $d'$ measures (Chapman and Feather, 1973). This latter finding raises the question of whether the parameters derived from signal detection theory are any more useful or valid than those obtained from the much simpler tolerance measure.

The aim of signal detection theory is to separate sensory and judgemental components of pain response (Clark, 1969). Certainly, when attempts are being made to detect a weak stimulus against background noise, these components can be separated. However, the assumption that this methodology can be extended to studies of pain, where the stimulus is often well above threshold levels, has been questioned (Rollman, 1977; Wolff, 1978).


The most common methods employed in the assessment of clinical pain are the verbal rating scale (Beecher, 1959), the visual analogue scale (Huskisson, 1974) and the McGill Pain Questionnaire (Melzack, 1975).

A.6.1. The Verbal Rating Scale.

The application of the verbal rating scale (VRS) to pain measurement was popularised by Beecher (1959) in his studies of the effects of various drugs on human pain. The scale typically consists of 5 to 7 categories carrying semantic labels reflecting increasing levels of pain experience. The subject indicates his / her current level of pain by selecting one of these categories.

A number of researchers have claimed this technique's sensitivity to analgesics as evidence for its validity (e.g., Lasagna, 1960; Loan, Morrison and Dundee, 1968). Subjects'
category judgements have also been reported to correlate with observer ratings of pain behaviour (Kast and Collins, 1966).

The VRS is used widely in studies of both acute (Wolff, 1978) and chronic (Dolce, Crocker and Doleys, 1986; Philips, 1987b) pain, including headache (Budzynski et al., 1973; Holroyd et al., 1984).

The VRS has been criticised for forcing subjects to translate a continuous sensation into discrete categories, the intervals between each of which cannot be demonstrated to be equivalent (Ohnhaus and Adler, 1975). The visual analogue scale which does not require category judgements has, therefore, been claimed as a superior measure (Huskisson, 1974).

A.6.2. The Visual Analogue Scale.

The visual analogue scale (VAS) consists of a straight line, the extremities of which are defined as the upper and lower limits of pain. For example, "no pain" and "pain as bad as it could be" (Scott and Huskisson, 1976, p. 176). Comparisons of the VRS and VAS have found scores on both scales to be highly correlated (e.g., Ohnhaus and Adler, 1975; Scott and Huskisson, 1976). However, there is some suggestion that scores on the latter may be more sensitive to reductions in the intensity of chronic pain following the administration of analgesics (Scott and Huskisson, 1976).

A.6.3. The McGill Pain Questionnaire.

Although the VAS and the VRS provide convenient measures of pain intensity, they each have the disadvantage of collapsing the multidimensional experience of pain into a unidimensional measure (Carlsson, 1983). Given the wide variation in the quality of pain that people experience, Melzack (1973, 1975) has argued that the unidimensional approach is seriously deficient:

"To describe pain solely in terms of intensity is like specifying the visual world only in terms of light flux without regard to pattern, colour, texture and the many other dimensions of visual experience." (Melzack, 1975, p. 278).

In order to assess the multidimensional experience of pain, Melzack (1975) developed the McGill Pain Questionnaire (MPQ). This consists of 78 verbal pain descriptors, organised into 20 categories designed to assess the discriminative, motivational-affective and evaluative qualities of pain. On the basis of the severity ratings assigned to each word by physicians,
patients and students, the words within each category were ordered in terms of intensity and assigned numerical values (Melzack, 1975). These numerical values provide measures of each of the three dimensions of pain postulated in the gate control theory (Melzack and Wall, 1965, 1982).

Factor analytic studies of the MPQ have consistently confirmed the existence of the discriminative (or sensory) and motivational-affective (or affective) dimensions (e.g., Crocket, Pricachin and Craig, 1977; Reading, 1979). Although these researchers were unable to substantiate the evaluative dimension, this component of the questionnaire has been identified in subsequent studies (Prieto et al., 1980; McCreary, Turner and Dawson, 1981).

The construct validity of the sensory and affective dimensions has been supported by studies showing that the sensory descriptors distinguish between pain syndromes (Dubuisson and Melzack, 1976; Leavitt and Garron, 1979), and by studies showing that the affective, but not the sensory descriptors, correlate with measures of emotional distress (McCreary et al., 1981; Parker, Doerfler, Tatten and Hewett, 1983).

The MPQ has been employed as an outcome measure in psychological treatments for chronic pain, and each of the three dimensions has been found to be sensitive to improvements in pain (e.g., Philips, 1987b; Rybstein-Blinchik, 1979). However, the extent to which each of the dimensions is reactive to particular interventions awaits further research.


Research on the role of cognitive processes in human pain may be divided into clinical and experimental studies.

In experimental pain studies the laboratory setting affords the experimenter control over the painful stimulus and makes for the manipulation of cognitive activity through the nature of the instructions offered to subjects (e.g., Farthing, Venvrino and Brown, 1984; Subotnik and Shapiro, 1984). However, the extent to which laboratory findings can be generalised to clinical pain has been questioned (e.g., Beecher, 1959). Experimental pain is usually of brief duration and does not generate the affective states of depression, demoralization or anxiety which often accompany chronic pain problems (Sternbach, 1974).

In clinical studies, ethical concerns make it difficult to assign subjects to no-treatment control groups and a number of strategies are often employed to try and help the suffering
patient, making it difficult to be specific about what exactly is being manipulated in the treatment (Weisenberg, 1984).


In studies involving experimental pain, cognitive activity has been manipulated by instructions (e.g., Friedman, Thompson and Rosen, 1985), suggestion (e.g., Feather, Chapman and Fisher, 1972), varying the level of perceived control over the stimulus (e.g., Glass et al., 1973) or teaching subjects cognitive coping strategies (e.g., McCaul and Haugtvedt, 1982). These studies have not typically assessed cognitive activity, but have assumed that the manipulations carried out have effected the desired cognitive change. Exceptions include studies by Spanos and his colleagues (Spanos, et al., 1979, 1981) who interviewed subjects about their ongoing cognitive activity during cold pressor pain, and studies suggesting that self-efficacy judgements for pain tolerance may be predictive of pain tolerance times (e.g., Dolce, Doleys et al., 1986; Litt, 1988; Vallis and Bucher, 1986).

The effects of the various cognitive manipulations on experimental pain are considered in the following sections.

A.7.1.1. Instructions.

Reports of pain have shown to be responsive to the instructions administered by the experimenter. For example, Wolff and his colleagues (Wolff and Horland, 1967; Wolff, Krasnegor and Farr, 1965) observed that, asking subjects to imagine that they would receive $1000 if they delayed terminating electrical stimulation for as long as possible, led to increases in pain tolerance relative to a control group. Similarly, asking subjects to be certain that the stimulus is painful, rather than strongly discomforting, has been found to result in an elevation of the pain threshold (Blitz and Dinnerstein, 1968). Indeed, it has been reported that the mere appearance of the word "pain", in a set of experimental instructions, can lower the pain threshold, pain tolerance and ratings of pain severity (Hall and Stride, 1954; Friedman et al., 1985).

A.7.1.2. Suggestion.

The administration of placebo medication with the suggestion that this will reduce pain, has been found to elevate the pain threshold (Clark, 1969; Feather et al., 1972). Suggestions
A.7.1.3. Perceived Control.

It has been reported that, when subjects believe that they have control over a noxious stimulus, this tends to increase pain tolerance levels (e.g., Bowers, 1968; Geer and Maisel, 1972; Staub, Tursky and Schwartz, 1971). However, many researchers have not found these manipulations to be effective in reducing ratings of pain intensity (e.g., Geer, Davison and Gatchel, 1970; Glass et al., 1973; Mills and Krantz, 1979). Weisenberg, Wolf, Mittwoch, Mikulincer and Aviram (1985) found that when subjects judged themselves as capable of regulating their own experience of pain, allowing them control over the noxious stimulus led to higher ratings of pain intensity than when control was in the hands of the experimenter. Conversely, when subjects judged themselves incapable of controlling their experience of pain, allowing them control over the stimulus was effective in lowering pain intensity ratings. These results highlight the need to consider individual differences in self-efficacy to manage pain when considering the effects of perceived control on pain perception.


Although such diverse cognitive strategies as hypnotic analgesia (Miller and Bowers, 1986), attention-focusing (Blitz and Dinnerstein, 1971), imagery (Westcott and Horan, 1977), distraction (McCaul and Hauftvedt, 1982), and self-statements (Girodo and Wood, 1979) have been reported to be effective in reducing experimental pain, reviews of the effectiveness of these techniques suggest that they may be no more effective than placebo or no-treatment control conditions (see Turk, 1978; Tan, 1982).

Spanos et al (1979) studied the effects of hypnosis and suggestion on cold pressor pain ratings and then interviewed subjects regarding their use of cognitive strategies. Hypnosis did not add to the effects of suggestion on pain report or strategy use. Strategy use was effective in lowering pain ratings only for those subjects whose cognitive activity did not focus on and exaggerate the aversiveness of the situation. In a subsequent study (Spanos et al., 1981), suggestions for analgesia was associated with reductions in cold pressor pain ratings, only for those subjects who were initially exaggerators but whose cognitive activity during the post-test
shifted such that, instead of focusing on aversive elements, they made positive self-statements or imagined events inconsistent with pain. Thus, individual differences in the tendency to catastrophise may be an important determinant of strategy effectiveness.

The existing literature on the role of cognitive appraisal processes in the perception of experimental pain suggests that although certain factors (such as suggestion, perceived control over pain and reliance upon cognitive strategies) may reduce pain for some subjects, more research is required into the role of individual differences in the determination of these effects.


The research on the role of cognitive appraisal processes in headache was discussed in Chapter 4. Accordingly, the following sections are confined to a consideration of pain syndromes other than headache.

A role for cognitive appraisal processes in clinical pain is suggested by the strength of the placebo effect in this area (Beecher, 1959, 1960; Evans, 1974a, b). For example, Beecher (1960) reviewed ten studies involving 831 patients complaining of headache, angina, cancer or post-operative pain and found that about 35% of patients reported pain relief following placebo medication.

Studies of cognitive factors in clinical pain may be subdivided into observational studies, where relationships between cognitive variables and pain are examined without intervention on the part of the experimenter (these studies have dealt almost exclusively with chronic pain) and treatment trials involving the manipulation of cognitive factors in a variety of chronic and acute pain syndromes.

A.7.2.1. Observational Studies on Chronic Pain.

Chronic pain has been described as pain which persists for six months or more (e.g., Hall, 1982; Webb, 1983). Most often, it is not accompanied by any detectable tissue damage (Stembach, 1968, 1974). Very few observational studies on cognitive appraisal and chronic pain have been carried out, thus, only the most tentative conclusions can be drawn.

It has been suggested that persons complaining of pain in the absence of any demonstrable organic pathology may be focusing their attention upon normal bodily sensations and, thus, amplifying their intensity (Barsky and Klerman, 1983). Employing the Body
Consciousness Questionnaire (Miller, Murphy and Buss, 1981), Ahles, Pecora and Riley (1987) observed that, within a sample of chronic pain patients, scores on private body consciousness were correlated positively with the level of pain report. Employing the Self-Consciousness Scale (Fenigstein, Scheir and Buss, 1975), Pennebaker and Skelton (1978) reported college students' scores on this scale to be correlated with summary scores on a measure of 12 physical symptoms including headache, chest pain and sore muscles. Similar results were reported by Ahles, Cassens and Stalling (1987). However, these correlational data make it impossible to draw any inferences regarding causality. It would be premature to regard the data as supportive of Barsky and Klerman's (1983) position, as it would seem equally likely that the presence of physical symptoms could lead persons to become more preoccupied with bodily sensations and, hence, to score higher on measures such as private self-consciousness.

Jensen, Karoly and Huger, (1987) developed the Survey of Pain Attitudes. This instrument assesses attitudes towards physicians and medical treatment, as well as levels of perceived social support, disability and the degree of control patients believe they have over their pain. These researchers obtained some evidence to suggest that attitudes may be predictive of pain coping behaviour amongst chronic pain patients. For example, the Pain Control Subscale was found to correlate positively with the use of active coping strategies, such as relaxation and exercise, amongst a group of hospitalized chronic pain patients. Relationships between attitudes to pain and pain experience were not explored.

The Coping Strategy Questionnaire (CSQ; Rosenstiel and Keefe, 1983) was developed to assess one behavioural strategy (increasing activity level) and six cognitive strategies (attention diversion, reinterpreting pain sensations, coping self-statements, praying or hoping and catastrophising) amongst chronic back pain patients.

Turner and Clancy (1986) administered the CSQ, together with the Sickness Impact Profile (Bergner, Bobbitt, Carter and Gilson, 1981), a measure of physical and psychosocial disability, and the Beck Depression Inventory (Beck et al., 1961), to chronic low back pain subjects. Subjects kept records of their pain for one week. Scores on the CSQ were found to account for significant proportions of the variance in depression and disability, but were unrelated to pain intensity ratings.

Keefe et al (1987) factor analysed the CSQ and identified two factors which they termed Coping Attempts, and Pain Control and Rational Thinking. Osteoarthritis patients scoring high on the latter factor were found to report lower pain levels, better health status and lower levels of psychological distress.
In a study involving arthritis patients, Shoor and Holman (1984) found that ratings of self-efficacy to control pain were correlated negatively with ratings of pain and disability obtained four weeks later.

The evidence obtained from observational studies does not make for any confident conclusion regarding the relationship between chronic pain and cognitive appraisal. There are suggestions that perceived control over pain (Keefe et al., 1987) and self-efficacy with respect to pain management (Shoor and Holman, 1984) might be associated with lower levels of pain, while a tendency to focus attention on bodily sensations might accompany increased pain ratings (e.g., Ahles, Cassens and Stalling, 1987; Pennebaker and Skelton, 1978). Much more research is required to substantiate these very tentative conclusions, and to address the issue of whether there exists any causal relation between the variables.

A.7.2.2. Cognitive Processes In The Treatment of Clinical Pain.

Attempts to relieve clinical pain through therapeutic procedures seeking to occasion some alteration in cognitive processes have dealt primarily with chronic, surgical and childbirth pain. The effects of these procedures on pain and cognitive processes are considered below.

Chronic Pain

Very few well controlled treatment studies involving cognitive interventions for chronic pain have been reported in the literature, making it difficult to offer any judgement on the efficacy of these cognitive techniques. Although a number of uncontrolled studies examining the effects on chronic pain of multi-faceted interventions, including some cognitive components, have been conducted (e.g., Gottlieb et al, 1977; Khatami and Rush, 1978; Sachs, Feurstein and Vitale, 1977; Stenn, Mothersill and Brooke, 1979), the absence of control and placebo-control groups, and the plethora of procedures employed in any one treatment approach, make it impossible to attribute the observed improvements in pain to any specific cognitive manipulation.

Turner and Clancy (1986) treated chronic pain patients with multi-faceted cognitive-behavioural therapy, or operant behavioural therapy or assigned them to a waiting list control condition. Changes in cognitive activity were assessed with the CSQ. Within the cognitive-behavioural group, there were significant increases in the use of coping self-statements and attention diversion strategies. Both experimental groups evidenced significant reductions in
catastrophising. A between-groups analysis of covariance demonstrated a greater increase in attention diversion for the cognitive-behavioural group than for the control group. The two experimental groups did not differ in their use of cognitive strategies over the course of treatment. Unfortunately, no between-groups analysis of pain intensity ratings was conducted. When the two treatment groups were combined, an increased use of praying and hoping strategies was positively correlated with reductions in pain intensity. A lesser reliance on catastrophising strategies was also associated with lower pain ratings and with decreased disability ratings.

Rybstein-Blinchik (1979) studied the effects on chronic pain of specific cognitive strategies, taught in groups, by assigning a mixed sample of chronic pain patients to one of the following conditions:

1. Control: Subjects shared their personal experiences of pain with other members of the group.
2. Somatisation: Subjects were instructed to relabel their pain as "a certain feeling", and to explore the sensations associated with that feeling.
3. Irrelevant Thoughts: Subjects were trained to replace any thoughts about pain with thoughts concerning other "important events" in their lives.
4. Relevant Thoughts: Subjects learned to reinterpret their experience of pain by relabelling the sensation as ticklish or numb.

At the post-treatment assessment, subjects in the Relevant Thoughts condition reported fewer pain behaviours and lower scores on the Sensory, Affective and Evaluative Scales of the MPQ than subjects in the remaining conditions. Subjects in the Relevant Thoughts condition also rated their pain as less intense than those in the Somatisation or Control groups, and ratings of pain intensity were lower in the Irrelevant Thoughts than in the Control condition. Although no follow-up assessment of patients was carried out, the findings do suggest that the cognitive relabelling of chronic pain, which is often employed in cognitive-behavioural treatments (e.g., Turk and Meichenbaum, 1984), may be of benefit, at least in the short term.

The effectiveness of distraction strategies, as employed in Rybstein-Blinchik's (1979) Irrelevant Thoughts condition, has been questioned by studies suggesting that these strategies may be associated with poorer treatment outcomes (Keefe and Dclon, 1986; Turner and Clancy, 1986).
Behavioural programmes for chronic pain, where subjects are required to increase their exercise levels gradually over the course of treatment, have been associated with increases in self-efficacy (Dolce, Crocker, and Doleys, 1986). These researchers offered chronic pain patients behavioural treatment and assessed their self-efficacy levels for exercise, work, and for coping with pain without medication at post-treatment. Follow-up data was collected six to twelve months after treatment. Post-treatment self-efficacy ratings were found to correlate significantly with follow-up measures of work status, exercise levels and medication consumption. Similar results have been obtained by other researchers (Kores et al., 1985), suggesting that self-efficacy expectations may be predictive of outcome from behavioural treatments for chronic pain.

Philips (1987b) examined relationships between perceived control over pain and outcome over the course of a cognitive-behavioural treatment for a mixed group of chronic pain patients. The subjects were assigned either to cognitive-behavioural treatment or to a waiting list control group. Although subjects receiving treatment demonstrated significant improvements on the MPQ and on diary recordings of pain intensity at post-treatment, eight week and one year follow-up assessments, no statistical analysis of differences between groups was reported. Perceived control over pain increased over the course of treatment, but this variable was not assessed in the control group.

Philips (1987b) studied relationships between concurrent measures of perceived control over pain and pain intensity. This raises the question of the extent to which her measure of perceived control over pain and ratings of pain severity were independent. If subjects undergo successful psychological treatment for pain, emphasising self-control techniques, it seems very likely that they might attribute reductions in pain to increases in control gained through treatment. Indeed, an interpretation of Philips' (1987b) results in terms of such confounding is suggested by the fact that her measures of perceived control and pain severity were uncorrelated at the pre-treatment assessment, but correlated 0.81 at the one year follow-up assessment.

Assessing cognitive constructs, such as perceived control or self-efficacy at post-treatment, and relating these measures to follow-up measures of pain (Dolce, Crocker and Doleys, 1986; Kores et al., 1985) provides a far more stringent test of hypotheses specifying relationships between these two sets of variables.

In conclusion, it would appear that self-efficacy to control pain may be predictive of outcome from behavioural treatments (Dolce, Crocker and Doleys, 1986; Kores et al., 1985). More well controlled studies, such as that of Rybstein-Blinchik (1979), are required before the effectiveness of particular cognitive interventions will be demonstrated.
Surgical Pain.

Research on the effects of cognitive factors on surgical pain has typically involved giving subjects preparatory information and/or teaching them cognitive coping skills (e.g., Johnson, Rice, Fuller and Endress, 1978; Wells, Howard, Nowlin and Vargas, 1986). Many of the studies conducted have not assessed subjective pain experience (e.g., Johnson, Morrisey and Leventhal, 1973; Johnson, Kirchoff and Endress, 1975). Those studies which have considered this dimension have not reported consistent effects for the procedures (e.g., Tan, Melzack and Poser, 1980; Wells et al., 1986).

Johnson and his colleagues (Johnson et al., 1973, 1975) compared information on likely sensations with procedural information given to subjects prior to undergoing painful medical procedures. Subjects receiving sensory information were rated as less distressed by observers, demonstrated lower heart rates and were less restless during the procedure than subjects receiving procedural information. Subjective ratings of pain were not obtained. When subjective pain has been studied procedural and sensory information have been found no more effective in reducing surgical or post-operative pain than placebo or no treatment control conditions (e.g., Johnson et al., 1978; Kendall et al., 1979).

Kendall et al (1979) compared cognitive-behavioural coping skills training with preparatory information for patients undergoing cardiac catheterisation. Attention-placebo and usual hospital routine control groups were also employed. Subjects in the two treatment groups reported lower levels of anxiety and were considered to have made a better adjustment to the procedure on the basis of "blind" observer ratings, than those in either of the control groups. However, no differences between groups emerged on patients' subjective pain ratings.

Stress Inoculation Training for pain control, which involves information and training in relaxation and a range of cognitive coping skills (Meichenbaum, 1975; Meichenbaum and Turk, 1976), has been applied to the management of surgical pain (e.g., Tan and Poser, 1982; Wells et al, 1986). Wells et al (1986) studied patients undergoing a variety of surgical procedures. Those receiving stress inoculation training reported lower levels of pain and anxiety after surgery than control subjects who simply went through the usual hospital routine. However, other studies have found Stress Inoculation Training to be no more effective in reducing ratings of surgical pain than control or placebo conditions (e.g., Tan, Melzack and Poser, 1980; Tan and Poser, 1982).

In general, studies of patients undergoing painful medical procedures have as yet provided no consistent evidence to indicate that specific cognitive manipulations have a reliable effect upon pain experience.
**Childbirth Pain.**

Preparatory childbirth training programmes have typically involved information, training in deep breathing, relaxation and / or attention focusing (e.g., Doering and Entwisle, 1975; Huttel, Mitchell, Fischer and Meyer, 1972; Scott and Rose, 1976). These authors have reported on the efficacy of such procedures, relative to control conditions, on outcome measures such as maternal satisfaction with the delivery. Other studies have reported that preparatory techniques reduce the amount of medication consumed during labour and delivery relative to controls (e.g., Chertok, 1969; Enkin, Smith, Dermer and Emmett, 1972). Manning and Wright (1983) found that self-efficacy to manage childbirth, following training, was predictive of medication consumption during labour and of medication latency during delivery. However, none of these studies assessed maternal pain experience. Thus the relationship between cognitive processes and childbirth pain has yet to be thoroughly explored (see Tan, 1982 for a review).

In concluding the literature on intervention studies in chronic and acute clinical pain (excluding the literature on the treatment of headache dealt with in Chapter 4), it is apparent that there is insufficient evidence to support the claim that training in cognitive strategies is any more effective in modifying pain than placebo conditions. Unfortunately, this conclusion is not based on the existence of well controlled studies; rather, it follows from the absence of adequate controls in studies of chronic pain and from the failure of many researches to include subjective pain ratings as outcome measures in studies of acute pain.

Within the context of cognitive and behavioural treatments for clinical pain, there is some evidence suggesting that self-efficacy to manage pain may be predictive of outcome from behavioural treatment programmes (Dolce, Crocker and Doleys, 1986; Kores et al., 1985). However, the role of self-efficacy has yet to be examined in studies investigating whether or not treatments, aimed at increasing self-efficacy, are any more effective in relieving clinical pain than placebo conditions with no specific focus on this construct.

**A.8. General Comments.**

Some evidence in support of the premise that cognitive factors contribute to pain perception comes from studies of experimental pain. Suggestions for pain relief have been found to reduce pain report (e.g., Dworkin et al., 1984; Feather et al., 1972) and self-efficacy judgements regarding pain management skills may be predictive of pain tolerance levels (e.g., Dolce, Doleys et al., 1986; Litt, 1988; Vallis and Bucher, 1986). A pre-occupation with the
aversive elements of experimental pain (catastrophising) may be associated with a reduced capacity to tolerate pain (Spanos et al., 1979, 1981).

The literature on clinical pain is characterised by a shortage of adequately controlled studies. Often, researchers have not assessed subjective pain experience and have relied instead upon less direct measures such as medication consumption (e.g., Johnson et al., 1973, 1975; Manning and Wright, 1983). No conclusion can be offered about the relative efficacy of particular cognitive strategies. Most of the evidence for the role of cognitive factors in clinical pain derives from studies of the placebo effect (e.g., Beecher, 1959, 1960; Evans, 1974a, b), which have not attempted to identify the particular cognitive processes involved. Some recent work suggests that increases in self-efficacy to manage pain may be associated with better treatment outcomes for chronic pain sufferers (e.g., Dolce, Crocker and Doleys, 1986; Kores et al., 1985).
APPENDIX B

DEVELOPMENT OF THE RATIONALITY SCALE

Instructions:

This is an inventory of the way you think about various things. There are a number of statements with which you will tend to agree or disagree. You will be given an answer sheet and asked to circle one of five possible answers to each item. For each statement, you should mark your answer sheet as follows, according to your own reaction to the item:

Circle D if you STRONGLY DISAGREE
Circle d if you MODERATELY DISAGREE
Circle n if you NEITHER AGREE NOR DISAGREE
Circle a if you MODERATELY AGREE
Circle A if you STRONGLY AGREE

It is not necessary to think over any item very long. Mark your answer quickly and go on to the next statement.

Try to avoid the neutral or "n" response as much as possible. Select this answer only if you really cannot decide whether you tend to agree or disagree with a statement.

Before you start, be sure to print your name, sex, and age at the top of the answer sheet where indicated. In the space for "grade", place the number of the highest school grade completed. If you have had college or university, one year would be 13, four years would be 16, etc.

Irrational Beliefs Test Items (Jones, 1968):

1. It is important to me that others approve of me. (I).
2. I hate to fail at anything. (E).
4. I usually accept what happens philosophically. (R)*.

5. If a person wants to, he can be happy under almost any circumstances. (R)*.

6. I have a fear of some things that often bothers me. (E).

7. I usually put off important decisions. (I).

8. Everyone needs someone he can depend on for help and advice. (I).


10. There is a right way to do everything. (I).

11. I like the respect of others, but I don't have to have it. (R)*.

12. I avoid things I cannot do well. (I).

13. Too many evil persons escape the punishment they deserve. (I).

14. Frustrations don't upset me. (E).

15. People are disturbed not by situations but by the view they take of them. (R).

16. I feel little anxiety over unexpected dangers or future events. (E).

17. I try to go ahead and get irksome tasks behind me when they come up. (R).

18. I try to consult an authority on important decisions. (I).

19. It is almost impossible to overcome the influences of the past. (I).

20. There is no perfect solution to anything. (R).

21. I want everyone to like me. (I).
22. I don't mind competing in activities where others are better than I. (R)*.
23. Those who do wrong deserve to be blamed. (I).
24. Things should be different from the way they are. (I).
25. I cause my own moods. (R).
26. I often can't get my mind off some concern. (E).
27. I avoid facing my problems. (I).
28. People need a source of strength outside themselves. (I).
29. Just because something once strongly affects your life doesn't mean it need do so in the future. (R).
30. There is seldom an easy way out of life's difficulties. (R).
31. I can like myself even when many others don't. (R)*.
32. I like to succeed at something but I don't feel I have to. (R)*.
33. Immorality should be strongly punished. (I).
34. I often get disturbed over situations I don't like. (E).
35. People who are miserable have usually made themselves that way. (R).
36. If I can't keep something from happening, I don't worry about it. (E).
37. I usually make decisions as promptly as I can. (R).
38. There are certain people that I depend on greatly. (I).
39. People over-value the influence of the past. (I).
40. Some problems will always be with us. (R).

41. If others dislike me, that's their problem, not mine. (R).

42. It is highly important to me to be successful in everything I do. (I).

43. I seldom blame people for their wrongdoings. (R).

44. I usually accept things the way they are, even if I don't like them. (R)*.

45. A person won't stay angry or blue long unless he keeps himself that way. (R).

46. I can't stand to take chances. (E).

47. Life is too short to spend it doing unpleasant tasks. (I).

48. I like to stand on my own two feet. (R).

49. If I had had different experiences I could be more like I want to be. (I).

50. Every problem has a correct solution. (I).

51. I find it hard to go against what others think. (I).

52. I enjoy activities for their own sake, no matter how good I am at them. (R)*.

53. The fear of punishment helps people be good. (I).

54. If things annoy me, I just ignore them. (E).

55. The more problems a person has, the less happy he will be. (I).

56. I am seldom anxious over the future. (E).

57. I seldom put things off. (R).
58. I am the only one who can really understand and face my problems. (R).

59. I seldom think of past experiences as affecting me now. (R).

60. We live in a world of chance and probability. (R).

61. Although I like approval, it's not a real need for me. (R)*.

62. It bothers me when others are better than I am at something. (E).

63. Everyone is basically good. (R)*.

64. I do what I can to get what I want and then don't worry about it. (E).

65. Nothing is upsetting in itself - only in the way you interpret it. (E).

66. I worry a lot about certain things in the future. (E).

67. It is difficult for me to do unpleasant chores. (I).

68. I dislike for others to make my decisions for me. (R).

69. We are slaves to our personal histories. (I).

70. There is seldom an ideal solution to anything. (R).

71. I often worry about how much people approve of and accept me. (E).

72. It upsets me to make mistakes. (E).

73. It's unfair that "the rain falls on both the just and the unjust". (I).

74. I am fairly easy going about life. (R)*.

75. More people should face up to the unpleasantness of life. (I).

76. Sometimes I can't get a fear off my mind. (E).
77. A life of ease is seldom very rewarding. (R).

78. I find it easy to seek advice. (I).

79. Once something strongly affects your life, it always will. (I).

80. It is better to look for a practical solution than a perfect one. (R).

81. I have considerable concern with what people are feeling about me. (E).

82. I often become quite annoyed over little things. (E).

83. I usually give someone who has wronged me a second chance. (R).

84. I dislike responsibility. (I).

85. There is never any reason to remain sorrowful for very long. (E).

86. I hardly ever think of such things as death or atomic war. (I).

87. People are happiest when they have challenges and problems to overcome. (R).

88. I dislike having to depend on others. (R).

89. People never change basically. (I).

90. I feel I must handle things in the right way. (I).

91. It is annoying but not upsetting to be criticised. (E).

92. I'm not afraid to do things which I cannot do well. (E).

93. No one is evil, even though his deeds may be. (R)*.

94. I seldom become upset over the mistakes of others. (E).
95. Man makes his own hell within himself. (R).

96. I often find myself planning what I would do in different dangerous situations. (I).

97. If something is necessary, I do it even if it is unpleasant. (R).

98. I've learned not to expect someone else to be very concerned about my welfare. (I).

99. I don't look upon the past with any regrets. (E).

100. There is no such thing as an ideal set of circumstances. (R).

* Indicates one of the 12 items on the Rationality Scale.

(E) Indicates items involving the self-disclosure of emotional states.

(I) Indicates remaining items scored in the direction of irrationality.

(R) Indicates remaining items scored in the direction of rationality.

Note: The above codes were not included in the questionnaire presented to subjects.
# IRRATIONAL BELIEFS TEST
## ANSWER SHEET

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<tr>
<td>98</td>
<td>D</td>
<td>d</td>
<td>n</td>
<td>a</td>
<td>A</td>
</tr>
<tr>
<td>99</td>
<td>D</td>
<td>d</td>
<td>n</td>
<td>a</td>
<td>A</td>
</tr>
<tr>
<td>100</td>
<td>D</td>
<td>d</td>
<td>n</td>
<td>a</td>
<td>A</td>
</tr>
</tbody>
</table>
Internal Consistency of The Rationality Scale.

The internal consistency of the Rationality Scale was assessed by means of the item-total correlation analysis presented below (N = 36). The item numbers are those assigned to the items on the Irrational Beliefs Test.

<table>
<thead>
<tr>
<th>Item Number</th>
<th>Mean</th>
<th>S.D.</th>
<th>Corrected Item-Total Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>2.61</td>
<td>1.32</td>
<td>0.50</td>
</tr>
<tr>
<td>5</td>
<td>2.58</td>
<td>1.27</td>
<td>0.28</td>
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<tr>
<td>11</td>
<td>2.39</td>
<td>1.20</td>
<td>0.53</td>
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<tr>
<td>22</td>
<td>2.31</td>
<td>1.19</td>
<td>0.38</td>
</tr>
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<td>31</td>
<td>2.56</td>
<td>1.16</td>
<td>0.43</td>
</tr>
<tr>
<td>32</td>
<td>2.86</td>
<td>1.33</td>
<td>0.60</td>
</tr>
<tr>
<td>44</td>
<td>2.86</td>
<td>1.20</td>
<td>0.49</td>
</tr>
<tr>
<td>52</td>
<td>2.56</td>
<td>1.30</td>
<td>0.61</td>
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<tr>
<td>61</td>
<td>2.94</td>
<td>1.19</td>
<td>0.49</td>
</tr>
<tr>
<td>63</td>
<td>2.75</td>
<td>1.11</td>
<td>0.29</td>
</tr>
<tr>
<td>74</td>
<td>3.17</td>
<td>1.25</td>
<td>0.48</td>
</tr>
<tr>
<td>93</td>
<td>2.97</td>
<td>1.13</td>
<td>0.53</td>
</tr>
</tbody>
</table>

The above analysis yielded a reliability coefficient alpha of 0.81.
APPENDIX C

HEADACHE RECORDING DIARY (CHAPTER 6)

Each diary consisted of 7 sheets of which the following is an example:

HEADACHE RECORDING SHEET

Name: Date: 

<table>
<thead>
<tr>
<th>Headache as</th>
<th>Could be</th>
</tr>
</thead>
<tbody>
<tr>
<td>No</td>
<td>Headache</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

am 6 _________________________________ 7 _________________________________ 8 9 10 11 12

pm 1 _________________________________ 2 _________________________________ 3 4 5 6 7 8 9 10 11 12

am 1 _________________________________ 2 _________________________________ 3 4 5

Medication (type and amount) .........................
Situation ............................................
Each Headache Recording Diary was prefaced by the following instructions sheet:

HEADACHE RECORDING INSTRUCTIONS

Name: 

Date: 

Record the intensity of your headaches for each hour of the day on the forms provided. A mark on the line towards the left represents a headache of lesser intensity and a mark towards the right represents a headache of greater intensity.

If you experience a completely headache-free day, write HEADACHE FREE on the form for that day.

If you have any problems, please phone Nicholas Francis-Jones at the Australian National University on 49-4003.
APPENDIX D

TREATMENT CREDIBILITY RATING SCALES

On the rating scales below, please indicate the following:

1) The likelihood of your recommending the treatment to a friend suffering from headaches.

I definitely would not recommend the treatment

I definitely would recommend the treatment

2) How important do you think it is that the treatment be made available to other headache sufferers?

Unimportant

Very Important
# APPENDIX E

**COMPARISON OF PRE-TREATMENT VARIABLE VALUES FOR RATIONAL-EMOTIVE (n = 14) AND RELAXATION (n = 15) GROUPS**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Rational-Emotive</th>
<th>Relaxation</th>
<th>t-value (df=27)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>39.14 (10.11)</td>
<td>42.20 (10.71)</td>
<td>0.79</td>
</tr>
<tr>
<td>Years of Education</td>
<td>14.14 (3.74)</td>
<td>14.13 (3.16)</td>
<td>0.01</td>
</tr>
<tr>
<td>Headache Frequency</td>
<td>5.21 (1.57)</td>
<td>4.50 (1.72)</td>
<td>1.17</td>
</tr>
<tr>
<td>Headache Duration</td>
<td>7.59 (4.06)</td>
<td>7.57 (3.18)</td>
<td>0.02</td>
</tr>
<tr>
<td>Headache Intensity</td>
<td>20.20 (12.37)</td>
<td>17.76 (12.65)</td>
<td>0.52</td>
</tr>
<tr>
<td>Medication*</td>
<td>1.91 (1.41)</td>
<td>1.64 (1.22)</td>
<td>0.55</td>
</tr>
<tr>
<td>Rationality</td>
<td>32.71 (8.37)</td>
<td>31.00 (8.56)</td>
<td>0.54</td>
</tr>
<tr>
<td>Beck Depression+</td>
<td>1.88 (0.96)</td>
<td>1.88 (0.77)</td>
<td>0.00</td>
</tr>
<tr>
<td>Trait Anxiety *</td>
<td>43.58 (10.40)</td>
<td>41.33 (9.85)</td>
<td>0.57</td>
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</tbody>
</table>

**Note:** The standard deviations are given in brackets.

* Indicates degrees of freedom = 25 (two values were missing for the rational-emotive group).

† Indicates that these variables were observed to be skewed and thus subjected to the transformation \( \ln (\text{variable} + 1) \) prior to the analysis.

None of the obtained t-values reached the 0.05 level of significance (2-tailed test).
APPENDIX F

SUMMARY TABLES FOR RESULTS OF ANALYSES OF COVARIANCE BETWEEN RATIONAL-EMOTIVE THERAPY AND RELAXATION TRAINING GROUPS ON POST-TREATMENT AND FOLLOW-UP HEADACHE ACTIVITY MEASURES WITH PRE-TREATMENT VALUES AS COVARIATES

Analysis of Post-Treatment Scores (N = 29)

Headache Frequency:

<table>
<thead>
<tr>
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<th>DF</th>
<th>MS</th>
<th>F-value</th>
<th>p-value</th>
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</thead>
<tbody>
<tr>
<td>Covariate</td>
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<td>53.63</td>
<td>16.92</td>
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<tr>
<td>Group</td>
<td>1</td>
<td>0.66</td>
<td>0.21</td>
<td>0.652</td>
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<tr>
<td>Error</td>
<td>26</td>
<td>3.17</td>
<td></td>
<td></td>
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Headache Intensity:

<table>
<thead>
<tr>
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<th>DF</th>
<th>MS</th>
<th>F-value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Covariate</td>
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<td>2100.76</td>
<td>28.12</td>
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<td>Group</td>
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<td>324.20</td>
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<td>Error</td>
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<td>74.71</td>
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Headache Duration:

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<tr>
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<th>MS</th>
<th>F-value</th>
<th>p-value</th>
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</thead>
<tbody>
<tr>
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<td>20.51</td>
<td>0.000</td>
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<tr>
<td>Group</td>
<td>1</td>
<td>18.66</td>
<td>1.94</td>
<td>0.176</td>
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<tr>
<td>Error</td>
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<td>9.63</td>
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<td></td>
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Medication Intake:

<table>
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<th>F-value</th>
<th>p-value</th>
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</thead>
<tbody>
<tr>
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<td>0.22</td>
<td>1.33</td>
<td>0.72</td>
</tr>
<tr>
<td>Group</td>
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<td>0.13</td>
<td>0.08</td>
<td>0.78</td>
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<tr>
<td>Error</td>
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<td>1.67</td>
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</table>

Analysis of Follow-Up Scores (N = 23)

Headache Frequency:

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<th>F-value</th>
<th>p-value</th>
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</thead>
<tbody>
<tr>
<td>Covariate</td>
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<td>21.54</td>
<td>8.96</td>
<td>0.007</td>
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<tr>
<td>Group</td>
<td>1</td>
<td>0.49</td>
<td>0.20</td>
<td>0.656</td>
</tr>
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<td>Error</td>
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### Headache Intensity:

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<thead>
<tr>
<th>SOURCE</th>
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<th>p-value</th>
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</thead>
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<tr>
<td>Covariate</td>
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</tr>
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<td>82.19</td>
<td>1.00</td>
<td>0.330</td>
</tr>
<tr>
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<td>82.30</td>
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### Headache Duration:

<table>
<thead>
<tr>
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<th>F-value</th>
<th>p-value</th>
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<tbody>
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<td>Covariate</td>
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<td>132.03</td>
<td>14.77</td>
<td>0.001</td>
</tr>
<tr>
<td>Group</td>
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<td>22.10</td>
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<td>0.131</td>
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<tr>
<td>Error</td>
<td>20</td>
<td>8.94</td>
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### Medication Intake:

<table>
<thead>
<tr>
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<th>p-value</th>
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<tbody>
<tr>
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<td>1.94</td>
<td>1.48</td>
<td>0.238</td>
</tr>
<tr>
<td>Group</td>
<td>1</td>
<td>0.22</td>
<td>0.17</td>
<td>0.684</td>
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<tr>
<td>Error</td>
<td>20</td>
<td>1.31</td>
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<td></td>
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</table>
### APPENDIX G

SUMMARY TABLES FOR RESULTS OF ANALYSES OF COVARIANCE BETWEEN RATIONAL-EMOTIVE THERAPY AND RELAXATION TRAINING GROUPS ON POST-TREATMENT RATIONALITY, BECK DEPRESSION AND TRAIT ANXIETY SCORES WITH PRE-TREATMENT SCORES AS COVARIATES

#### Rationality Scale (N = 29)

<table>
<thead>
<tr>
<th>SOURCE</th>
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<th>MS</th>
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<th>p-value</th>
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<td>Group</td>
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<td>Error</td>
<td>26</td>
<td>16.76</td>
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</table>

#### Beck Depression Scale (N = 29)

<table>
<thead>
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<th>p-value</th>
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<td>Group</td>
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<td>0.123</td>
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<td>Error</td>
<td>26</td>
<td>0.52</td>
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<td></td>
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#### Trait Anxiety Scale (N = 27)

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<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Covariate</td>
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<td>506.08</td>
<td>12.62</td>
<td>0.002</td>
</tr>
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<td>Group</td>
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<td>0.60</td>
<td>0.445</td>
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<tr>
<td>Error</td>
<td>24</td>
<td>40.11</td>
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<td></td>
</tr>
</tbody>
</table>
APPENDIX H

SELF-CONTROL-EFFICACY SCALE

Please indicate the extent to which you agree or disagree that each of the following items are descriptive of yourself by circling the appropriate letter:

A = Strongly Agree.
a = Somewhat Agree.
d = Somewhat Disagree.
D = Strongly Disagree.

1. If I sense that I'm about to act too rashly or impulsively, I am usually able to stop myself.

2. Quite often I cannot overcome unpleasant thoughts that bother me.

3. Once I know what has to be done, I am usually confident about my ability to carry it through.

4. I cannot avoid thinking about mistakes I have made in the past.

5. When I get angry about something, I can usually get over those feelings quickly once the situation has passed.

6. Although it makes me feel bad, I cannot avoid thinking about all kinds of possible catastrophes in the future.

7. I often find it difficult to overcome my feelings of nervousness and tension without outside help.
8. When I feel depressed, I can usually do something to make myself feel better.  

9. I need outside help to get rid of some of my bad habits.

Note: Items 1, 2, 4, 6, 7 and 9 are reverse score items.
Internal Consistency of the Self-Control-Efficacy Scale:

The internal consistency of the scale was assessed by means of the item-total correlation analysis presented below (N = 120).

<table>
<thead>
<tr>
<th>Item Number</th>
<th>Mean</th>
<th>S.D.</th>
<th>Corrected Item-Total Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>3.09</td>
<td>0.88</td>
<td>0.15</td>
</tr>
<tr>
<td>2</td>
<td>2.27</td>
<td>1.06</td>
<td>0.58</td>
</tr>
<tr>
<td>3</td>
<td>3.48</td>
<td>0.78</td>
<td>0.31</td>
</tr>
<tr>
<td>4</td>
<td>2.37</td>
<td>1.04</td>
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<tr>
<td>5</td>
<td>2.78</td>
<td>1.06</td>
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<td>2.88</td>
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<td>8</td>
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<tr>
<td>9</td>
<td>2.93</td>
<td>1.08</td>
<td>0.46</td>
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</tbody>
</table>

The above analysis yielded a reliability coefficient alpha of 0.76.
APPENDIX I

SELF-MONITORING DIARY

The Self-Monitoring Diary consisted of the instructions sheet given below plus seven sheets with each of the two subsequent pages of this Appendix (see over) printed on each side.

Name:............................

Date:...........

Please record the intensity of your headaches for each hour of the day on the form. Indicate the level of headache pain for each hour by placing a cross (x) under the number which best describes the intensity of your headache at that time. The scale is as follows:

0 = No Headache.
1 = Barely Noticeable Headache.
2 = Mild Headache.
3 = Moderate Headache.
4 = Severe Headache.
5 = Extremely Intense Headache.

If you experience a completely headache free day, write - HEADACHE FREE on the form for that day.

It is important that headaches be recorded AS THEY OCCUR, rather from memory. Thus, please try to keep the diary with you at all times.

At the end of each day, please complete the EVENT RECORDING section of the form. If an irritating or upsetting event did occur during the day, please be sure to answer ALL of the items on BOTH SIDES of the form. If more than one such event occurred, choose the one event that was most upsetting.

If you have any problems, please phone Nicholas Francis-Jones at Prince Henry Hospital on 694-5611 or leave a message on 694-5680. Thankyou.
**EVENT RECORDING**

1. Was there anything today that you found at all irritating or upsetting? This can be an event that actually occurred today, or one that you recalled from the past or some future event that you thought about. 
   Please circle your answer: YES NO 

If NO, please stop at this point. If YES, please be sure to complete ALL of the following items:

2. Briefly describe the event. 

3. Roughly, at what time did this event start? .......am/pm (time to nearest hour and cross out that which does not apply).

4. Please indicate how much this event MATTERED to you AT THE TIME by circling the appropriate number: 
   NOT MUCH .................. 1 
   SOMEWHAT .................. 2 
   QUITE A BIT ................ 3 
   A LOT  ..................... 4 

5. Please indicate how much you thought you could CHANGE this event AT THE TIME it occurred: 
   NOT AT ALL .................. 0 
   A LITTLE ................... 1 
   SOMEWHAT .................. 2 
   A LOT  ..................... 3 

**IMPORTANT :** THERE IS MORE ON THE OTHER SIDE! PLEASE TURN OVER THE PAGE AND BE SURE TO ANSWER ALL OF THE ITEMS

---

**HEADACHE RECORDING**

<table>
<thead>
<tr>
<th>Time</th>
<th>Intensity</th>
<th>Type</th>
<th>Quantity</th>
</tr>
</thead>
<tbody>
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<td>2</td>
</tr>
<tr>
<td>6</td>
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</table>

Today's medication 
Type ............
Quantity ..........
EVENT COPING SCALE:

(6) Listed below are some ways in which people react to events. Please read each item and indicate, by circling YES or NO, whether or not you reacted this way to the event you described. Please answer every item:

1. Doubled my efforts and tried harder to make things work out. YES NO
2. Postponed dealing with the problem. YES NO
3. Avoided discussion of the issue. YES NO
4. Wished that I could change what had happened. YES NO
5. Tried to relax myself. YES NO
6. Avoided things that reminded me of the problem. YES NO
7. Wished the situation would go away or somehow be over with. YES NO
8. Fantasized about how things might turn out. YES NO
9. Talked to someone who could do something about the problem. YES NO
10. Tried to find out more about the situation. YES NO
11. Went over in my mind what I would say or do. YES NO
12. Changed something to try and make things work out. YES NO
13. Reassured myself that things would work out all right. YES NO
14. Tried to view the situation as a challenge. YES NO
15. Wished I was a stronger person - more optimistic and forceful. YES NO
16. Reminded myself how much worse things could be. YES NO
17. Tried to think of a number of ways to sort out the problem. YES NO
18. Concentrated on something good that could come out of the whole thing. YES NO
19. Even though the problem had not been sorted out, I tried to forget about the whole thing. YES NO

(7) On the scale below, please indicate how upset you felt during the event you described. Do this by placing a small vertical line across the scale at the point that represents how upset you felt during the event:

Not at all upset As upset as I have ever been

(8) Did you have a headache today? YES NO (circle answer)

If YES, did the EVENT you described start:

BEFORE the headache DURING the headache AFTER the headache had gone

(Please CIRCLE your answer)
**Internal Consistency of Event Coping Subscales:**

The internal consistency of the Event Coping Subscales was assessed by means of the item-total correlation analyses presented below ($N = 935$):

**Direct Coping Scale**

<table>
<thead>
<tr>
<th>Item Number</th>
<th>Mean</th>
<th>S.D.</th>
<th>Corrected Item-Total Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.47</td>
<td>0.50</td>
<td>0.38</td>
</tr>
<tr>
<td>9</td>
<td>0.38</td>
<td>0.49</td>
<td>0.43</td>
</tr>
<tr>
<td>10</td>
<td>0.42</td>
<td>0.49</td>
<td>0.52</td>
</tr>
<tr>
<td>11</td>
<td>0.54</td>
<td>0.50</td>
<td>0.38</td>
</tr>
<tr>
<td>12</td>
<td>0.37</td>
<td>0.48</td>
<td>0.49</td>
</tr>
<tr>
<td>17</td>
<td>0.61</td>
<td>0.49</td>
<td>0.51</td>
</tr>
</tbody>
</table>

The above analysis yielded a KR-20 reliability coefficient of 0.72.
### Avoidance Coping Scale

<table>
<thead>
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<th>Mean</th>
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<th>Corrected Item-Total Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>0.21</td>
<td>0.40</td>
<td>0.37</td>
</tr>
<tr>
<td>3</td>
<td>0.24</td>
<td>0.43</td>
<td>0.34</td>
</tr>
<tr>
<td>4</td>
<td>0.68</td>
<td>0.47</td>
<td>0.30</td>
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<tr>
<td>6</td>
<td>0.27</td>
<td>0.44</td>
<td>0.41</td>
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<tr>
<td>7</td>
<td>0.78</td>
<td>0.42</td>
<td>0.35</td>
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<tr>
<td>8</td>
<td>0.37</td>
<td>0.48</td>
<td>0.23</td>
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<tr>
<td>15</td>
<td>0.34</td>
<td>0.47</td>
<td>0.33</td>
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<tr>
<td>19</td>
<td>0.38</td>
<td>0.49</td>
<td>0.36</td>
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The above analysis yielded a KR-20 reliability coefficient of 0.64.
Affective Regulation Scale

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<th>S.D.</th>
<th>Corrected Item-Total Correlation</th>
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</thead>
<tbody>
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<td>5</td>
<td>0.70</td>
<td>0.46</td>
<td>0.28</td>
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<tr>
<td>13</td>
<td>0.66</td>
<td>0.47</td>
<td>0.43</td>
</tr>
<tr>
<td>14</td>
<td>0.31</td>
<td>0.46</td>
<td>0.36</td>
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<tr>
<td>16</td>
<td>0.40</td>
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<td>18</td>
<td>0.41</td>
<td>0.49</td>
<td>0.43</td>
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The above analysis yielded a KR-20 reliability coefficient of 0.61.
Event Coping Scale:
Rotated Factor Matrix After Principal Components Analysis
With Varimax Rotation Extracting 3 factors (N = 935)

<table>
<thead>
<tr>
<th>Item Number</th>
<th>Factor 1 (Direct)</th>
<th>Factor 2 (Avoidance)</th>
<th>Factor 3 (Affect. Reg.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.41873*</td>
<td>-0.19783</td>
<td>0.42288*</td>
</tr>
<tr>
<td>2</td>
<td>-0.04931</td>
<td>0.63124*</td>
<td>-0.06973</td>
</tr>
<tr>
<td>3</td>
<td>-0.18808</td>
<td>0.57242*</td>
<td>-0.02927</td>
</tr>
<tr>
<td>4</td>
<td>0.21527</td>
<td>0.47964*</td>
<td>-0.07211</td>
</tr>
<tr>
<td>5</td>
<td>-0.04500</td>
<td>0.24202</td>
<td>0.46401*</td>
</tr>
<tr>
<td>6</td>
<td>-0.05498</td>
<td>0.60434*</td>
<td>0.18202</td>
</tr>
<tr>
<td>7</td>
<td>0.16993</td>
<td>0.52509*</td>
<td>0.02282</td>
</tr>
<tr>
<td>8</td>
<td>0.41402*</td>
<td>0.33071</td>
<td>0.11045</td>
</tr>
<tr>
<td>9</td>
<td>0.69125*</td>
<td>0.05135</td>
<td>-0.09391</td>
</tr>
<tr>
<td>10</td>
<td>0.72798*</td>
<td>-0.05828</td>
<td>0.03039</td>
</tr>
<tr>
<td>11</td>
<td>0.55935*</td>
<td>0.27092</td>
<td>0.15543</td>
</tr>
<tr>
<td>12</td>
<td>0.58618*</td>
<td>-0.16943</td>
<td>0.21475</td>
</tr>
<tr>
<td>13</td>
<td>0.08891</td>
<td>0.08056</td>
<td>0.67343*</td>
</tr>
<tr>
<td>14</td>
<td>0.20940</td>
<td>-0.09931</td>
<td>0.61526*</td>
</tr>
<tr>
<td>15</td>
<td>0.12911</td>
<td>0.44117*</td>
<td>0.20841</td>
</tr>
<tr>
<td>16</td>
<td>-0.00785</td>
<td>0.22433</td>
<td>0.52466*</td>
</tr>
<tr>
<td>17</td>
<td>0.63985*</td>
<td>0.13248</td>
<td>0.21094</td>
</tr>
<tr>
<td>18</td>
<td>0.21265</td>
<td>-0.01878</td>
<td>0.63361*</td>
</tr>
<tr>
<td>19</td>
<td>-0.15601</td>
<td>0.53271*</td>
<td>0.27114</td>
</tr>
</tbody>
</table>

Variance | 17.9% | 12.9% | 7.8%

* Indicates a factor loading of 0.3 or greater which was accepted as significant (Comrey, 1973; Gorsuch, 1983).
Inter-correlations of Event Coping Scales (N = 114)

<table>
<thead>
<tr>
<th></th>
<th>Direct</th>
<th>Avoidance</th>
<th>Affect. Reg.</th>
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</thead>
<tbody>
<tr>
<td>Direct</td>
<td>1.00</td>
<td>0.34</td>
<td>0.43</td>
</tr>
<tr>
<td>Avoidance</td>
<td></td>
<td>1.00</td>
<td>0.48</td>
</tr>
<tr>
<td>Affect. Reg.</td>
<td></td>
<td></td>
<td>1.00</td>
</tr>
</tbody>
</table>

All correlations were significant on a 2-tailed test (p < 0.001).
**APPENDIX I**

RESULTS OF ANALYSES OF VARIANCE BETWEEN TENSION, TENSION-VASCULAR AND MIGRAINE SUBJECTS ON VARIABLES STUDIED IN CHAPTER 7

<table>
<thead>
<tr>
<th>Variable</th>
<th>Tension</th>
<th>Tension-Vascular</th>
<th>Migraine</th>
<th>N</th>
<th>F-value</th>
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<tbody>
<tr>
<td>Self-Control Efficacy</td>
<td>26.91</td>
<td>25.18</td>
<td>26.97</td>
<td>120</td>
<td>1.48</td>
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<tr>
<td></td>
<td>(3.84)</td>
<td>(5.60)</td>
<td>(5.28)</td>
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<tr>
<td>Social Desirability</td>
<td>9.82</td>
<td>10.17</td>
<td>11.09</td>
<td>120</td>
<td>0.81</td>
</tr>
<tr>
<td></td>
<td>(4.89)</td>
<td>(3.95)</td>
<td>(3.61)</td>
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<td></td>
</tr>
<tr>
<td>Headache Frequency</td>
<td>4.22</td>
<td>3.92</td>
<td>3.67</td>
<td>115</td>
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</tr>
<tr>
<td></td>
<td>(1.98)</td>
<td>(1.78)</td>
<td>(1.71)</td>
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<tr>
<td>Headache Intensity</td>
<td>2.12</td>
<td>2.00</td>
<td>2.19</td>
<td>115</td>
<td>1.46</td>
</tr>
<tr>
<td></td>
<td>(0.59)</td>
<td>(0.46)</td>
<td>(0.58)</td>
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<td></td>
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<tr>
<td>Events</td>
<td>9.43</td>
<td>7.72</td>
<td>9.06</td>
<td>114</td>
<td>1.17</td>
</tr>
<tr>
<td></td>
<td>(5.92)</td>
<td>(4.60)</td>
<td>(5.91)</td>
<td></td>
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<tr>
<td>Mattered Ratings</td>
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<td>2.85</td>
<td>3.06</td>
<td>114</td>
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<tr>
<td></td>
<td>(0.39)</td>
<td>(0.55)</td>
<td>(0.54)</td>
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<td>Change Ratings</td>
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<td>0.96</td>
<td>114</td>
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<tr>
<td></td>
<td>(0.42)</td>
<td>(0.65)</td>
<td>(0.56)</td>
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<tr>
<td>Avoidance Coping</td>
<td>2.90</td>
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<td>3.31</td>
<td>114</td>
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<tr>
<td></td>
<td>(1.40)</td>
<td>(1.58)</td>
<td>(1.43)</td>
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<td>Direct Coping</td>
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<td>114</td>
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<tr>
<td></td>
<td>(1.33)</td>
<td>(1.28)</td>
<td>(1.27)</td>
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<tr>
<td>Affective Regulation</td>
<td>2.12</td>
<td>2.43</td>
<td>2.64</td>
<td>114</td>
<td>1.39</td>
</tr>
<tr>
<td></td>
<td>(1.21)</td>
<td>(0.96)</td>
<td>(1.28)</td>
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<td>Upset Ratings</td>
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<td>44.83</td>
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</tr>
<tr>
<td></td>
<td>(14.41)</td>
<td>(15.82)</td>
<td>(15.13)</td>
<td></td>
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</table>

The standard deviations are entered in brackets. None of the obtained F-values reached the 0.05 level of significance.
APPENDIX K

SUMMARY TABLES FOR RESULTS OF ANALYSES OF VARIANCE OF P\textsubscript{1}-N\textsubscript{1} AND N\textsubscript{1}-P\textsubscript{2} SOMATOSENSORY AVERAGE EVOKED POTENTIAL AMPLITUDES

K-1

Matched Pairs of Headache (n = 36) and Control (n = 36) Subjects on P\textsubscript{1}-N\textsubscript{1} Amplitude Over Four Levels of Stimulus Intensity.

<table>
<thead>
<tr>
<th>SOURCE</th>
<th>DF</th>
<th>MS</th>
<th>F-value</th>
<th>p-value</th>
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</thead>
<tbody>
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<td><strong>Between Subjects:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group</td>
<td>1</td>
<td>147.72</td>
<td>7.82</td>
<td>0.008</td>
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<tr>
<td>Pairs</td>
<td>35</td>
<td>24.44</td>
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<td><strong>Within Subjects:</strong></td>
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<td></td>
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</tr>
<tr>
<td>Linear Trend:</td>
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<td></td>
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<tr>
<td>Intensity</td>
<td>1</td>
<td>249.42</td>
<td>96.74</td>
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<td>Group x Int.</td>
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<td>1</td>
<td>2.23</td>
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<td>1.93</td>
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Headache Subjects in Headache and Headache-Free States (Status) on P1-N1 Amplitude Over Four Levels of Stimulus Intensity (N = 16)

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<tr>
<td>Error</td>
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<tr>
<td><strong>Within Subjects:</strong></td>
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<tr>
<td>Linear Trend:</td>
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Pain Sensitive and Pain Insensitive Headache Subjects on P1-N1 Amplitude Over Four Levels of Stimulus Intensity (N = 23)

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<td>Linear Trend:</td>
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<td>1.00</td>
<td>0.330</td>
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<td>1.20</td>
<td>0.46</td>
<td>0.504</td>
</tr>
<tr>
<td>Error</td>
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<td>2.60</td>
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</tr>
<tr>
<td>Cubic Trend:</td>
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PS = Pain Sensitivity
Pain Sensitive and Pain Insensitive Control Subjects on P1-N1 Amplitude Over Four Levels of Stimulus Intensity (N = 28)

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| **Within Subjects:**    |    |      |         |         |
| **Linear Trend:**       |    |      |         |         |
| Intensity               | 1  | 50.47| 14.35   | 0.001   |
| PS x Int.               | 1  | 0.58 | 0.16    | 0.688   |
| Error                   | 26 | 3.52 |         |         |

| **Quad. Trend:**        |    |      |         |         |
| Intensity               | 1  | 0.09 | 0.06    | 0.814   |
| PS x Int.               | 1  | 5.06 | 3.35    | 0.079   |
| Error                   | 26 | 1.51 |         |         |

| **Cubic Trend:**        |    |      |         |         |
| Intensity               | 1  | 1.48 | 0.72    | 0.403   |
| PS x Int.               | 1  | 0.21 | 0.10    | 0.751   |
| Error                   | 26 | 2.05 |         |         |

PS = Pain Sensitivity
Tension Headache (n = 15), Migraine (n = 13) and Control (n = 36) Subjects on P1-N1 Amplitude Over Four Levels of Stimulus Intensity

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**Pairwise Comparisons Between Groups on Linear and Quadratic Trend for P1-N1 Amplitudes**

Migraine ($n = 13$) and Tension ($n = 15$) Headache Subjects

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Tension Headache ($n = 15$) and Control ($n = 36$) Subjects

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### Migraine (n = 13) and Control (n = 36) Subjects

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K-5

Tension Headache (n = 15), Migraine (n = 13) and Control (n = 36)
Subjects on N1-P2 Amplitude Over Four Levels of Stimulus Intensity

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| **Within Subjects:** |    |       |         |         |
| Linear Trend:        |    |       |         |         |
| Intensity            | 1  | 625.90| 48.21   | 0.000   |
| Group x Int.         | 1  | 10.01 | 0.77    | 0.467   |
| Error                | 61 | 12.98 |         |         |

| Quad. Trend:         |    |       |         |         |
| Intensity            | 1  | 3.31  | 0.86    | 0.358   |
| Group x Int.         | 1  | 9.11  | 2.36    | 0.103   |
| Error                | 61 | 3.85  |         |         |

| Cubic Trend:         |    |       |         |         |
| Intensity            | 1  | 0.18  | 0.09    | 0.767   |
| Group x Int.         | 1  | 0.56  | 0.28    | 0.761   |
| Error                | 61 | 2.02  |         |         |
Matched Pairs of Headache (n = 36) and Control (n = 36)
Subjects on N1-P2 Amplitude Over Four Levels of Stimulus Intensity

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### Headache Subjects in Headache and Headache-Free States (Status) on N1-P2 Amplitude Over Four Levels of Stimulus Intensity (N = 16)

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Pain Sensitive and Pain Insensitive Headache
Subjects on N$_1$-$P_2$ Amplitude Over Four Levels
of Stimulus Intensity (N = 23)

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PS = Pain Sensitivity
Pain Sensitive and Pain Insensitive Control
Subjects on $N_1$-$P_2$ Amplitude Over Four Levels
of Stimulus Intensity ($N = 28$)

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<tr>
<td>PS x Int.</td>
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<td>0.27</td>
<td>0.17</td>
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<tr>
<td>Intensity</td>
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<td>0.09</td>
<td>0.04</td>
<td>0.843</td>
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<tr>
<td>PS x Int.</td>
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PS = Pain Sensitivity
APPENDIX L

SUMMARY TABLES FOR RESULTS OF ANALYSES OF VARIANCE FOR DIFFERENCES BETWEEN TENSION (n = 15), MIGRAINE (n = 13) AND CONTROL (n = 36) SUBJECTS (GROUP) ON PAIN MEASURES

### Electrical Pain Threshold

<table>
<thead>
<tr>
<th>SOURCE</th>
<th>DF</th>
<th>MS</th>
<th>F-value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between Subjects:</td>
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<td></td>
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<td></td>
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<tr>
<td>Group</td>
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<td>188.12</td>
<td>4.11</td>
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### Electrical Pain Tolerance

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<th>p-value</th>
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<tbody>
<tr>
<td>Between Subjects:</td>
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<tr>
<td>Group</td>
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### Temporal Ice Pain Threshold

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<th>p-value</th>
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<td>Group</td>
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