Minimal-Contact Cognitive-Behavioural Treatment of Chronic Daily Headache: The Role of Cognition in the Efficacy and Mechanisms of Treatment

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by

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Abstract

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Objectives. To determine the efficacy of minimal-contact cognitive-behavioural therapy (MC-CBT) and conventional therapy-intensive cognitive-behavioural therapy (I-CBT) in the treatment of chronic daily headache (CDH) and to examine the cognitive mechanisms through which treatment might work.

Design and Methods. Study 1 (N =37) employed a split-plot design to compare pre to post effects in three treatment groups (I-CBT, MC-CBT, and waiting list controls). This Study also used a correlational approach to investigate a hypothesised association between cognitive changes and outcome changes. Study 2 (N = 20) also employed a split plot design to compare MC-CBT with an almost identical minimal-contact treatment in which the explicit cognitive training component was replaced by an unstructured positive-coping-skills block (MC-PCS). Study 3 (N = 6) employed single-case methodology to investigate daily fluctuations in self-efficacy and how these relate to the application of cognitive and non-cognitive treatment strategies.

Results. *Study 1.* MC-CBT and I-CBT were significantly and equally effective in terms of positive outcome and adaptive cognitive change (chiefly, less catastrophising, and increased perceived self-efficacy). Moderate correlations were obtained between cognitive changes and outcome changes but a substantial part of the variance in outcome was not explained by the cognitive changes considered here. *Study 2.* MC-CBT was found to be significantly more effective, than MC-PCS, in maintaining treatment gains at 6-month follow-up. *Study 3.* A strong inverse association was found between daily change in perceived self-efficacy and daily ratings of headache activity. However, the learning of explicit cognitive strategies appeared not to make an immediate impact.

Conclusions. In contrast to previous negative findings with this supposedly refractive headache population, minimal-contact CBT is a cost-effective treatment option for CDH sufferers. Cognitive variables (particularly appraisal style and perceived self-efficacy) appear to be at the heart of the treatment mechanism, as assumed in CBT models, but the important question of causality remains unanswered. However, explicit cognitive training seems to be essential with regard to maintenance of treatment gains. The theoretical and clinical implications of these findings are discussed, and a revised CBT model for chronic headaches is proposed.

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Chapter 1

General Introduction

1.1 Overview

This thesis is the culmination of a four-year research programme that examined conventional therapy-intensive and minimal-contact formats of cognitive-behavioural treatment (CBT) for chronic daily headache in adults. The role of cognitive factors in headache coping and as possible mechanisms of treatment change was of particular interest. The whole programme consisted of an evolution of three consecutive studies.

1.2 Rationale

The rationale for this research was threefold. First, it was of interest to study a sub-population of headache sufferers who experienced daily or near daily headaches and who presented frequently at the headache clinic in which the researcher worked. This group of chronic daily headache (CDH) sufferers is interesting for a number of reasons: (a) they have tended to be poor responders to psychological and medical treatments but are common at headache clinics; (b) CDH does not fit in with established headache nosology and challenges the validity of the migraine-tension headache split that pervades the literature; and (c) there is very little psychological outcome research with this group, and certainly none that has investigated the efficacy of CBT.

Secondly, motivation to enhance clinical practice has encouraged the evaluation of more resource-efficient modes of treatment. One promising area is the development of minimal-contact CBT (MC-CBT). However, further clinical research is needed to evaluate the efficacy of MC-CBT compared to conventional format therapy-intensive CBT (I-CBT).

Thirdly, while much outcome research has supported the efficacy of psychological treatment of chronic benign headaches (Blanchard, 1992) there is less understanding of the role of specific components and processes within these multicomponent treatment packages. Therefore, an investigation of the mechanisms through which

CBT is presumed to operate in headache treatment was considered to be of central importance. The role of cognitive factors (e.g. perceived self-efficacy) in coping behaviour and adjustment, and as mediators of treatment change in headache sufferers has attracted much attention but empirical support has tended to come from analogue research. Further empirical research in the clinical arena is required.

These concerns were the primary focus of the research programme. While the investigation of these issues can potentiate a better understanding of the CBT treatment of chronic daily headache, it has wider relevance: to headache and pain treatment generally; and to the understanding of process and outcome links in other applications of CBT.

1.3 Aims

1.3.1 General Aims

The general aims of this research programme were:

a) To enhance the understanding of the aetiology, maintenance and symptomatology of chronic headache and chronic daily headache (CDH) through the use of a more multidimensional model that includes psychosocial as well as physiological factors.

b) To generate a better understanding of how CBT treatments and other psychological treatments operate in their application to headache disorders and, hence, to improve the psychological treatment of chronic daily headache.

c) To illuminate the wider relevance of the above to other applications of CBT, particularly within health psychology.

1.3.2 Specific Aims

The specific aims of this research programme were:

a) To evaluate the efficacy of CBT and a minimal contact-version of this in the treatment of CDH in terms of outcome on the following measures: frequency, duration and intensity of headaches; affect (anxiety and depression); headache behaviour and adjustment (medication consumption, social impact).

b) To examine the role of the explicit cognitive component in CBT in relation to treatment change and outcome.

c) To examine the role of the following cognitive variables as mediators of treatment change in CBT treatment of CDH: perceived self-efficacy; locus of control; appraisal style (coping and catastrophising thoughts).

1.4 Note on Terminology

The headache literature is replete with different terminology for headaches and terminology itself has been the subject of much research. As will be seen in later discussion this largely relates to the complexity of headache, and important theoretical and clinical concerns about definition of headache types. For the purposes of ongoing discussion in the present context, and to aid the review of the literature, the following working definitions are adopted:

Chronic Daily Headache (CDH) is used specifically to refer to any benign headache (migraine, tension-headache or mixed headache) that recurs at a daily or near-daily level (at least 3 days per week) and which has been a problem for the sufferer for at least the past six months.

Chronic Headache is used as the generic term to refer to any benign headache disorder (migraine, tension headache or mixed headache) that has been a problem for the sufferer for at least the past six months. The headaches do not occur as frequently

as in CDH. In the literature, chronic headache has been used interchangeably with terms such as 'chronic recurrent headache', 'chronic benign headache', and 'benign recurring headache'. This position is also adopted here but the simpler term of chronic headache is used wherever this does not distort an author's original meaning.

The term *benign* is used in accordance with medical convention to refer to headache types that are not part of, or secondary to, any known medical disease (e.g. brain tumour).

Chronic is used to refer to a headache disorders that are not transitory and can endure over a long period: medical convention stipulates a period of at least the past six months. Even a cursory review of headache and pain literature shows that there is some confusion in use of the term - 'chronic'. Researchers have used the term to refer to (a) the temporal persistence of the overall headache disorder, and/or (b) the severity of the disorder. Most linguistic authorities agree that this first use of the term (i.e. indicating something that is of long-standing) is correct while the second use (i.e. indicating something that is bad or severe) is, strictly speaking, incorrect (Thompson, 1996). The interchangeable use of chronicity with severity is perhaps based on the assumption that the more enduring a problem is the more severe it must be. This is examined in the context of the literature review.

Chapter 2

The Problem of Chronic Headache

2.1 Introduction and Overview

In this chapter, aspects of the background headache literature are critically reviewed and relevant developments from other areas of psychology are also analysed, with a view to elucidating the gaps in knowledge pertaining to chronic daily headache (CDH). Historically, the vast majority of headache research and debate on headache has been concerned with the two most prevalent forms of chronic headache: migraine, and tension-headache. It is necessary, therefore, to review aspects of this literature base with a view to examining the evolution of ideas that are relevant in the current context.

In order to structure the review, the *problem of chronic headache* can be divided into a number of sections: (1) The nature of the problem; (2) classification of chronic headaches; (3) the emergence of CDH; (4) clinical features of chronic headache; and (5) psychological approaches to chronic headache.

2.2 The Nature of the Problem

2.2.1 Headaches in History

The recognition of headache has a long history. For example, in the Ebers Papyrus, which is acknowledged to be the oldest comprehensive medical text (dating back to Egypt 1500 BC) there is reference to a "sickness of half the head" (Microsoft Corp., Encarta CD-ROM, 1998). This seems to relate to a typical presentation of what we now know as migraine.

The Greek physician, Hippocrates, was amongst the first to identify different headache types when he introduced the notion of headache that was *secondary* to

another disease and headache that was *primary* (i.e. a disorder in itself). This distinction has remained useful to the present day and has been incorporated in modern classification systems although primary headaches are usually referred to as benign headaches, in accordance with medical convention on nosology. Within benign headaches, migraine was the first headache type to have been differentiated, probably because of the intense, more obvious manner in which it was seen to present (Raskin, 1988).

Over the centuries, rudimentary classification of headaches evolved in fits and starts but the differentiation of migraine as a blinding, unilateral headache (first proposed by the Roman-Greek physician, Galen 150 AD) became established. A second major type of headache that was separated out from the wide spectrum of 'not migraine, not secondary' headaches, was recognised by the end of the nineteenth century: this was given various labels such as psychosomatic headache, psychogenic headache, muscle-contraction headache and tension headache (Giammarco, Edmeads, & Dodick, 1998).

2.2.2 The Prevalence of Chronic Headache

Headache is one of the commonest disorders known and it has been described as a "universal plague" (Diamond & Diamond, 1988). Although many studies have looked at prevalence rates for the benign headache types, interpretation has been hampered by a number of problems: (a) Studies have used varying diagnostic criteria (e.g. locality-specific clinical diagnosis versus operationalised criteria set out in headache classification systems); (b) methods of collecting data have varied (e.g. telephone surveys, clinical interview, questionnaires); (c) the time frame of sampling has varied (e.g. some researchers have defined headache caseness based on reported symptoms over the last year whereas others have used a shorter period); (d) as well as coming from different countries (primarily Western), samples have been drawn from a number of different populations (e.g. college students, headache clinic patients and the general population). Despite these problems and so long as interpretation of prevalence data is tempered with some caution, a number of patterns can be discerned.

Prevalence in the general population has usually been measured as the number of people who have had a defined headache in the past year. Research indicates that benign headaches account for the vast majority (around 95%) of headaches and that around 60% to 80% of the adult population will suffer some headaches (Lipton and Stewart, 1993; Waters, 1973; Ziegler, Hassenian, and Couch, 1977). Prevalence of migraine is around 18% for women and 6% for men, whereas 86% of women and 63% of men will experience tension headache within any year (Rasmussen, 1993; Rasmussen, Jensen, Schroll & Olesen, 1991). Therefore, for both migraine and tension-type headache more women than men report headaches, and overall, tension-type headache appears to be far more prevalent than migraine.

Most people are familiar with a headache of some description and would label it as an unpleasant event. More relevant to the current discussion is the observation that for many people, episodes of headache become a long-term, recurring 'problem' that is rated as serious or disabling. Nikiforow and Hokkanen (1978) studied a Finnish population and found that 24.3% of the sample reported suffering mild headaches at least once a week, and 9.9% experienced severe headaches at least once a week

Rasmussen, *et al.* (1991) found that 3% of the general population suffer from chronic tension-type headache. In a telephone based survey of adults aged over twenty years Kryst and Scherl (1994) found that 13.4% of a sample of 647 headache sufferers rated their headache as severe and just over 1% reported daily headaches. Forgays, Rzewnicki, Ober, and Forgays (1993) compared the findings of four studies of headache prevalence in different college populations. The prevalence of suffering at least three headaches per week ranged from 5.5% to 24.6% across the studies; prevalence of daily headache ranged from 1.3% to 3.4%.

2.3 Headache Classification and the Emergence of CDH

2.3.1 History of Classification

There is limited space here to go into all the details of headache classification (see: Marcus, 1992; Rapaport, 1992; Silberstein, 1994). However, as work on CDH has been informed by identified weaknesses in established classification systems and as the vast amount of relevant literature, underpinned by these systems, still pertains to the separation of the two major benign headache types - migraine and tension headache - it is important to briefly examine the historical evolution of headache classification.

Following on from centuries of rudimentary classification the latter part of the twentieth century heralded major advances in the form of purposefully designed classification systems with documented criteria for diagnosing or classifying headache types. The first such system was proposed by the Ad Hoc Committee of the National Institute of Neurological Diseases and Blindness (Ad Hoc Committee, 1962) This was supplanted by a more detailed and modified system of classification proposed by the Headache Classification Committee of the International Headache Society (IHS, 1988).

2.3.2 The Ad Hoc Committee Headache Classification

The Ad Hoc Committee (Ad Hoc, 1962) divided headache into 15 categories of which, the first three - deemed as the main primary or benign headache types - are relevant in the present context. These are: (1) vascular headache of migraine type; (2) muscle-contraction headache; and (3) combined vascular and muscle-contraction headache. The original definitions for these are shown in Table 2.1. These benign headaches were known to form the vast majority of headache disorders. For example, an early study by Lance, Curran, & Anthony (1965) revealed that 95% of patients at a headache clinic could be classified into these three diagnostic groups.

Table 2.1 The Ad Hoc Committee (1962) Classification of Benign Headache

HEADACHE TYPE
Migraine:
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.. Evidence supports cranial arterial dilation/distension in the pain phase but no permanent damage to vasculature (Ad Hoc Committee, 1962, p. 717).
Muscle-Contraction 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 to life stress. The ambiguous and unsatisfactory terms "tension", "psychogenic", and "nervous" headache refer largely to this group (Ad Hoc Committee, 1962, p. 717).
Combined Headache: Vascular and Muscle-Contraction
Combinations of vascular headache of migraine type and muscle-contraction headache, prominently coexisting in an attack (Ad Hoc Committee, 1962, p. 717).

Five subtypes of migraine were identified of which two are relevant in the present context: classic migraine (aura type symptoms before headache); and common migraine (no aura). The other types (cluster headache; hemiplegic or opthalmoplegic migraine; and lower half headache) are rare variants of vascular headache: the interested reader is referred to standard texts such as Raskin (1988).

The Ad Hoc Committee (1962) classification system was known to have weaknesses for a long time in a number of respects. In particular, it was felt to be too brief, descriptive, and vague (Rapoport, 1992). Most writers also agreed that it was based on poor scientific evidence concerning the presumed physiological mechanisms (this is discussed in a later section). The problems with the Ad Hoc (1962) classification led to a revision by the Headache Classification Committee of the International Headache Society (IHS, 1988).

2.3.3 The IHS Headache Classification

Since the Ad Hoc (1962) classification, the mushrooming of research in headache in the intervening period emanating from the growth of a wider, more challenging scientific culture, was perhaps evidenced by the more voluminous document produced

by the IHS (1988): 96 pages, compared to the two-page paper produced by the Ad Hoc Committee. The IHS classification system established itself as the predominant tool in headache research and is currently still in use though its length and detail has proved to be too cumbersome for routine clinical purposes (Marcus, Nash & Turk, 1994).

The IHS classification defines 12 types of headache (a summary of the IHS system is presented in Appendix B) and retains the distinction between migraine and tension headache as the two most common forms of benign headache. It, further, gives a far more detailed and operationalised breakdown of subcategories within these.

Seven categories of migraine are defined of which the two most common are migraine with aura and migraine without aura (corresponding, respectively, to the Ad Hoc (1962) categories of classic migraine and common migraine). Migraine is associated with the following features: duration of headache of 4-72 hours; unilateral onset; prodromal symptoms some of the time; throbbing type head pain; other associated gastric and sensory symptoms; and post-headache feelings. The aura refers to the complex of symptoms (e.g. focused neurological and/or sensory motor disturbances such as flashing lights) that precede, initiate or accompany the onset of a migraine attack and that are usually transitory. Migraine with aura is thought to be far less common, presenting in about 10% of migraine sufferers (Lance & Goadsby, 1998).

The term muscle-contraction headache has been dropped in favour of tension-type headache in view of the mounting evidence that tension headaches do not always relate to changes in the pericranial muscles (Flor & Turk, 1989; Martin, Marie, & Nathan, 1992; Pearce & Morley, 1981). Tension-type headache is therefore divided into two broad types depending on whether contractions of pericranial muscles are involved. A further sub-division is delineated within each of these two categories depending on whether the headache is chronic or episodic. This recognises the empirical evidence pointing to the frequent presentation of patients with chronic headache at headache clinics. A summary of the IHS (1988) classification of migraine and tension-type headache is presented in Table 2.2.

Table 2.2 Summary of IHS (1988) Classification of Headache

Headache Type	
1	**Migraine**
	1.1 Migraine without aura
	1.2 Migraine with aura
2	**Tension-type headache**
	2.1 Episodic tension-type headache
	2.1.1 Episodic tension-type headache associated with disorder of pericranial muscles
	2.1.2 Episodic tension-type headache unassociated with disorder of pericranial muscles
	2.2 Chronic tension-type headache
	2.2.1 Chronic tension-type headache associated with disorder of pericranial muscles
	2.2.2 Chronic tension-type headache unassociated with disorder of pericranial muscles
3-12	**Other headache categories**

Source: Headache Classification Committee of the International Headache Society (IHS, 1988, pp. 13-15)

Although both the Ad Hoc Committee (1962) and the IHS (1988) classification systems allow for the co-existence of migraine and tension headache, these are still seen as essentially separate disorders. There is no category for the more complex syndrome of chronic daily headache, which appears to straddle the established boundaries of migraine and tension-headache without fitting into either category exclusively.

2.4 Chronic Daily Headache (CDH)

2.4.1 Definition of CDH

Chronic daily headache (CDH) has been proposed as an umbrella term for a number of clinically observed headache syndromes that share the common feature that the sufferer has experienced daily or near daily headaches (usually with a mixture of migrainous and tension headache symptoms) for at least the past six months (Solomon

& Cappa, 1987; Saper, 1986, 1990; Sheftell, 1992; Vanast, 1987 a, b, c). A working definition of CDH, suggested by Saper (1986) is "daily or almost daily discomfort with superimposed migrainous events at varying frequencies." (p.19).

Various alternative terms have been used to describe the phenomenon of CDH and these include chronic tension-type headache, transformed migraine, mixed headache syndrome, chronic headache complex, tension-vascular headache, high medication headache and chronic refractory headache. This variability in terminology is perhaps a sign of the relative newness of the phenomenon as a subject of scientific inquiry. The different terms also reflect the heterogeneous nature of CDH and the variety of purported mechanisms that have been proposed to explain this.

Researchers have proposed a number of hypotheses to explain the development of CDH. One view is that CDH largely evolves from the 'transformation' of a previous episodic migraine disorder into a chronic mixed headache syndrome (known as *transformed migraine*) as the headaches become ever more frequent (Mathew, 1993; Mathew, Reuveni, & Perez, 1987; Mathew, Stubits, and Nigam, 1982; Saper, 1986).

Mathew *et al.* (1982) first introduced the idea that CDH might evolve from a history of episodic migraine after observing that many CDH sufferers also reported a history of episodic migraine. In a later study of 630 patients with CDH, Mathew *et al.* (1987) found that 78% could be classified as having transformed migraine. Saper (1986) studied 615 patients with CDH and found that all had started with intermittent migraine; by age forty-five 90% of the group developed CDH. According to Mathew (1993), CDH patients classified as transformed migraine

> ...usually start with episodic migraine in their teens and early 20's. As time passes, the migraine attacks become more frequent, and the patients gradually develop low-grade interictal headaches. By the age of 35 or 40 years, they reach a stage of daily or near-daily headache with mixed features of migraine and tension-type headache. (p. 26)

Another explanation is that some sufferers develop high frequency rebound-headache syndrome that is linked to a frequent intake of analgesics or other symptomatic

medications such as ergotamine (Kudrow, 1982; Rapoport, 1987; Von Korff, Galer, & Stang, 1995). Whilst many studies have found that a high proportion of CDH sufferers use frequent medication, the relationship between medication overuse and headache is still largely unclear, and the precise mechanisms are yet to be identified. The existing evidence is based largely on correlational type studies and retrospective surveys and these provide only weak support for the link. Furthermore, Rapoport, Weeks, and Sheftell (1986) observed that many headache patients and most non-headache patients who use high amounts of analgesics do not go on to suffer CDH. This led them to conclude that only a proportion of headache patients may be susceptible to a rebound syndrome - suggesting that other unknown variables are important.

Mathew *et al.* (1987) were the first researchers to attempt to quantify the different types of CDH. In a sample of 630 CDH patients they found that 78% could be classified as having transformed migraine, 13% had tension-type headaches without migrainous features, and 9% had headaches with a mixture of migraine and tension features but with no previous history of episodic headaches (i.e. CDH was 'new' headache disorder in a previously headache-free individual). Seventy-three percent of the sample was assessed as being medication overusers. This study appeared to confirm many clinical observations and tentative findings from other studies of CDH. Thus, the momentum to understand and classify CDH gained strength.

2.4.2 Proposed Classification of CDH

Although there is a category for chronic tension-type headache (CTTH) in the IHS (1988) classification (tension-type headache for at least 15 days per month and 180 days per year) this does not cater for the typical CDH syndrome which includes a history of migraine and/or the current co-existence of migrainous and tension type symptoms in the sufferer. Messinger, Spierings, and Vincent (1991), for example, used the IHS criteria to classify 410 people with a headache history of more than two years. Only 9.1% could be strictly classified as having chronic tension-type headache (CTTH) and nearly 40% could not be unequivocally classified into any category. Furthermore, 86% of the CTTH group suffered from two or more migrainous features.

With the problems of the IHS (1988) system in mind and taking into account the known clinical features of CDH (including high medication use in some sufferers), Silberstein, Lipton, Solomon, and Mathew (1994) suggested a revision to headache classification to accommodate CDH. Three main types of CDH were proposed as shown in Table 2.3 (a fourth, very rare type of CDH known as hemicrania continua was also proposed).

Table 2.3 Proposed Headache Classification for Chronic Daily Headache (CDH)

Chronic Daily Headache:
(daily or near daily headache lasting >4 hours/day for >15 days/month)

Transformed Migraine (TM) with medication overuse without medication overuse	previous history of episodic migraine; as headaches become more frequent, intensity of migraine symptoms diminish (e.g. nausea); features of both migraine and tension headache occur.
Chronic Tension-Type Headache (CTTH) with medication overuse without medication overuse	previous history of episodic tension-type headache; headaches often diffuse and bilateral; absence of prior migraine history; migrainous symptoms are generally absent but may occur.
New Daily Persistent Headache (NDPH) with medication overuse without medication overuse	no prior history of episodic migraine or tension-type headache; abrupt onset over less than three days; aetiology likely to be heterogeneous.

Adapted from Silberstein *et al.* (1994, p. 3)

The nosology for CDH proposed by Silberstein *et al.* (1994) is beginning to gain wide acceptance amongst researchers. It has been based on a body of accumulating clinical observations together with some empirical research that has started to grow in recent years. However, until further empirical support emerges, the proposed nosology remains a useful working framework rather than an established classification system.

2.4.3 Prevalence of CDH

To date, the only study that has looked at the prevalence of CDH in the general population, as based on Silberstein *et al.*'s (1994) nosology, has been by Scher, Walter, Stewart, Liberman, and Lipton (1998). Using the operational definition of CDH as 'at least 180 days in the past year with any headache', they found an overall prevalence rate of 4.1% in a sample of 13, 343 people in Maryland, USA surveyed via telephone interview. Those with CDH were categorised as having chronic tension-type headache (53%), transformed migraine (31%), or NDPH (16%). A criticism of the study is its over-reliance on respondents' retrospective self reports and the possibility that a telephone interview precluded a more comprehensive clinical assessment or probing. Nevertheless, Scher *et al.* provided an initial empirically supported prevalence figure for CDH that was not too divergent from previous estimates of around 2%. (Saper, 1990).

A more crucial observation is that CDH is a very common complaint at headache clinics and possibly the most common complaint (Mathew, 1993; Saper, 1990; Sheftell, 1992). This is understandable if one assumes that CDH by its very nature is more debilitating and, therefore, most sufferers are likely not only to seek specialist medical help but also to keep returning for treatment. At the same time non-CDH sufferers either do not feel the need to seek specialist help or are adequately treated elsewhere in the health system. Hospital based studies have found a prevalence of CDH of 25% to 75% (Mathew, 1993; Mathew, *et al.*, 1987; Rothrock, Patel, Lyden, and Jackson, 1996; Saper, 1990; Srikiatkhatchorn and Phanthumichinda, 1997).

2.5 Clinical Features of Chronic Headache

2.5.1 The Continuum of Chronic Headache

Psychological approaches to the understanding and treatment of headaches have largely been based on the medically determined classification that has differentiated the 'vascular' features of migraine from the 'musculo-skeletal' features of tension headache. The assumption was that the associated set of symptoms were mutually exclusive (Marcus, 1992). The presumed vascular symptoms included neurological

aura, unilateral location of pain, pulsating or throbbing pain, nausea, vomiting and increased sensitivity to sensations; the defining features of tension headache were thought to be the presentation of dull, band-like and bilateral head pain and the absence of vascular features.

The presumed differences in headache symptoms between migraine and tension headache have been challenged by a number of studies that have concluded that there is actually considerable overlap in how symptoms present and evolve in the two primary headache types and it is not easy to identify or separate out a set of migraine features from a set of tension features. This has led to an increasingly prevalent view amongst researchers that migraine and tension headache are not distinct headache types but may be part of an overall chronic headache continuum or spectrum along which the balance of migraine-type and tension-type symptoms can vary (Waters, 1973; Featherstone, 1985; Raskin, 1988; Bakal, 1982; Marcus, 1992; Schade, 1997). One representation of this headache continuum is shown in Figure 2.1.

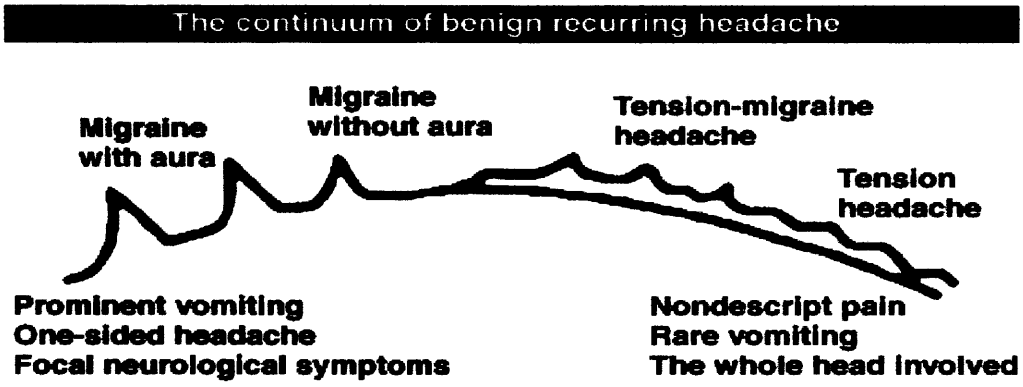


Figure 2.1 The Continuum of Chronic Headache (Source: American Council for Headache Education website: http://www.achenet.org/whatcause.htm)

A number of lines of evidence tend to support the concept of a headache continuum. Firstly, the presentation of both migrainous and tension type features in CDH, and the evolution of CDH from episodic migraine suggests a common underlying headache disorder. Secondly, there is now increasing evidence of a common neurogenic mechanism in headache pathophysiology and shared physiological processes in

migraine and tension-headache (Lance, 1993; Lance & Goadsby, 1998). Thirdly, a body of studies that have examined the symptoms of both migraine and tension-headache have found considerable commonality between these. Two lines of research evidence have emerged from this: (a) questionnaire based studies on non-diagnosed participants have consistently failed to identify the symptom clusters predicted by the traditional view of headache differentiation (Martin, Milech & Nathan, 1993; Peck & Attfield, 1981; Waters, 1973; Ziegler, Hassanein & Hassanein, 1972); (b) studies of reported symptoms across previously diagnosed groups have shown that purportedly migraine-specific or tension-specific symptoms are experienced by substantial numbers of people in both headache groups (Bakal & Kaganov, 1977; Kaganov, Bakal, & Dunn, 1981; Schade, 1997).

In view of the above evidence, many have argued that there has been unnecessary preoccupation with attempting to identify psychological symptom profiles that were specific to different headache types. Bakal (1982) has been one of the more ardent proponents of an alternative view that rejected the notion of distinct headache categories. He argued that, from a psychological viewpoint, it did not make sense to separate the headache types as the accompanying psychological symptoms and effects were largely similar across them, and it was other variables such as cognitive appraisal and headache frequency that determined symptomatology. This view is generally well supported by the empirical evidence reviewed above and it was also the position taken by the researcher in the context of the research programme reported here.

2.5.2 General Phenomenology and Impact of Chronic Headache

Apart from its common occurrence, and not incompatible with a continuum model, a prominent aspect of chronic headache is the menacingly diverse manner in which it seems to emerge and manifest itself, both within and across individuals. Headaches can vary in terms of several physical and psychosocial parameters. Physically, variability on measures such as frequency, duration, severity, and location of pain can be discerned. In addition, headaches are known to present with many different constellations of physical and psychological symptoms. Studies have consistently found associations between chronic headache and avoidance of activities, pain

behaviours, increased medication consumption, and poor affect (Merikangas, 1994; Nicholson, Blanchard, and Appelbaum, 1990; Philips and Jahanshahi, 1985; Waters, 1973). Chronic headache impacts not only on the individual sufferers through interfering with their health and quality of life, particularly in the areas of physical functioning and social functioning (Cavallini, Micieli, Bussone, Rossi, and Nappi, 1995; Kryst and Scherl, 1994; Solomon, Skobieranda, & Gragg, 1993), but also on society through such things as economic costs of lost working days and exhaustion of health-care resources (Lipton, Stewart, and Korff, 1997; Stang and Osterhaus, 1993).

2.5.3 The Role of Stress

Many researchers accept that stress factors play a role in the aetiology and/or exacerbation of chronic headache. However, there is considerable debate in the literature as to the definition of stress and the nature of the stress-headache relationship. There is not space here to do justice to the large body of literature on stress and headaches but an outline of the essential findings and ideas will be presented.

The majority of research in this area has assumed stress to be a discrete unpleasant event or series of events. A popular hypothesis based on this assumption has been that there is some sort of direct linear relationship between the occurrence and quantity of stress and the severity of headache. This line of reasoning has been at the heart of research that has investigated whether headaches can be triggered and/or made worse by stress.

Evidence for a possible link between stress and headaches has come from a number of areas: (a) *Retrospective studies.* Findings from retrospective studies have consistently indicated that stress or mental tension is the most commonly reported trigger factor for headaches, being reported by approximately half of headache sufferers (Drummond, 1985; Rasmussen, 1993). The conclusions need to be tempered in view of the possibility of memory distortions that are inherent in retrospective designs; (b) *Prospective studies.* A number of prospective studies have found significant correlations between headache activity, and stress occurring at the same time or in the days leading up to the headache (Henry-Gutt and Rees, 1973; Kohler & Haimerl,

1990; Robbins, 1994; Sorbi & Tellegen, 1988). These studies, whilst addressing the flaws in retrospective studies do not completely eradicate expectancy effects and demand characteristics; (c) *Laboratory based studies*. These studies have had the advantage of experimentally manipulating stress and testing the impact either directly, in terms of self-reported headache, or indirectly, on physiological processes presumed to signify headache such as scalp muscle tension (Gannon, Haynes, Cuevas, Chavez, 1987; Philips & Hunter, 1982a). However, ecological validity of laboratory induced stressors is suspect, and physiological indices of change (e.g. such as scalp muscle tension) may have a much weaker relationship to either stress or headaches than previously assumed.

A major flaw in the above body of research is that it fails to explain the observed variability in the impact of stress within and across headache sufferers. Furthermore, many patients with chronic headache report that that their headaches appear to start or continue in the absence of identifiable stress. This has led to two related theoretical explanations that have helped to further the understanding of the relationship between chronic headache and stress.

First, following on from the work of Lazaras and Folkman (1984), stress has been defined, not as a feeling or negative event, but as a dynamic process that is based on the transaction between the person and their environment, mediated by ongoing cognitive appraisal of situational threats and coping resources. Researchers have shown that ongoing cognitive appraisal of events or situations (e.g. personal meaning, how negative the event is) and coping resources (e.g. perceived social support) is a potent mediator in the stress-headache relationship (De Benedittis, Lorenzetti, & Pieri, 1990; Ehde & Holm, 1992; Marlowe, 1998a).

Second, based on a number of empirical studies that investigated the cognitions of chronic headache patients, Bakal and colleagues have put forward the hypothesis that as an individual's headaches become more severe and chronic, then life events or situational stresses become less important (and may not even be necessary to fuel the headaches), as it is the stress of having the headache disorder itself that becomes the insidious driving force (Bakal, 1982; Demjen & Bakal, 1986; Demjen, Bakal, & Dunn, 1990). Therefore, over time, there is a *cognitive shift* "whereby the patient's

primary concern moves from situational and interpersonal stress to distress associated with the headache itself." (Demjen & Bakal, 1986, p. 187).

2.5.4 Emotional Factors

The co-existence between chronic headache or pain and emotional suffering has been recognised for a long time. Initially, emotional factors were presumed to be part of an overall personality dysfunction. Based on clinical observations, Wolff (1937), described migraine sufferers as perfectionist, driven by goals, obsessive, and having lots of repressed anger. Tension headache sufferers were thought to be generally anxious, dependent, angry, and depressed. Empirical validation was not undertaken, nor was there a readiness to discuss possible alternative hypotheses. For example, the patients who presented to clinics may have shared particular personality characteristics that were not truly representative of people with similar headaches who did not seek help, and their psychological presentation could have been largely a consequence of their chronic headache rather than a predisposing factor.

Later work on personality and psychopathology in chronic headache and pain sufferers was based on a more empirically grounded approach using operationalised criteria for psychopathology, through instruments such as the Minnesota Multiphasic Personality Inventory (MMPI) (Hathaway & McKinley, cited in Merikangas, 1994) and the Eysenck Personality Questionnaire (EPQ) (Eysenck & Eysenck, 1975). This genre of research failed to support the idea of global personality or psychopathological dysfunction in headache sufferers. Instead, there was some support that, for a subset of headache patients, scores are elevated on the Hypochondriasis, Depression, and Hysteria subscales of the MMPI and the Neuroticism scale of the EPQ (Blanchard, Andrasik, & Arena, 1984).

The rise of behavioural and CBT approaches heralded a new approach to headache psychopathology and emotion. This approach has been characterised by two important changes in emphasis. Firstly, researchers have become more interested in tracking the specific types of psychological symptoms that certain headache sufferers may be prone to rather than being preoccupied by overall personality traits in all headache sufferers. Secondly, there is now general acceptance that psychological

symptoms can also be a consequence of having chronic headache (Bakal, 1982, Martin, 1993).

While the debate about cause and effect rages on, most researchers and clinicians agree that chronic headache as a syndrome and specific headache attacks are commonly accompanied by various psychological symptoms. Anxiety and depression have been the focus of most research but other clinical symptoms such as anger have also been studied (Adler, Adler, & Packard, 1987; Merikangas, 1994; Philips & Hunter, 1982b; Tschannen, Duckro, Margolis, & Tomazic, 1992).

Studies have usually employed psychiatric inventories to measure the emotional component and it is possible that the overlap of some items (e.g. fatigue) with headache symptoms may have given a falsely inflated psychiatric score. Nevertheless, the general conclusion from this body of research is that anxiety and depression symptoms tend to be elevated in a chronic headache population compared to non-clinical groups but not as elevated as those for clinical psychiatric populations. Overall, bearing in mind the psychometric variability of scales used across studies, approximately 50% of chronic headache patients show clinical levels of anxiety and around 30% show clinical levels of depression (Puca, Genco, Savarese, Prudenzano, D'Ursi, *et al.*, 1992). In addition, an interaction between emotions such as anxiety and depression and, avoidance or inactivity, has also been well supported in the mental health and pain literature (Beck, Rush, Shaw, & Emery, 1979; McCraken, Faber, & Janeck, 1998; Philips & Jahanshahi, 1985).

2.5.5 Specific Clinical Features of CDH

The mounting empirical research on CDH suggests that notwithstanding the obvious differences pertaining to frequency of headaches and possibly more adverse impact on psychosocial functioning (Cavallini *et al.*, 1995), the clinical features of CDH are not substantially different to those of chronic headache generally. However, it seems important to consider what little research has been done on the specific clinical features of CDH.

Solomon, Lipton, & Newman (1992) studied, through a semi-structured clinical interview, 100 consecutive patients with features of CDH (defined as headache for at least 6 days per week for at least 6 months). Fifty percent of the sample experienced a steady ache while 29% described throbbing-type pain. However, many patients reported more than one site of pain and quality of pain was also quite variable within and across patients. Only 13% of patients felt that their headaches were fairly unproblematic. The most common associated features were photophobia (42%) and phonophobia (37%). Psychological symptoms were only assessed through informal questioning but anxiety, depression, and irritability were common. Twenty percent reported stress as the main aggravating factor. Daily analgesic use was a feature in 47% of patients. The researchers concluded that CDH sufferers comprise a clinically heterogeneous group in terms of headache severity, pain sensations, and associated symptoms. Similar findings were reported by Srikiatkhatchorn & Phanthumichinda (1997) who investigated the prevalence and clinical features of daily headache in a headache clinic in Thailand.

Blanchard, Appelbaum, Radnitz, Jaccard, & Dentinger (1989) were interested in whether CDH sufferers are more poorly adjusted psychologically than sufferers with less frequent headaches. No significant differences were found on a battery of psychometric instruments (e.g. Beck Depression Inventory, Spielberger State-trait Anxiety Inventory, Psychosomatic Symptoms Checklist) between a CDH group and two groups of patients with less frequent headaches. A criticism of Blanchard et al's study is that they relied on a strict definition of CDH, i.e. headaches that occurred everyday. In fact, the two comparison groups also suffered frequent headaches (one group consisted of patients who experienced only 1-2 headache-free days per week and the other group had patients with 3-5 headache-free days per week). Therefore, most patients suffered daily or near-daily headaches and the finding of no group differences concerning psychological symptoms was, perhaps, to be expected.

2.6 Overview of Psychological Approaches to Chronic Headache

2.6.1 Historical Trends

Several advances have been made in the course of this century regarding the psychological understanding of headaches and pain. Three major trends, approximately chronological, can be discerned: (1) Psychoanalytic and personality approaches which dominated until the 1960s; (2) Biofeedback approaches which had their heyday from the 1960s to the 1980s; (3) Cognitive and cognitive-behavioural approaches which have been in ascendance from the late 1970s onwards. In addition, behavioural and relaxation approaches have been important throughout - initially co-existing with biofeedback but later tending to be incorporated into cognitive-behavioural treatments. Each of these approaches except CBT will be discussed below. CBT approaches are discussed in more detail in the next chapter.

2.6.2 Personality and Headache

Personality as a predisposing factor in chronic pain and headache sufferers has appealed to researchers and clinicians for most of this century. Early approaches derived from creative application of psychoanalytic theory and selective clinical observations rather than rigorous empirical tests. Later, with the growth of psychosomatic medicine and development of instruments such as the MMPI, much research effort was invested in looking for a 'headache-prone' personality (Alexander 1950). However, empirical evidence, based on the MMPI, has rejected the notion of a deviant headache personality. Because accurate measurement of this complex and elusive variable, and specification of its links in headache aetiology, has been fraught with so many difficulties, most researchers have now abandoned it as a useful avenue to understand headaches (Harrison, 1975; Kohler & Kosanic, 1992; Martin, 1993; Philips, 1976).

2.6.3 Biofeedback

Biofeedback approaches were applied either as separate focused treatments or, more usually, together within a treatment package. Biofeedback training became popular in

many applications of psychology in the 1970s (Yates, 1980). The procedure essentially involved the electronic monitoring and feedback of various physiological processes (e.g. scalp muscle activity) that were ordinarily difficult to perceive through senses alone. Because the feedback was transformed into a more intelligible signal (e.g. a needle on a display or audible tones) the patient could learn to control the desired physiological processes through monitoring the feedback signal.

Three different versions of biofeedback training were developed for headaches, based on purported physiological processes thought to underlie different headache types: (1) Thermal biofeedback (TBF) i.e. feedback of skin temperature of the hand or finger. This is based on the rationale that peripheral warming leads to a cooling or constriction of vascular arteries of the head - a physiological change that is thought to abort the headache in migraine which itself is presumed to result from dilation of vascular cranial arteries; (2) Cephalic vasomotor biofeedback or blood volume pulse biofeedback (BVP). This consists of feedback of blood flow through cranial blood vessels (usually the temporal or extra-carotid arteries). The rationale is again based on the assumption that a disturbance of the vascular system is involved in migraine; (3) Electromyographic biofeedback (EMG) of muscle tension levels of the neck and scalp. This has been primarily used for tension-headaches - the rationale based on the assumption that the pain of tension-headaches is caused by significant neck/scalp muscle tension.

While outcome studies tended to support the efficacy of biofeedback procedures it was consistently shown that behavioural relaxation training could achieve similar treatment gains without the expenditure and clinical inconvenience presented by the use of biofeedback gadgetry (Blanchard & Andrasik, 1987). This led to the decline of biofeedback in the treatment of headaches, and the rise of behavioural treatment packages that incorporated relaxation training and management of pain behaviour based on learning theory (Fordyce, 1976).

2.6.4 Relaxation Training

Relaxation approaches to headache treatment have taken two main forms which have often complemented each other in treatment packages: (1) Progressive muscular

relaxation training (PMRT) based on the work of Jacobson (1938), and later shortened and adapted by Bernstein & Borkovec (1973). In PMRT, patients are taught to relax various muscle groups by practising repeated cycles of tension-relaxation. All muscle groups are typically covered in a 20-40 minute session. A number of sessions together with regular home-practice with audiocassette are usually offered; (2) Autogenic Training (AT) based on the work of Schultz & Luthe (1959, 1969). In AT, patients are taught to control distressing physiological processes through the use of autogenic phrases or words that are presumed to represent the opposite of these processes. For example, feelings of 'heaviness' and 'warmth' are thought to signify the relaxing of muscle tension and the lessening of arterial constriction (i.e. dilation) respectively.

Both biofeedback and relaxation treatments also made certain assumptions (often implicit) about the aetiology of headaches and the mechanism of treatment action. These assumptions have not always stood up well to empirical testing. As many of these procedures were also incorporated in the third and ascendant treatment approach to headache - cognitive-behavioural therapy (CBT) - a critical evaluation is presented in the context of reviewing CBT approaches to chronic headache. This is presented in the next chapter.

2.6.5 Behavioural Approaches

Most psychological treatment packages for headache incorporate various behavioural strategies such as activity planning and management of medication intake. The rationale for this came, initially, from the work of researchers in the 1970s who emphasised the role of conditioning, operant reinforcement and social modelling in the long-term maintenance of observable 'pain behaviours' such as avoidance of activity, verbal/non-verbal complaints, and habitual medication intake (Fordyce, 1976; Philips & Hunter, 1981). For example, a chronic pain sufferer might learn that his pain rewards him with new found sympathy from his partner (i.e. secondary gains) or that his pain disappears for a few hours each time he takes an analgesic, or that he can stop the pain from becoming worse by avoiding walking. It was held that, through these processes of learning, certain pain behaviours would, over time, become (a) disproportionate to the pain disorder, and (b) self-maintaining, irrespective of the

severity of the pain. Therefore, the behavioural reaction to the pain, could itself become a central component of the chronic pain disorder.

Lethem, Slade, Troup, and Bentley (1983) extended this behavioural framework by adding a cognitive dimension in their fear-avoidance model of chronic pain. It was postulated that the anticipated fear of the pain becoming worse in any situation led to two opposing reactions: avoidance or confrontation. Persistent avoidance was seen as maladaptive and served to perpetuate chronic pain through rendering the sufferer ever more fearful of engaging in activity. Confrontation was seen as an adaptive strategy that promoted activity and helped to desensitise the individual to their fear of pain

A number of researchers have attempted to test, empirically, the relationship between chronic pain and behavioural variables through various pain behaviour questionnaires, experimental manipulation or treatment manipulation (Appelbaum, Radnitz, Blanchard, & Prins, 1988; Hursey & Jacks, 1992; Philips & Hunter, 1981; Philips & Jahanshahi, 1985, 1986; Radnitz, Appelbaum, Blanchard, Elliot, & Andrasik, 1988). The general findings from these studies tend to support the behavioural formulations discussed above.

Chapter 3

Cognitive Behavioural Approaches to Chronic Headache

3.1 Introduction and Overview

As previously mentioned, the recognition of Chronic Daily Headache (CDH) has only emerged very recently and there are, as yet, no studies that have directly examined the application of CBT to CDH. The vast majority of the literature on CBT approaches to chronic headache is based on the traditional headache categories of tension headache and migraine. The position taken here, based on both the headache continuum model and the symptomatology of CDH, is that most of this literature is directly relevant to CDH.

In critically reviewing CBT approaches to chronic headache in the context of the aims of the present research, the following areas will be examined: (1) theoretical underpinnings to the CBT treatment package and different components within it; (2) the efficacy of conventional CBT and minimal-contact approaches; and (3) treatment mechanisms and the role of cognitive processes in treatment change.

3.2 Theoretical Background to CBT

3.2.1 Assumptions

CBT has evolved from a diverse range of sources and has been applied in many different ways. It is therefore, not surprising, that CBT is not a single unified model but it instead represents a broad spectrum of approaches, all of which share four sets of assumptions: (a) that people actively process information to give meaning to their world, rather than being passive recipients of an external reality; (b) that cognitive or appraisal processes are central to the understanding of health function and dysfunction and should, therefore, be the prime targets for treatment intervention; (c) that any disorder can only be understood or described in terms of the reciprocal interaction of four systems - cognitive, emotional, behavioural and physiological; and (d) that patients can and will actively participate in their own treatment through implementing a range of cognitive and behavioural strategies introduced in therapy. Given that these

assumptions also lay at the heart of the current research programme, an evaluation of their validity forms a central theme in the ensuing literature review.

In the treatment of chronic pain generally and chronic headache specifically, a certain clinical convention has been established in that CBT is usually offered as a multicomponent treatment package that incorporates cognitive, behavioural, emotional, physiological and stress-management interventions. On the one hand, impressive results from outcome studies with chronic pain patients appear to vindicate this approach and perhaps explain why it has dominated (Bradley, 1996). However, a more critical evaluation suggests that the CBT 'package' has been developed and clinically implemented in an ad hoc manner without there being a clear unifying theoretical rationale or CBT model that might enhance the understanding of chronic headache and help to refine clinical practice (Morley, 1986). A further, related, charge is that treatment mechanisms are still poorly understood. For example: does change occur through the cognitive modality as assumed in some CBT interventions? Finally, if some type of positive cognitive change is the main mediator of positive outcome then there is the attractive possibility that such change could also be induced through a treatment format of CBT that minimises therapist-contact and maximises learning of home-based self-management strategies.

3.2.2 Evolution

CBT approaches emerged in the mid 1970s as a merger between the established behaviour therapy and the then newly rising cognitive therapies. Lack of space does not permit a detailed analysis of the interesting history of CBT approaches within psychology generally (see Beck, 1993; Hawton, Salkovskis, Kirk, & Clark, 1989; Rachman, 1997). However, in order to contextualise the CBT variants that have emerged in the treatment of chronic headache, a very brief outline of the evolution of CBT will be presented.

The roots of the behavioural component CBT lie in three influential developments in behavioural theory. These related to: classical conditioning, operant conditioning, and social learning theory. They all shared the central tenet that the emergence, maintenance, and decline of any behaviour can be explained by various mutually

compatible learning processes. Wolpe (1958) was amongst the first to formulate clearly, how these behavioural principles could be applied clinically (e.g. systematic desensitisation of fear in phobia treatment through the reinforcement or conditioning of responses that are incompatible with fear). This led to an explosion, from the 1960s onwards, of behavioural strategies designed to condition desired behaviours while eliminating undesired symptoms. In many disorders (including chronic pain and headaches), the reduction or elimination of unpleasant physiological symptoms was usually the patient's immediate concern. Therefore, treatment strategies such as relaxation training and biofeedback became an important aspect of behaviour therapy applications to chronic headaches. These treatment components usually came to be incorporated in the overall treatment package with the advent of joint cognitive and behavioural approaches.

The cognitive therapy component of CBT initially emerged from three main sources - which all shared the central tenet that faulty or maladaptive thinking processes were important in the aetiology and maintenance of psychopathology: (1) Beck and colleagues developed cognitive therapy for emotional disorders such as anxiety and depression (Beck, 1967, 1976; Beck, Emery, & Greenberg, 1985; Beck *et al.*, 1979); (2) Ellis and colleagues developed Rational Emotive Therapy (Ellis, 1962; Ellis & Grieger, 1977); (3) Meichenbaum (1975, 1977) developed cognitive-behaviour modification and Stress Inoculation Training. Aspects of all of these approaches have been included in different CBT treatment packages but Beck's and Meichenbaum's models have had the biggest influence in the treatment of chronic pain and headaches. The clinical implication seemed straightforward - change patients' faulty cognitions and negative beliefs to effect change in their behaviour and emotion. Consequently, various cognitive restructuring strategies became central to cognitive therapy.

By the mid 1970s, while behaviour therapy became well established and was backed up by reasonable empirically based support in specific areas, its neglect of cognitive processes was increasingly seen as a major limitation. Learning processes alone failed to explain the complex presentation of many psychiatric and psychophysiological disorders (Meichenbaum, 1976). Likewise, it was recognised from the outset that 'pure' cognitive therapy would not usually be enough to effect treatment change and that behavioural strategies would also be required. These limitations gave rise to a

natural rapprochement between the two therapies and, from the 1980s onwards, cognitive behavioural therapy established itself as the dominant approach within Western clinical psychology. An initial misunderstanding of CBT, perhaps encouraged by earlier emphasis on cognition, was that cognitive processes and cognitions were always at the starting point in the cause of behavioural or emotional difficulties. This has been modified in recent years and most people now accept that, while cognitive factors remain central, they have a reciprocal and complex interaction with emotional, behavioural and physiological factors.

3.2.3 Rapprochement with Gate-Control Theory of Pain

In the area of chronic pain and headaches, a further 'retrospective rapprochement' is apparent between CBT and the influential gate control theory of pain originally proposed by Melzack and Wall (1965). The gate-control theory transformed the whole conceptualisation of pain from being viewed as a unidimensional sensory-physiological disturbance to be seen as a multidimensional phenomenon in which psychological as well as physiological variables interact at an aetiological and symptomatic level. Melzack and Wall postulated a neurochemical gating mechanism in the spinal chord that acts as a meeting point between various peripheral impulses, and messages that are sent down descending pathways from the brain. The balance of these impulses determined the level of pain experienced. The most important contribution of this theory was the postulate that psychological variables, such as thought processes, emotion, and behaviour, modulate the pain experience through influencing the descending neural messages. While much research still needs to be done on developing gate-control theory and testing its postulates, it remains as the most useful working model in the area of chronic pain treatment.

A number of researchers, incorporating the broad ideas from gate-control theory, have developed specific CBT applications for chronic pain and headaches, and these have shaped most of the subsequent work in this area. Turk, Meichenbaum, & Genest (1983) provided a detailed practical CBT approach to pain based on Meichenbaum's (1977) approach to cognitive-behaviour modification. The emphasis was primarily on using cognitive modification techniques (e.g. positive self-talk) to manage pain indirectly through dealing with stress and negative moods. Holroyd & Andrasik

(1982a) outlined this same approach in the treatment of chronic headache and Blanchard & Andrasik (1985) incorporated this in their influential work. The assumption underlying this variant of CBT was that stress played a big role in exacerbating pain. As will be seen later, this assumption can be challenged, and other researchers, most notably, Bakal (1982), took a different approach to CBT in the treatment of chronic headache. In this approach, the emphasis was primarily on using cognitive-behavioural strategies to control headaches directly. The assumption being that it is the headaches themselves that are stressful rather than being exacerbated by external stressors. This assumption is also examined later.

3.3 Treatment Outcome

As the continuum model of headache has only begun to establish itself in the last few years, it is not surprising that the vast majority of the outcome literature on chronic headaches retains a distinction between tension-headache and migraine (though some studies have also defined a group termed 'mixed headache'). This distinction is retained here for the purposes of review. The review will focus on cognitive-based treatments, and on how these have compared with biofeedback and relaxation treatments. The voluminous literature on the efficacy of biofeedback and relaxation treatments alone, in combination, or against each other has been reviewed by several authors (e.g. Blanchard, 1992; Blanchard & Andrasik, 1982; Martin, 1993). The general conclusions are that both treatments are effective compared to no-treatment and to drug treatment but there is no difference in efficacy between them. Longer-term maintenance is less well researched but the few studies that exist indicate that maintenance of treatment effects is good from six months to upwards of two years.

With regard to cognitive-based treatments, reviews undertaken by prominent researchers (e.g. Blanchard 1992; Blanchard and Andrasik, 1982; Gauthier, Ivers, & Carrier, 1996; Martin, 1993) indicate that studies have consistently established treatment efficacy in comparison with no-treatment and found comparable efficacy to other treatments, particularly for tension headache. However, a closer inspection of individual studies, reveals a number of related difficulties that are not evident in summarised reviews: (a) many published studies give only very poor information

about treatment content or procedures; (b) in some studies, labels for the cognitive-based treatments seem face-valid but have poor content validity with regard to the actual treatment described; and (c) the extent of the 'cognitive component' in the overall treatment has varied greatly.

Consequently, a major problem in interpreting this body of evidence is that there is no standard treatment protocol for CBT, and studies have varied widely in their definition of the CBT treatment. This is evidenced in the variety of labels given to the purported cognitive-based treatments, e.g.: cognitive therapy, cognitive coping training, coping skills training, cognitive skills training, and stress coping therapy.

A large part of this variance clearly relates to the adoption of different theoretical perspectives concerning the link between headache, cognitions, and stress, discussed above. While the commonality amongst the cognitive therapy models of Beck, (Beck, 1976; Beck *et al.*, 1979), Meichenbaum (1977), and Ellis (1962) has ensured that elements from these have been blended into most cognitive-based treatments of headache, the aims of treatment and the targets for intervention have depended on assumptions made about the headache-cognitions-stress relationship. Most studies can be categorised loosely according to whether the cognitive-based treatment adopts the *stress-coping* approach first applied by Holroyd, Andrasik, & Westbrook (1977) and best described by Holroyd and Andrasik (1982a) or the *headache-related distress* approach first applied by Bakal, Demjen, and Kaganov (1981) and best described by Bakal (1982). In the stress-coping approach, headaches are presumed to be triggered and/or aggravated by psychosocial stress; in the headache-related distress approach, it is suggested that the reaction to headache itself is stressful and it is this, which fuels further headaches.

Two further factors are also likely to have played a part in encouraging the apparent diversity of cognitive-based treatments. Firstly, the very nature of cognitive phenomena means that there is scope for a far wider range of strategies across treatment studies than might be the case for relaxation training and biofeedback procedures. Secondly, the trend to combine different cognitive strategies with different behavioural/relaxation treatments has made it possible for many permutations of CBT to develop.

Despite the differing approaches adopted within cognitive-based treatments for chronic headaches, two concerns, roughly chronological, have preoccupied researchers' investigations of outcomes: (1) the efficacy of conventional, multi-session CBT; and (2) the efficacy of minimal-contact CBT. Before both of these lines of investigation are considered, it is necessary to discuss how *treatment outcome* itself has been measured.

3.3.1 Measuring Outcome

Headache outcome research has primarily relied on the self-monitoring of the frequency, intensity, and duration of headache to evaluate the success of treatment intervention. While a reliance on self-report seems understandable given that pain is largely a private and subjective experience, most researchers seem to have focused solely on the sensory component of this experience. This is at odds with the universal support for a multidimensional model of pain and headache. Thus, most outcome studies have failed to use a range of outcome measures encompassing cognitive, behavioural, and emotional dimensions as well as the sensory component.

Where studies have used additional outcome measures for these other cognitive, behavioural and affective measures, the specification of these and the instruments/methods used to measure them has been varied and inconsistent. It is therefore difficult to interpret different outcome studies on this basis. This has led to a dependence on self-reported changes in headache activity (i.e. frequency, intensity and duration of headaches) monitored through a headache diary as the only measures on which different studies can be reliably compared. The usual method has been to ask patients to rate their headaches four times per day on a six point scale (0 = no headache to 5 = most intense headache). A mean daily *headache index* has usually been derived by summing the 28 ratings for the week and dividing by seven. (The headache diary is reviewed later in a later section).

In addition to evaluating change in headache diary ratings through statistical significance, Blanchard and colleagues have also encouraged the reporting of the extent of *clinically significant changes* (Blanchard & Andrasik, 1985; Blanchard &

Schwarz, 1988). This has usually been defined simply in terms of a cut-off in percentage improvement on the headache index (e.g. at least 30% improvement). Following the suggestions of Blanchard's research group the convention has been to define clinical significance as at least a 50% improvement in the headache index from pre-treatment to post-treatment (Blanchard & Schwarz, 1988). These researchers have also encouraged the reporting of the percentage of patients per treatment group who achieve clinically significant change. Though not all earlier studies report this latter variable, it has increasingly become a convention in headache outcome research. Some studies also report clinically significant changes on other measures.

3.3.2 The Efficacy of CBT Treatment

No study has yet investigated a cognitive-based treatment of CDH. However, two studies reported by Blanchard's research team have investigated the behavioural treatment (mainly a mixture of biofeedback and relaxation training) of different CDH subgroups. In the first study it was found that chronic daily high intensity headache sufferers had a significantly poorer response to behavioural treatment than matched chronic headache sufferers (12.7% mean improvement on the headache index vs. 49.8% improvement) (Blanchard, Appelbaum, Radnitz, Jaccard, & Dentinger, 1989). In the second study, it was found that a CDH subgroup that consumed high amounts of analgesic medication had a much poorer response to behavioural treatment (Michultka, Blanchard, Appelbaum, Jaccard, & Dentinger, 1989).

The conclusion from both these studies was that CDH is particularly refractive to behavioural treatment. However, a criticism is that, in both studies, the methodology was based on a post-hoc case-control design whereby a database of previously treated headache sufferers was used to allocate to CDH and non-CDH groups. It is possible that a planned experiment would have led to different results through enabling more control over things such as treatment content.

Although no outcome studies have been conducted that have investigated the efficacy of CBT specifically in the treatment of CDH, much work has been done on the CBT treatment of chronic headache as a general disorder. It is therefore relevant to consider this literature base. Up until March 1999 there were approximately twenty published

studies that investigated a cognitive-based treatment for chronic headache. One further unpublished study (Laher, 1994) will also be discussed here as it is of direct relevance. Based on either the Ad Hoc (1962) or the IHS (1988) classification systems, the majority of these studies have focused on tension-headache while the remainder have investigated migraine or a combination of these headaches.

Approximately half of these studies constituted controlled trials, and the others were a mixture of single-case studies and uncontrolled investigations. Two strategies have been employed to investigate efficacy of cognitive treatments: (1) Studies that have evaluated the whole cognitive treatment by way of conventional pre-treatment to post-treatment/ follow-up comparisons, usually also involving a direct comparison with other treatment conditions; (2) Studies that have evaluated the incremental efficacy of the cognitive treatment.

3.3.2.1 Outcome Efficacy of the Whole Cognitive Treatment

Mitchell and White's (1976) single-case study of a chronic tension-headache patient was the first study that incorporated an explicit cognitive-based intervention. The intervention was based on a modified stress inoculation procedure proposed by Meichenbaum (1975), with the emphasis being clearly on managing stress situations that were presumed to aggravate headaches. A six-week baseline period was followed by fourteen weeks of intervention that included cognitive and relaxation strategies. Cognitive strategies included thought-stopping and cognitive rehearsal in preparation for stress situations. At post-treatment, the patient's headache activity had reduced to zero and this was maintained at 3-month and 6-month follow-up.

Holroyd, *et al.* (1977) provided one of the first controlled evaluations of cognitive treatment (they termed this stress-coping training). Tension-headache sufferers were assigned to three groups: stress coping training (n=10), frontal EMG biofeedback (n=11) and a waiting-list control group (n=10). Cognitive change strategies within the stress-coping training were based on the rationale that

> ...disturbing emotional and behavioural responses are a direct function of specifiable maladaptive cognitions. It was emphasised that tension headache results from psychological stress and that

> stress responses are determined by cognitions about an event or situation. (p.125)

The two treatment groups were given individual sessions every fortnight for a total of eight sessions. Mean reduction in headache activity was significantly greater for the stress-coping group. Statistically, the EMG biofeedback group did no better than waiting-list controls. Furthermore, all but one of the stress-coping group achieved clinically significant improvement (at least 50% reduction in headache activity) compared to just three in the EMG biofeedback group and none in the controls. These results were generally maintained at 15-week follow-up and also at 2-year follow-up (Holroyd & Andrasik, 1982b).

Holroyd and Andrasik (1978) attempted to replicate the above findings by administering cognitive treatment in a group format. Tension-headache patients were assigned to four groups: stress-coping training (n=10), stress-coping training combined with relaxation training (n=7), a headache discussion group with no explicit training of cognitive strategies but who were taught to analyse headache provoking situations just like the 'active' treatment groups (n=7), and a control group that merely monitored headache symptoms (n=7). The three treatment groups were given five sessions on a weekly basis. Results showed that all treatment groups improved significantly compared to controls (who remained unchanged on average) but that there was no difference between them. Thus, adding relaxation training to stress-coping training did not appear to add to treatment gains. The results were maintained at brief follow-up of six weeks.

Bakal, Demjen, and Kaganov (1981) investigated a CBT treatment based on Meichenbaum's cognitive theory of self control (Meichenbaum, 1977) but, unlike most other researchers, their focus was not on coping with antecedent stress but on the control of headache-related distress and other reactions that were assumed to be directly consequential to chronic head pain (the assumptions underlying this alternative approach were discussed earlier). Following three weeks of baseline monitoring of headaches, a mixed group of 45 chronic headache sufferers (17 tension headache, 15 migraine, 13 combined) were given twelve sessions of treatment on a weekly basis. As well as cognitive coping strategies, such as learning to reappraise

pain sensation in a less catastrophic manner, patients received relaxation training and EMG biofeedback as part of the CBT package. Pre-treatment to post-treatment comparisons showed significant mean reductions on the three dependent measures: headache hours per day, headache intensity and medication consumption. When the results were compared across the three different headache types in the mixed sample, no significant mean differences were found, lending support to the notion of a headache continuum model. Treatment effects were maintained at six-month follow-up.

The lack of a control group in Bakal *et al.*'s (1981) study means that the results need to be treated with some caution. Inspection of their data reveals large individual variation in treatment responsiveness, and a number of patients' baseline monitoring showed a spontaneous downward trend in headache activity. Bakal *et al.* also identified a group of patients with continuous or near continuous headache who were the poorest responders to treatment, suggesting that this form of CBT might be ineffective for people that would now be known as CDH sufferers.

Knapp and Florin (1981) attempted to test the efficacy of cognitive therapy for long-term migraine sufferers. Twenty patients were allocated to one of four groups: cognitive therapy (n=4), cephalic vasomotor biofeedback (n=4), a combination of these two treatments (n=4 with cognitive therapy first, n=4 with cognitive therapy given second), or waiting list controls (n=4). The treatment groups were seen for ten sessions of individual therapy over 5 weeks. All groups showed significant reductions in headache activity and the effects seemed to be maintained at 8-week follow-up. However, only the cognitively trained groups showed meaningful improvements in other psychological measures (irritability, depression, and positive self-evaluation). This seemed to support the widely held belief that cognitive therapy can have more wide-ranging treatment benefits. Knapp (1982) followed up these patients after one year and found that while the initial reduction in headache activity was maintained for all treatment groups (controls were not followed up) the gains on the psychological variables, shown initially by the cognitive groups, had disappeared. On the basis of this finding, Knapp concluded that broader stress factors are unlikely to play a big part in the aetiology of migraine and that perceived self-efficacy with regard to management of trigger situations might be more important. However, these interesting

findings must be seen as extremely tentative, given the very small number of participants per treatment cell.

Gerhards, Rojahn, Boxan, Gnade, Petrik, and Florin (1983) made an attempt to overcome the sample size problem reported in the Knapp and Florin (1981) study. Twenty-five migraineurs were allocated to either a cognitive stress coping group (n=13) or to a vasoconstriction biofeedback group (n=12). Following ten sessions of treatment, both groups achieved similar improvements in headache activity, thus replicating the findings of Knapp and Florin. However, changes on the psychological variables for the cognitive group were not as marked as those reported in the Knapp and Florin study.

In a study of the differential efficacy of stress coping training (n=16) and relaxation training (n=13) in migraineurs, Sorbi and Tellegen (1986) found that the two treatments were equally effective in terms of post-treatment measures of headache activity and medication consumption. However, there was a trend for stress-coping training to be more effective at eight-month follow-up and this treatment also appeared to enhance social assertiveness skills significantly. In one of only a handful of studies looking at longer-term efficacy of cognitive-based treatments Sorbi, Tellegen, and Du Long (1989) reported on a three-year follow-up of twenty-four patients from the Sorbi and Tellegen study. A strength of this study was that rather than relying on global retrospective information derived from one-off telephone or postal contact the researchers conducted follow-up interviews and also undertook further prospective data collection over a few weeks. It was found that, overall, treatment effects were maintained for both treatments and there were no significant differences between them. However, stress-coping training was found to be more effective in maintaining improvements in assertiveness, active problem solving, and depression. The over-emphasis on headache activity data that pervades the literature is evident in Sorbi *et al.*'s surprising conclusion that cognitive-behavioural treatments are no more superior than unimodal treatments such as relaxation training. In fact their findings suggest that efficacy of cognitive treatments *is* superior when a broader range of outcome measures are considered.

Martin, Nathan, Milech, and van Keppel (1989) were interested in investigating whether the association of depressive symptoms in chronic headache sufferers required a more conventional form of cognitive therapy. A newspaper-recruited mixed group of chronic headache sufferers (tension, migraine, and combined) were allocated to either a cognitive therapy treatment (n=26) based on Beck *et al.* (1979), or to self-management training (n=26). This latter approach was based on a package of 'self-care' developed by Winkler, Underwood, James, and Fatowich (1982) and included a number of strategies such as self-monitoring, relaxation training, and cognitive restructuring. Martin *et al.* also added cognitive components such as attention-diversion training and thought management borrowed from a manual for CBT described by Bakal (1982). Both treatments were administered in small group format with 4-6 per group and twelve groups in total. Groups met weekly for two-hour sessions for twelve weeks. At post-treatment, cognitive therapy and self-management training were equally effective at decreasing headaches and depressive symptoms on most measures. No correlation was found between changes in headaches and depressive symptoms in either treatment condition but cognitive therapy was found to be more effective for patients with more chronic and depressive symptoms.

Apart from the use of a non-clinical sample, an obvious criticism of Martin *et al.*'s (1989) study is that the two treatment conditions appeared to be more similar than dissimilar - both of them had a strong cognitive component. Therefore, the absence of major differences in treatment effects is not surprising. However, the study can be seen to provide useful information as to what the primary targets for cognitive-based interventions should be in different cases: e.g. the presence of significant depression may require an emphasis on more formal cognitive therapy rather than training patients in specific headache self-management strategies.

Murphy, Lehrer, and Jurish, (1990) compared the efficacy of cognitive-coping skills training (this treatment was based on that described by Holroyd *et al.*, 1977) to a relaxation training package consisting of progressive muscular relaxation and autogenic training. Twenty-three tension headache sufferers were allocated to one of these two treatments. Both treatments were delivered in small group format through eight weekly sessions. Cognitive therapy was found to be superior on the daily headache index, particularly on a measure of number of days with headache per week.

The proportion of participants who achieved clinically significant improvement in headache activity (i.e. ≥ 50% change) was much greater for the cognitive-coping skills group than for the relaxation group (9 out of 12 vs. 2 out of 11, respectively). In a similar study, but with a mixed group of chronic headache sufferers, ter Kuile, Spinhoven, Linssen, and Houwelingen (1995) sought to compare the efficacy of cognitive self-hypnosis treatment and autogenic relaxation treatment. Both these treatments achieved modest reduction of headache ratings (approximately 29%) compared to a waiting list control condition and no significant differences in outcome were found between the two at post-treatment or at six-month follow-up.

Two further studies, in which the primary focus was on the investigation of treatment mechanisms of CBT (and are, therefore, also discussed later) provided further evidence of the outcome efficacy of CBT treatment of chronic headaches. Firstly, in a controlled study, Newton and Barbaree (1987) found that CBT treatment for a group of headache sufferers with mixed symptomatology was clearly superior to no-treatment on measures of headache activity and medication consumption. The CBT treatment included a package of cognitive, behavioural and relaxation strategies, with the emphasis on headache-coping skills rather than the identification and management of stress. Treatment consisted of seven weekly sessions in which patients were seen in small groups of three or four. Secondly, a controlled study of a mixed group of chronic headache sufferers conducted by James, Thorn and Williams (1993) showed that two versions of CBT (differentiated by the presence or absence of explicit treatment goals) were superior to a waiting list control condition. Treatment effects were marked (statistically and clinically) on measures of headache activity, medication consumption, and pain coping but were less pronounced on psychological measures such as anxiety and depression.

3.3.2.2 Incremental Efficacy of Cognitive Treatment

An early single-case study of a female chronic tension headache sufferer treated by a cognitive-biofeedback package was published by Reeves (1976). Three phases of treatment were administered. The first phase focused on identifying possible trigger situations; in the second phase, the patient was taught cognitive coping skills to deal with stressful situations; and the final phase involved EMG biofeedback training. A

33% reduction in headache activity was found following the cognitive coping phase suggesting that this component added its own increment to treatment efficacy. Further reductions in headache activity were obtained following the addition of EMG biofeedback training with treatment effects being maintained at 6-month follow-up. This would indicate that a combined package was useful. The lack of a crossover control or a multiple baseline design in Reeves' study means that the possibility of spontaneous improvement and the confound of carryover effects cannot be ruled out.

Kremsdorf, Kochanowicz, and Costell (1981) addressed these problems through two single-case studies of tension headache in which they sought to evaluate the incremental effect of cognitive skills training in a treatment that also included EMG biofeedback training. Through varying the order of presentation of the cognitive and biofeedback components, they were able to show that headache reduction was associated with the cognitive component and that EMG biofeedback training was not additive. Interestingly, this was despite the influence of EMG biofeedback on frontalis muscle activity, thus suggesting that the presumed link between scalp muscle tension and tension headache is questionable.

Anderson, Lawrence, and Olson (1981) also used single-case methodology to investigate differential as well as the combined efficacy of cognitive stress-coping training and relaxation training. In an interesting and elaborate design, fourteen single cases were paired into seven experiments that investigated the single and combined effects of the two treatments; reversals and multiple baselines were included. All participants' headaches reduced significantly from baseline levels and the findings were inconclusive with regard to the superiority of any of the treatment permutations. A closer examination of Anderson *et al.*'s study shows that the relaxation treatment included the training of significant autogenic phrases - these might be considered to be part of a 'cognitive coping response'. Therefore, the finding of no difference across treatments together with the impressive outcome efficacy suggests that a common factor such as cognitive coping may be involved. Its is also possible that changes on variables other than headache activity (e.g. anxiety) may show a greater response to cognitive treatment. Laher (1994) provided tentative evidence for both of these possibilities, through a series of five single-case studies that investigated the CBT treatment of migraine.

Figueroa (1982) attempted to evaluate the extent of non-specific effects in cognitive treatments of headache. Fifteen chronic tension headache sufferers were assigned to one of three treatment conditions: (1) self-monitoring, (2) a traditional psychotherapy group in which headaches and underling conflicts were discussed and, (3) cognitive stress-coping training. The two active treatment conditions consisted of treatment in group format for seven weekly sessions. Only the cognitive group achieved significant decreases in frequency and severity of headaches, medication consumption, and disability of pain. It can be argued that the psychotherapy condition was not a credible treatment of psychotherapy given the brief duration of the programme. Nevertheless, even if that condition was no more than an attention-placebo, the efficacy of the cognitive stress-coping treatment still stands out.

Two large studies by Blanchard's team have provided further information about the incremental efficacy of cognitive treatment, one focusing on tension-headache treatment and the other on migraine treatment. Blanchard, Appelbaum, Radnitz, Michultka, Kirsch, *et al.* (1990) randomly assigned sixty-six tension-headache sufferers to one of four treatment conditions, two of which were active treatments and the other two were control conditions: (1) progressive muscle relaxation (PMR), (2) PMR plus cognitive therapy, (3) a credible attention-placebo called pseudomeditation and, (4) waiting-list headache monitoring. After eight weeks of treatment, both the active conditions achieved superior outcomes on headache activity and medication consumption ratings but, statistically, there was no advantage to adding cognitive therapy. However, a measure of clinically significant change in the headache index showed a trend for the combined PMR and cognitive therapy treatment to be more effective than PMR alone.

In a similar study, Blanchard, Appelbaum, Radnitz, Morrill, Michultka *et al.* (1990) split the data from several of their past studies of migraine patients (total N= 116) to one of four treatment conditions that had a standardised length of eight weeks: (1) thermal biofeedback training (TBF), (2) TBF plus cognitive therapy, (3) pseudomeditation and, (4) waiting-list headache monitoring. Analyses showed that the three treatment conditions achieved significantly better outcomes than waiting-list controls and no difference was found between these three. Thus, adding cognitive

therapy to TBF did not significantly improve outcome in migraine treatment. The researchers argued that this finding was consistent with the view that a stress component is more proximal and identifiable in tension-headache than it is in migraine and, therefore, cognitive therapy was more appropriate in the former rather than the latter. This view presumes that, (a) there is a linear link between stress and tension-headache and, (b) that cognitive therapy is purely useful as a stress-coping treatment. Both these assumptions are open to challenge. For example: appraisal processes may be important in mediating the stress response, and cognitive therapy strategies can also be directly aimed at pain coping and pain appraisal. A further interesting finding from Blanchard *et al.*'s study was that the attention-placebo condition seemed to be equally successful to the active treatments. This supports the possibility that cognitive changes are important in the treatment process.

3.3.3 The Efficacy of Minimal-Contact CBT

The empirical evidence to support the efficacy of CBT packages for chronic pain and headaches continues to accumulate. This body of outcome research has given credence to the convention of delivering CBT in the format of individual or group therapy, consisting of regular therapy sessions that blend in-session guidance and teaching with between-session practise by the patient. Therapy is usually delivered through weekly sessions with the whole programme lasting from around a minimum of eight sessions to upwards of sixteen. However, it is still not clear whether this convention of delivering CBT is always appropriate or even necessary. Researchers and clinicians have increasingly turned their focus on shorter forms or *minimal-contact* versions of CBT therapy (MC-CBT) with promising results (Haddock, Rowan, Andrasik, Wilson, Talcott, *et al.*, 1997; Primavera & Kaiser, 1992; Rowan & Andrasik, 1996). These MC-CBT treatments are largely reliant on home-based self-administration of treatment strategies guided by manuals or audiotapes with occasional therapist contact. If the efficacy of MC-CBT treatments can be shown to match that of conventional CBT this has many attractive implications concerning cost-effectiveness of clinical practice, service organisation, and training of therapists, as well as helping to add to the understanding of theoretical issues regarding the mechanisms of CBT.

Early uncontrolled studies supported the efficacy of home-based relaxation training in the treatment of tension headaches (Sherman, 1982; Steger & Harper, 1980). Apart from the lack of a control condition, a methodological weakness of these studies was that the treatment was ill-defined and this made it likely that it was applied inconsistently across participants. Later studies attempted to overcome these difficulties through comparing with a clinic-based treatment condition and ensuring that home-based and clinic-based treatments were well-defined (e.g. by use of a detailed treatment manual) and differed only in the amount of therapist contact /clinical sessions. These controlled studies consistently showed that home-based treatment (relaxation training alone or in combination with self-administered biofeedback training) was equally effective to matched intensive clinic based treatments for both tension headache (Blanchard, Andrasik, Appelbaum, Evans, Jurish, et. al., 1985; Teders, Blanchard, Andrasik, Jurish, Neff, & Arena, 1984) and migraine (Blanchard et al, 1985; Holroyd, Holm, Hursey, Penzien, Cordingley, & Theofanous, 1988; Jurish, Blanchard, Andrasik, Teders, Neff, & Arena, 1983). These findings have encouraged the routine use of home-based relaxation training even within an intensive clinic-based CBT treatment package. A logical question that arises from this is: can the cognitive component to treatment be self-administered as well, thus creating a complete home-based minimal-contact CBT package?

Mitchell and White (1977) were amongst the first to investigate the MC-CBT treatment of chronic headache through a group design. They developed a four stage treatment programme for migraine based on Meichenbaum's (1975) Stress Inoculation Training and consisting of headache monitoring as the first phase, followed by the phases of stress monitoring, self-controlled relaxation skills and finally, cognitive coping skills. Treatment was largely delivered through audiocassette tapes with minimal sessions with the therapist. Twelve migraine patients were assigned equally to four groups based on number of phases of treatment offered (i.e. from first phase only, right up to the full four phases). Length of time in 'treatment' and therapist were controlled. It was found that reduction of headache activity was inversely related to number of phases received. Thus, the three patients who received the full treatment including cognitive coping skills improved the most (83% mean reduction in headache activity compared to 55%, 4%, and 0% respectively for the other groups). The effects were maintained at three-month follow-up. While these findings tend to

support the incremental efficacy of the cognitive component in MC-CBT treatment the conclusions might have been more stronger had the research design controlled for treatment order effects and had the cell sizes (n=3) been considerably greater.

Kohlenberg and Cahn (1981) in their study of migraine treatment minimised clinical time even further by administering cognitive-coping treatment combined with relaxation and thermal biofeedback through a detailed self-administered treatment manual. Therapist contact was restricted to telephone or mail. A control group of migraineurs was also given a treatment manual but with general information about headaches rather than explicit instructions about CBT coping strategies. At 6-month follow-up, the control group reported a mean reduction in headache frequency of just 14% compared to a mean reduction of 62% for the group with the explicit treatment manual. While, these findings clearly supported the general efficacy of MC-CBT the specific contribution of cognitive strategies was not assessed.

Attanasio, Andrasik, and Blanchard (1987) investigated the specific efficacy of cognitive-coping component in a minimal-contact home-based treatment. They compared three treatment conditions for tension headache: (1) home-based relaxation only, (2) home-based cognitive coping only and, (3) clinic-based combined cognitive coping and relaxation treatment. Patients in all three conditions achieved significant decreases in headache activity but there was no difference between the conditions. Two flaws in Attanasio *et al.*'s study tend to reduce the validity of the findings. Firstly, the cell size for treatment completers was only seven or less for each of the three conditions. Secondly, patients in the home-based treatment were given five clinical sessions versus eleven sessions for the clinic-based group. Whether this sufficiently distinguished minimal-contact from intensive contact is questionable.

A later study by Appelbaum, Blanchard, Nicholson, Radnitz, Kirsch *et al.*, (1990) also investigated the incremental efficacy of cognitive therapy in MC-CBT in the treatment of tension headache. A home-based relaxation-only group was compared to a home-based combined cognitive and relaxation group. A waiting-list control group was also included in the design. Both treatment groups produced significant improvement over the controls on headache activity data but, again, no difference was found between the two home-based treatments. A further study by Blanchard's

research group (Blanchard, Appelbaum, Nicholson, Radnitz, Morill *et al.*, 1990) found that the addition of home-based cognitive coping training to a home-based combination of thermal biofeedback and relaxation training failed to add significantly to treatment outcome in a group of migraine and mixed headache sufferers. However, the two home-based treatments (with and without cognitive coping training) were superior to a waiting-list headache-monitoring group.

Other studies have suggested that minimal-contact cognitive therapy does have an additive effect in home-based treatments (Tobin, Holroyd, Baker, Reynolds, and Holm, 1988). Tobin *et al.* compared two home-based treatments for tension headache, namely, relaxation only and, relaxation combined with cognitive therapy. Patients in both treatments improved but the gains in the combined treatment were significantly greater. These results were maintained at 3-month follow-up. The incremental efficacy of cognitive therapy was further supported by the finding that the combined treatment was significantly more effective than relaxation-only in the treatment of headache patients who reported high levels of stress. These findings are at odds with those reported in other studies (reviewed above). A closer inspection of Tobin *et al.*'s treatment protocol for the cognitive based treatment seems to give a partial explanation for this discrepancy: Cognitive strategies in their MC-CBT treatment were individually tailored whereas in other studies a standardised cognitive protocol, aimed at stress coping, was administered to all patients without discriminating as to who would be most likely to benefit (i.e. the standardised treatment may have been quite inappropriate for some patients).

In a sample of migraineurs (N=48), Richardson and McGrath (1989) found that a combined relaxation and cognitive-coping training package administered as a minimal contact treatment was significantly more effective in reducing headache activity than a waiting list control condition and just as effective as a clinic-based format of the same treatment. Results were maintained at 6-month follow-up. The minimal-contact treatment was delivered through audiocassettes and a self-help treatment manual, which was controlled by mailing one chapter to the patient each week. Patients were seen at the beginning of treatment and once gain in the fifth week. The clinic-based treatment required patients to attend for eight, weekly, 60-minute sessions. Based on the ratio of percent change in headache index to therapist contact time, Richardson &

McGrath concluded that the minimal-contact version of CBT was clearly more cost-effective.

Two studies have also compared MC-CBT with pharmacological treatment (Holroyd, Nash, Pingel, Cordingley, and Jerome, 1991; Penzien, Johnson, Carpenter, & Holroyd, 1990). In the earlier study, Penzien *et al.* focused their investigation on a comparison between a home-based CBT-thermal biofeedback treatment and long-acting propranolol in the treatment of migraine. Both treatments achieved significant reductions in headache activity and there was little difference between them on most measures. However, the drug treatment was significantly more effective in reducing the peak headache intensity. This suggests that home-based CBT treatment has a role to play in migraine prevention and in the management of mild headaches but once an attack takes hold CBT coping strategies are ineffective.

In the other study, Holroyd et al (1991) compared a combined home-based treatment that included relaxation training and cognitive-coping training (n=20) with prophylactic medication (amitriptyline HCL) (n=21) for tension headache. In the home-based treatment, patients were given audiocassettes and a manual and, in addition, they were seen for three 1-hour treatment sessions at the start, middle, and end of the 8-week treatment. Both treatments obtained substantial reduction in the headache index (56% for MC-CBT and 27% for amitriptyline) but statistically significant difference was not found on this or other measures. However, in terms of clinical change, the cognitive-based treatment was consistently superior, particularly on psychological measures such as locus of control. Neurologists' ratings of patient improvement corroborated this pattern of results but the percentage ratings tended to be inflated by over 30% compared to the daily self-monitoring data.

3.3.4 Summary of Outcome Literature

In summary, the research evidence has consistently supported the efficacy of CBT treatment for chronic headache compared to no-treatment or attention-placebo conditions. Improvements in headache activity of around 50% have been obtained by most studies that have investigated a CBT treatment and this is supported by meta-analyses of the literature (Bogaards & ter Kuile, 1994; Penzien, Holroyd, Holm, & Hursey, 1985a). Reliable follow-up data is scarce but the indications are that treatment

gains are well maintained from 6-24 months after treatment. Minimal-contact CBT appears to be just as effective as conventional CBT. In terms of comparative efficacy, CBT does not appear to be statistically superior or inferior to other psychological and pharmacological treatments. However, when clinically significant changes are taken into account and a broader range of outcome measures are considered, over a longer period, then CBT approaches have been shown to have some advantage. A similar conclusion emerges from studies that have considered the incremental efficacy of CBT. These conclusions need to be tempered by a number of methodological limitations such as very small sample sizes in many studies, poor description and consistency of treatment protocols, and a reliance on the sensory component of pain as an outcome measure.

3.4 Treatment Mechanisms

While much energy and resources have been invested in empirical research pertaining to treatment efficacy, less research has been conducted on treatment mechanisms. Miller and Morley (1986) note

> Proving that a treatment has some therapeutic benefit is quite a different thing from showing why it works. A treatment like systematic desensitisation may be effective in reducing fears but its real mechanism of action may be quite different from that implied by the rationale on which the treatment was originally based. (p.182)

There is now growing interest not just in the outcome of treatment but also in the *process or mechanisms of treatment.* The investigation of treatment mechanisms is important from both a clinical and theoretical perspective. Firstly, from a clinical perspective, more efficient and refined treatment might be developed through improved understanding of the processes at work. Secondly, the theoretical framework and assumptions about the variables/processes that are presumed in the aetiology and maintenance of a disorder can be tested or better understood, i.e. does treatment work by modifying the purported variables or processes predicted by the theory?

In biofeedback and relaxation based treatments of chronic headache, several studies, despite obtaining good outcomes, have failed to find the changes in physiological processes that were presumed to mediate these outcomes (reviewed in next section). As a result, a growing tide of interest has centred on the role of cognitive factors as possible mediators of treatment change. This whole area has been informed by findings from the chronic pain literature which have consistently supported the efficacy of cognitive coping strategies in psychological treatments of pain, over and above known expectancy effects (see reviews by: Fernandez & Turk, 1989; Gamsa, 1994a, b; Jensen, Turner, Romano, & Karoly, 1991).

A parallel body of literature, with a specific focus on headache treatment, has accumulated in recent years. This body of literature is highly relevant to the understanding of CBT treatment of headache given the emphasis placed on modifying cognitions in this treatment. As psychological treatments (including CBT) have also made assumptions about headache pathophysiology, and as the case for cognitive factors has strengthened in the light of empirical inadequacies of pathophysiological models it is instructive to consider the physiological basis of psychological input first. This leads on to a consideration of the role of cognitive changes in non-CBT and CBT treatments. Finally, the wider literature is reviewed in order to understand the importance of cognitive factors in the context of the CBT model and CBT treatment for chronic headaches

3.4.1 Physiological Basis to Psychological Input

The headache field has been dominated by the notion that migraine is largely a vascular phenomenon and tension headache is largely a muscular phenomenon. Much psychological work was based on this dual framework and, assessment and treatment strategies were tailored accordingly. Blanchard and his colleagues were the strongest advocates of this approach (see Figure 3.1) and their prolific output of research had a major influence in shaping psychological approaches to headache (e.g. Andrasik, 1986; Blanchard & Andrasik, 1985).

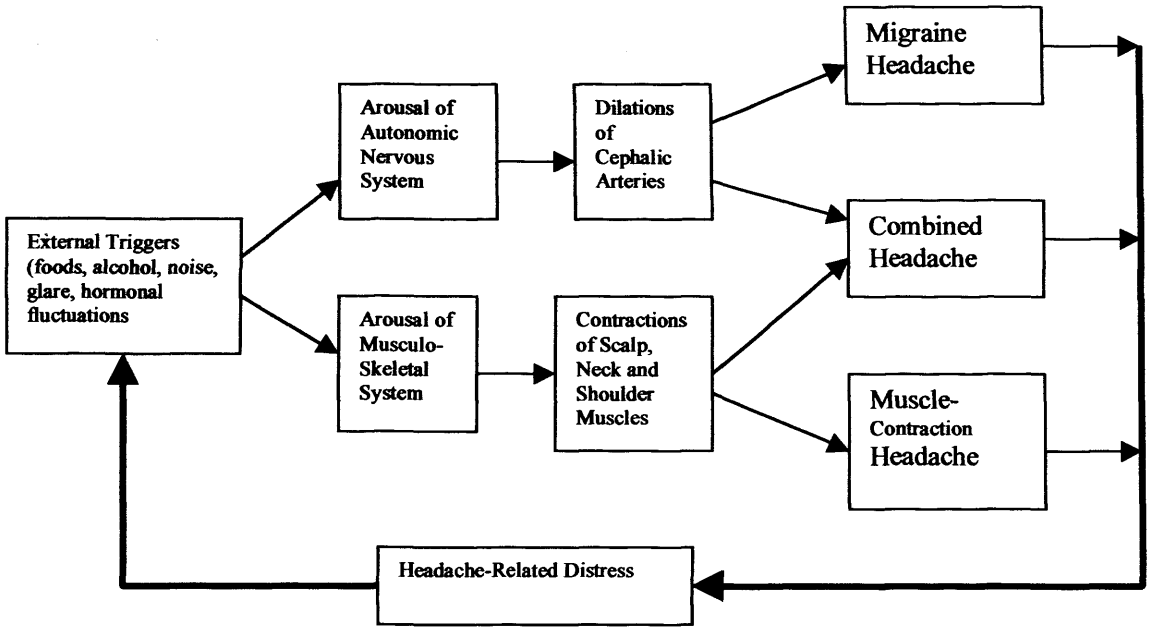


Figure 3.1 Headache pathophysiology and psychological factors
(From: Andrasik, 1986, p. 224)

On the one hand, the work of Blanchard and colleagues strengthened the credibility of psychological approaches to headache and increased the acceptance of psychology as a major player in a medically dominated field. On the other hand, this approach seems to have perpetuated the dual model of pathophysiology, the validity of which was becoming increasingly tenuous. A number of findings have questioned the validity of both the muscle-contraction hypothesis for tension headache and the vascular hypothesis for migraine.

3.4.1.1 Muscular Hypothesis

The pathophysiology of tension-headache (previously known as muscle-contraction headache) was thought to relate to prolonged contraction of the skeletal muscles, particularly in the neck and scalp in response to stress. Therefore, early psychological treatment focused on the application of biofeedback and relaxation training to counter muscle contraction in these regions (Budzynski, Stoyva & Adler, 1970).

However, the association of tension headache with muscle-contraction has shown to be variable and inconsistent rather than definite. Initial support for the link came from

Budzynski, Stoyva, Adler, and Mullaney (1973) who showed that frontalis muscle EMG training helped to reduce tension headaches. Such early studies naively assumed that any outcome change must be due to the input component (EMG training), and therefore, this was held up as support for the muscle-contraction hypothesis. This line of thinking, though logically consistent, is empirically flawed as other mechanisms, that were not measured or not controlled (e.g. cognitive expectancy), could also be responsible for headache reduction (Holroyd, Penzien, Hursey, Tobin, Rodgers, *et al.*, 1984). In fact, in the Budzynski *et al.* (1973) study the treatment also included general relaxation training.

Apart from studies looking at treatment response, the muscle-contraction hypothesis has not held up well when subjected to a number of further tests: (a) studies have failed to show a consistent difference between headache sufferers and controls either during headaches or in terms of generalised elevation of muscular tension (Flor & Turk, 1989); (b) when headache sufferers' muscular activity has been measured in headache and headache-free states the predicted differences have not materialised (Philips, 1977; Martin, Marie & Nathan, 1992); (c) a meaningful rise in head pain has not followed even when scalp muscle activity has been experimentally manipulated, through biofeedback, to levels higher than baseline or higher than those reported in other studies (Pearce & Morley, 1981).

The accumulation of these type of findings in the face of scant evidence to the contrary constituted a compelling case for the revision of the Ad Hoc system (1962). As previously mentioned, the IHS (1988) took heed of this evidence in distinguishing between tension-type headache that was either associated or unassociated with disorder of the pericranial muscles. However, the supposition in both the Ad Hoc and the IHS systems that significant muscular tension is unique to tension headache, has also been challenged by evidence showing that EMG levels in migraineurs are as high or even higher than those for tension headache (Bakal & Kaganov, 1977; Pozniak-Patewicz, 1976).

3.4.1.2 Vascular Hypothesis

The accepted model of migraine pathogenesis that underpinned the Ad Hoc (1962) classification was of a two-phase vascular process. The first phase was thought to involve constriction of intra-cranial and extra-cranial arteries in response to a trigger, and the second phase was presumed to involve reactive prolonged dilation of these arteries. Vasoconstriction was thought to cause the prodromes preceding migraine attacks, as blood supply to the brain became restricted, while vasodilation was thought to cause pain through pulsatile blood flow and inflammation of pain sensitive structures in and around the distended arteries. Evidence for this two-phase process was put forward by Wolff and his colleagues in the 1930s who used the known vasoconstrictive effects of the fungus ergot in the form of the drug ergotamine tartrate (Graham & Wolff, 1938). This drug appeared to reduce pain in migraine sufferers through cranial vasoconstriction. A later study by Tunis and Wolff (1953) appeared to confirm the link between arterial distension and pain - the pulse amplitude in the frontal branch of the superficial temporal artery was observed to be greatly heightened during migraine.

This view of migraine pathophysiology became well accepted for most of the subsequent decades and laid the basis for medical treatment, principally through ergotamine. It also underpinned early psychological approaches in the form of biofeedback training to induce cephalic vasoconstriction either through feedback of peripheral temperature, usually in the hands/fingers (Sargent, Walters & Green, 1973) or through presumed direct feedback of cephalic blood flow, usually in the temporal artery (Friar & Beatty, 1976).

The two-phase vascular process of vasoconstriction followed by vasodilation was, for a long time, thought to be the *sine qua non* of migraine headache. This was granted prominence in the criteria for both the Ad Hoc Committee (1962) and the IHS (1988) classifications and shaped the dialogue about headache mechanisms for many years.

However, empirical support for this two-phase vascular theory has been equivocal with regard to a number of crucial predictions, and studies have also raised further thorny questions: a) the predicted changes in arterial blood flow during supposed

vasoconstriction and dilation have not always been observed (Olesen & Edvinsson, 1988; b) more embarrassingly, for this theory, constriction and dilation phases have not been consistently associated with their purported effects of prodromes and pain, respectively (Feuerstein, Bortolussi, Houle & Labbe, 1983); c) while vasodilation, during the headache phase, has been well-supported, evidence for vasoconstriction before headache onset has remained tentative (Silberstein, 1992); d) the level to which any vascular dysfunction generalises (e.g. to other cranial arteries or even to the general vasomotor system) has remained unanswered, and studies that addressed this question have been hampered by significant methodological limitations to allow any firm conclusions (Morley, 1977); e) vasoconstrictive substances such as ergotamine have had mixed success in reducing acute migraine pain and have also had unexpected therapeutic effects in tension headache (Saper, 1989); f) finally, the assumption that significant vascular abnormalities are specific to migraine headache has been questioned by studies that have shown similar abnormalities in tension-type headache (e.g. Martin, Marie & Nathan, 1992).

While research into the vascular mechanisms of migraine continued to provide some interesting findings, particularly concerning reactivity to triggers and chronicity, most researchers were perhaps still too preoccupied with the whole notion of a vascular mechanism. Consequently, a serious consideration of possible alternatives was neglected. Nevertheless, a steady stream of writers began to dispute aspects of the putative vascular mechanism and challenged the presumed primacy of vascular processes (Raskin, 1988; Saper, 1989).

3.4.1.3 An Integrated Neurogenic Model

In recent years, in view of the equivocal research evidence for traditional theories of headache pathophysiology and in the light of several advances in neurophysiological assessment technology, the counter-evidence against established theories of headache pathophysiology has strengthened: neither the vascular theory of migraine nor the muscle-contraction theory of tension headache are, by themselves, considered to be viable any longer.

The current position adopted by most researchers, is that while vascular and muscular mechanisms are still important, these are subordinate to and induced by central neural processes (Silberstein, 1992; Lance, 1993; Giammarco *et al.*, 1998). Through the help of advances in medical measurement technology, a more detailed and sophisticated picture of headache pathophysiology is emerging such that specific neural pathways, neurotransmitter systems (particularly serotonin and adrenaline), and receptor sites have been shown to be involved in the neurovascular and/or neuromuscular mechanisms of headache. Furthermore, while neurogenic mechanisms are seen to be primary in headache pathogenesis and symptomatology, the modulating influence of descending messages from the brain as well as messages from peripheral systems are seen to play a crucial role. The overall picture, then, is of a complex, dynamic interplay of several factors driven mainly by central neural processes. This multifactorial model is consistent with the variability with which headaches seem to present and the frustratingly different ways in which headaches respond to treatment. More important, it is consistent with the view that psychological factors may have some bearing on the onset and modulation of different headache symptoms.

While this appears to be an exciting new development in the headache field (and it has been heralded as such by some medically trained researchers), there is a clear similarity with Melzack and Wall's gate-control theory of pain proposed over 30 years ago (Melzack & Wall, 1965). This exposes a hiatus in headache research in that, while psychological researchers have been aware of gate-control theory and its applications for many years, the research agenda in the headache field has been driven by researchers entrenched in an older more biomedical base. Nevertheless, these recent developments in headache physiology are not only compatible with CBT approaches but also provide a further rationale for CBT in the treatment of headache. This parallels developments in psychological approaches to chronic pain which saw gate-control theory fitting in nicely into the CBT approach to chronic pain since the early 1980s.

3.4.2 Cognitive Changes in Non-CBT Treatment

In a landmark study, Holroyd, *et al.* (1984) presented compelling evidence to suggest that in the EMG biofeedback treatment of tension headache, reduction in headache activity may be mediated primarily by cognitive changes in perceived self-efficacy (greater self-efficacy) and locus of control (more internal) rather than actual self-regulation of physiological processes. Forty-three college students suffering from tension-headaches were trained to either increase or decrease frontal EMG levels and everyone was given the same rationale, namely that this would decrease muscle tension. Within each of these two groups, half the group were given bogus computer feedback indicating that they were highly successful in achieving the required EMG levels, and half were given bogus feedback indicating that they were only moderately successful. Results showed that, regardless of actual changes in EMG levels and whether they were instructed to increase or decrease EMG levels, participants in the bogus 'high-success' group achieved significantly better reduction in headache activity than those in the bogus 'moderate-success' group. Furthermore, headache activity was significantly correlated with ratings of perceived self-efficacy and locus of control but not with changes in EMG levels.

Holroyd *et al.*'s (1984) study has been much quoted in the literature to explain anomalies such as no change in headache activity despite good training in biofeedback, or reductions in headache activity in the absence of measurable self-regulated physiological changes (some of this evidence was reviewed earlier in the discussion on the putative physiological processes in chronic headache). Surprisingly, while support of the role of cognitive variables has come from a number of other sources, replication of Holroyd *et al.*'s influential findings has only been attempted recently. Using the same bogus-feedback paradigm, Blanchard, Kim, Hermann, and Steffek, (1993) replicated these findings in a small clinical sample of tension-headache patients. However, a study by French, Gauthier, Roberge, Bouchard, and Nouwen (1997) failed to support the mediating role of perceived self-efficacy in the thermal biofeedback treatment of migraine. This was despite the researchers claiming that they were able to manipulate perceived success in the same way as the previous two previous studies. However, a closer examination of the data suggests that the manipulated difference between the 'high perceived self-efficacy' and 'moderate

perceived self-efficacy' condition, while statistically significant, might not have been as clinically large as the previous studies. It is also possible that the discrepant findings relate to the different ways in which self-efficacy and locus of control have been measured.

A more recent study by Rokiki, Holroyd, France, Lipchik, France and Kvaal, (1997) used combined biofeedback and relaxation training in the treatment of forty-four tension-headache sufferers. A control no-treatment condition was also included. It was found that while the treated group improved significantly on headache activity measures compared to the controls, such improvement was correlated with increases in self-efficacy and internal locus of control rather than with biofeedback induced changes in EMG activity. The researchers concluded that the biofeedback-relaxation treatment was effective but worked through a mechanism whereby it induced positive cognitive change rather than directly changing EMG activity. This is also consistent with the observation of many researchers that the whole process and gadgetry associated with biofeedback training may induce, in some patients, certain positive expectancies which in themselves can induce positive change independent of the actual treatment.

Using established psychometric questionnaires, Mizener, Thomas, and Billings (1988) assessed a number of cognitive variables in migraine patients (N=25) who received six weekly sessions of thermal biofeedback training. Change was evaluated through repeat measures at pre-, mid, and post-treatment. It was found that, as treatment progressed, patients became more internal about their beliefs to control their general health, were more confident in their ability to control physiological processes, and were ignoring their pain sensations more. There was a non-significant trend for patients to increase the use of coping self-statements and to decrease catastrophising. Some significant correlations were found between cognitive changes and physiological changes, and some of these correlated with outcome. However, a criticism of this study is that headache outcome was only measured on a short global scale, so it is difficult to interpret the link between cognitive changes and outcome.

3.4.3 Cognitive Changes in CBT Treatment

In a study by Holroyd and Andrasik (1978) (reviewed above), the surprising result that improvement in headache activity shown by the headache discussion group matched that of the two cognitive treatment groups led the researchers to examine the coping strategies used by all patients. Through post-treatment interviews, they found that those who received cognitive treatment reported using the cognitive strategies that they were taught while those in the headache discussion group appeared to develop their own cognitive coping strategies. This led the researchers to speculate whether the most important component of cognitive therapy was giving the patients means to problem-solve rather than the training of specific cognitive-coping strategies.

Knapp (1982) found that at 1-year follow-up of migraine patients treated in a previous study (Knapp & Florin, 1981) patients maintained initial headache improvement despite the re-emergence of psychological symptoms. This seemed surprising to the researchers since they had assumed that the post-treatment improvement in psychological symptoms mediated the reduction of headache activity at that time. Based on verbal self-reports of the patients, Knapp and Florin speculated that another mechanism, such as increased perceived self-efficacy in the management of specific trigger situations might be at work.

Newton and Barbaree (1987) sought to investigate whether CBT treatment worked by inducing cognitive changes such as less catastrophising, as predicted by the CBT model. In a controlled study, thirty-six patients with mixed headache symptoms were randomly allocated to either a CBT treatment condition or a waiting-list condition. Besides the use of conventional measures such as the headache diary, a novel addition by the researchers was the use of a 'thought-sampling' procedure to measure cognitions. This entailed the participants providing a series of cognitive reports via telephone contact during each headache episode. The contents of these reports were structured according to a previously piloted category of cognitions relating to: general appraisal, coping (cognitive avoidance or problem-solving), affect, and sensory appraisal. CBT patients, in comparison with controls, appraised headache attacks in a more positive manner and reported a more frequent occurrence of coping thoughts of

a problem-solving nature. Crucially, in support of the multifactorial CBT model of headache, changes in cognitive appraisal correlated with reductions in headache intensity. Disappointingly, the researchers did not assess affective or behavioural changes.

Ter Kuile *et al.* (1995) also attempted to disentangle the active cognitive ingredients of change in a cognitive-based therapy, which they termed 'cognitive self-hypnosis training'. This consisted of a blend of stress-coping and headache-coping cognitive strategies used by other researchers, with the addition of relaxation and several imagery strategies. The cognitive self-hypnosis training was contrasted with an autogenic training treatment condition. Each treatment condition was delivered for seven weekly sessions with taped exercises being provided for home practise. Cognitive variables of interest included catastrophising, perceived self-efficacy, and locus of control. The researchers hypothesised that: (a) only the cognitive-based treatment would produce significant changes on the cognitive variables as that treatment explicitly taught cognitive strategies; and (b) changes on these cognitive variables would be predictive of treatment outcome. It was found that cognitive therapy was more effective than the autogenic training in changing the use of cognitive coping strategies and appraisal processes that were the direct targets of treatment. However, the second hypothesis remained inconclusive, as treatment effects (headache activity and psychological distress) were only weakly correlated to cognitive changes. Patients in both treatment conditions improved and the outcomes were not significantly different. In the absence of a waiting-list or attention-placebo condition it is difficult to draw firmer conclusions about the specificity of cognitive changes and outcomes for each of the treatments.

Laher (1994) arrived at similarly inconclusive findings regarding the specificity of cognitive changes in cognitive treatment. This study reported on a series of five single-case experiments investigating the efficacy of CBT treatment of migraine. Three hypotheses were tested: (a) cognitive changes are specific to the cognitive therapy component; (b) the cognitive therapy component is more effective than the relaxation component; and (c) cognitive changes mediate positive clinical outcome. Four weeks of baseline headache monitoring was followed by eight weeks of individual therapy consisting of four weeks relaxation training followed by four

weeks of cognitive therapy. A post-treatment monitoring phase was also included. Outcome measures were headache activity, anxiety, and depression. Cognitive change measures of interest were self-efficacy and locus of control (both measured via a published instrument). It was found that headache activity was reduced by 50% in four out of the five patients and by 23% in the fifth. Clinically meaningful changes were also obtained in the other outcome measures.

Though positive changes were obtained, these were not specific to the cognitive therapy component. In fact, in some patients, marked cognitive changes were observed before the cognitive therapy component was introduced, supporting the view that cognitive change can take place in treatment conditions that are 'non-cognitive' in terms of explicit training of cognitive strategies. The study was inconclusive with regard to the second hypothesis as a crossover/ reversal of the two components was not included in the design. However, all patients reported that strategies learnt in the cognitive therapy phase were the most useful. Tentative support of the third hypothesis was provided by the correlation of outcome changes with cognitive changes.. However, in view of well-known problems in interpreting correlations, especially from a small sample, and without adequate control data even this tentative conclusion needs to be treated with caution.

3.4.4 The Role of Cognition in the CBT Model

The defining characteristic of CBT approaches to psychiatric and health problems has been their emphasis on modifying cognitions. Cognitive factors such as *appraisal* of pain and stress, *attribution* of causality, and *expectations* of personal and external coping resources are widely assumed to play some part in the way people cope and in how they respond to treatment (Bakal, 1982; Brewin, 1988; Haaga, 1997; Jensen *et al.*, 1991; Turk, *et al.*, 1983; Turk & Rudy, 1986, 1992; Williams, 1997). However, while there has been a ready acceptance of the role of cognition within a CBT framework, leading to a routine application of cognitive intervention strategies, a more detailed theoretical analysis and measurement of the putative cognitive constructs has only gained momentum in recent years. In this time, much work has been invested in delineating specific cognitive processes and structures, and how these interrelate with physiological, behavioural, and affective factors.

Besides the premises of cognitive therapy models (Beck, *et al.*, 1979; Ellis, 1962; Meichenbaum, 1977), and gate-control theory (Melzack & Wall, 1965, 1996), work on the role of cognition in chronic headache and pain has been informed by a number of other diverse sources. These include social cognition theory (Bandura, 1977, 1986, 1997; Leventhal, Meyer, & Nerenz, 1980; Leventhal, Nerenz, & Steele, 1984), the transactional model of stress (Lazaras and Folkman, 1984), and various theories of information-processing derived from cognitive psychology. These developments have led to many suggested cognitive constructs thought to be involved in treatment mechanisms and headache coping. However, the definition of the putative cognitive constructs remains inconsistent, and the proposed relationships between them are either unspecified or have not been empirically validated. Despite, this apparent lack of order, three constructs have received considerable attention and offer promising avenues for further investigation of treatment mechanisms in CBT. These relate to: (1) appraisal processes, (2) self-efficacy beliefs, and (3) locus of control beliefs.

3.4.4.1 Appraisal

Appraisal strategies, self-statements, and imagery pertain to the content and style of thinking - about general psychosocial circumstances and about pain specifically. Strategies to change negative beliefs (e.g. cognitive restructuring) have been central to CBT approaches to chronic headache. However, beyond the generalised propositions of the CBT model and gate-control theory, the rationale for these has varied depending on assumptions made about the stress-headache relationship.

In the stress-coping approach, the emphasis is on appraisals pertaining to the meaning of stressors and the evaluation of coping resources; CBT treatment is therefore based on cognitive reappraisal of stress situations. In the headache-related distress hypothesis the emphasis is on appraisal of the headache disorder itself and reactions to the headache with the result that CBT treatment has an emphasis on cognitive strategies to deal with headache symptoms directly. While CBT approaches to headache have tended to be based on one or the other of these hypotheses, the stress-coping approach has dominated CBT treatment of headaches. Empirical research tends to support both the stress-coping hypothesis and the headache-related distress

hypothesis (Demjen, Bakal, & Dunn, 1990; Marlowe, 1998a, b). However, the possibility that both are relevant to the understanding and CBT treatment of chronic headache seems to have been overlooked by most researchers in their preoccupation with technical differences. This is surprising given the widely held subscription to a multifactorial model of chronic headache in which both appraisal of pain and appraisal of stress can play a role and, furthermore, can interact with each other.

Most of the evidence to support the role of appraisal in headache coping has come from the general chronic pain literature. Early reports, based on clinical observations or ad-hoc examination of patients' self-reports in studies that had an alternative focus, suggested that coping thoughts and catastrophising mediated pain coping. More recently, the advent of several robust psychometric instruments designed specifically to measure pain appraisals (DeGood & Shutty, 1992) has enabled more rigorous and operationalised investigation of the cognitive variables. The general conclusions tend to support the predictions of a cognitive model i.e. negative thoughts (particularly 'catastrophising') are associated with poorer adjustment, and adaptive cognitions are associated with better adjustment (Flor & Turk, 1988; Flor, Behle, & Birbaumer, 1993; Lefebvre, 1981; Gil, Williams, Keefe, & Beckham, 1990; Newton & Barbaree, 1987; Rosentiel & Keefe, 1983;). Two interesting caveats to this line of evidence need to be noted. Firstly, pain appraisals appear to be more strongly related to the emotional and behavioural aspects of pain adjustment than to pain intensity. Secondly, it is the lessening of 'catastrophising' rather than the increase in positive cognition that seems to be the best predictor of improved adjustment. Both these points have implications concerning the focus of CBT strategies and the measurement of headache outcome.

3.4.4.2 Self-Efficacy Expectations

Self-efficacy refers to the belief in being able to perform an action. Bandura's initial concept of perceived self-efficacy which he defined as "the conviction that one can successfully execute the behaviour required to produce outcomes" (1977, p.193) has remained remarkably durable over the years. He later proposed a slight modification to encompass self-efficacy beliefs not just in behaviours but in any course of action (behavioural, affective and cognitive) (Bandura, 1986, 1997). Bandura hypothesised

that self-efficacy beliefs are determined by four types of information. In order of presumed potency with regard to determining self-efficacy beliefs and subsequent behaviour, these are: past performance or mastery, vicarious learning based on significant others, verbal/social persuasion, and emotional or physiological arousal.

Self-efficacy expectations are presumed to predict the range of coping strategies that an individual might consider, the amount of effort expended in these, and how long the individual might persevere with strategies in the face of obstacles. Thus, the construct of self-efficacy is useful in understanding adjustment to chronic pain and headache and the CBT treatment of these.

The empirical support for the role of self-efficacy in chronic pain and headaches has come from five main sources. Firstly, laboratory studies have shown that self-efficacy beliefs mediate the pain response through either directly influencing physiological mechanisms that are presumed to be involved in pain mechanisms (Bandura, 1992; Bandura, O'Leary, Taylor, Gauthier, & Gossard, 1987) or through moderating perceived pain control (Litt, 1988). Secondly, studies have shown that self-efficacy beliefs moderate the stress response which is thought to play a role in headache aetiology (Marlowe, 1998b). Thirdly, several studies have found a positive correlation between adjustment to pain and perceived self-efficacy (Lackner & Carosella, 1996; Spinhoven, ter Kuile, Linssen, & Gazendam, 1989; Rosentiel & Keefe, 1983). Fourthly, studies have found that self-efficacy beliefs predict compliance with the use of coping strategies that are taught within a treatment programme (Dolce, Crocker, Moletteire, & Doleys, 1986; Jensen, Turner, & Romano, 1991). Fifthly, as discussed earlier, a number studies that have directly investigated treatment mechanisms or that have commented on treatment mechanisms have consistently cited changes in self-efficacy beliefs as possible mediators of treatment outcome in chronic headache (Blanchard *et al.*, 1993; Holroyd, *et al.*, 1984) and chronic pain (Kores, Murphy, Rosential, Elias, & North, 1990; O'Leary, Shoor, Lorig, & Holman, 1988).

3.4.4.3 Locus of Control Beliefs

Locus of control (LOC) refers to the degree to which individuals attribute responsibility for undertaking any action as within themselves (internal locus) or

within other forces such as fate or doctors (external locus). The initial formulation of this concept was proposed by Rotter (1966) who constructed an Internal-External Scale (I-E) based on "a generalised expectancy that reinforcing events are either contingent upon a person's own behaviour (internal control) or upon forces outside one's own control (external control)" (p.80). Levenson (1972) proposed an extension to this scale by hypothesising three dimensions: internal control, control due to chance or fate, and control by powerful others. The application of the LOC construct has subsequently been extended to explain coping behaviour and treatment response in health-related problems and while this has involved a number of modifications to how LOC is measured, the tripartite scaling has remained useful and robust (Wallston, Wallston, & DeVellis, 1978; Wallston & Wallston, 1982; Martin, Holroyd & Penzien, 1990). Empirical research supports the importance of the LOC construct in the understanding of coping and response to treatment for chronic headaches and pain. A number of studies have shown that a stronger sense of internal control may be associated with less hopelessness, more confidence in being able to manage headaches, more effort expended in coping strategies, and better adjustment, while an external locus of control may be associated with resignation, catastrophising, over-reliance on health professionals, and poorer adjustment in headache sufferers (Martin *et al.*, 1990; Scharff, Turk, & Marcus, 1995) as well as in chronic pain patients (Fisher & Johnston, 1998; Harkapaa, Jarvikoski, & Vakkari, 1996; Jensen *et al.*, 1991).

3.5 Summary of the Literature Review and Gaps Identified

1. Although much research has been invested in the study of chronic headaches, the study of chronic daily headache (CDH) has emerged only very recently. CDH has been delineated as a variant of chronic headache that has both migrainous and tension-type features. It has also been identified as a clinically prevalent but difficult to treat disorder. However, studies of the treatment of CDH are still very scarce and, as yet, no study has investigated a CBT treatment package for CDH.

2. The distinction between migraine and tension headache has been increasingly questioned on theoretical and clinical grounds. Much empirical evidence has

shown considerable overlap in symptomatology and in purported physiological mechanisms, and evidence for some of the presumed defining features of each headache type has been inconsistent. Many researchers have therefore emphasised the notion of a headache continuum model. While the old distinctions have not been abandoned and may still be useful in some situations, most researchers agree that, from a psychological viewpoint, the headache continuum model seems more appropriate. The physiological and psychological features of CDH are consistent with a continuum model.

3. Paralleling developments in the understanding of pain and the movement away from a strictly biomedical approach, most researchers subscribe to multifactorial models of headache aetiology and maintenance. A dynamic and reciprocal interplay between different psychosocial factors (cognitive, affective, and behavioural) as well as different biophysiological factors is seen to be important. Within this, the role of cognitive factors and cognitive processes are thought to be particularly influential. Empirical support has centred on the roles of perceived self-efficacy, locus of control, and appraisal style in the aetiology and maintenance of chronic headache and in coping/adjustment. However, understanding of these cognitive variables and their role in chronic headache is still poor.

4. The apparent efficacy of cognitive-behavioural treatment (CBT) packages for chronic headache has been established and there has also been tentative empirical support for shorter (minimal-contact) versions of CBT. However, many studies have suffered from very poor sample sizes and have used restricted outcome measures. Furthermore, the application and efficacy of minimal-contact versions of CBT to CDH have not been experimentally tested.

5. There is less understanding about the treatment components that are effective or the specific change mechanisms that operate in CBT and non-CBT treatments. Some research has suggested that cognitive changes such as changes in perceived self-efficacy might be important and that these might also play a part in non-CBT treatments. The presumed role of cognitive

change as the mechanism of action within CBT approaches has not been adequately tested. In addition, it is not clear as to what extent the specific cognitive components of CBT are important.

3.6 Research Questions

The limitations identified in the literature, lead to the following research questions which were the focus of this research.

3.6.1 Treatment Efficacy

1. Is CBT effective in the treatment of CDH?
2. Is a minimal-contact home-based format of CBT as effective as a conventional (therapy-led) format?
3. Are any treatment gains from a CBT treatment programme maintained at longer-term follow-up?

3.6.2 Treatment Mechanisms

4. Does CBT lead to changes in cognitive processes (appraisal self-statements, self-efficacy, and locus of control beliefs)?
5. Do the cognitive changes mediate treatment outcome?
6. How important is the cognitive component in the CBT treatment package?

Specific hypotheses, based on the above questions, are stated at the beginning of the report for each study.

Chapter 4

Study 1: A Controlled Investigation of Therapist-Directed CBT versus Minimal-Contact CBT in the Treatment of Chronic Daily Headache

4.1 Introduction

This was a controlled treatment study which compared the outcome efficacy of minimal-contact CBT (MC-CBT) with conventional format clinically-intensive CBT (I-CBT) treatment for chronic daily headache. The study also investigated the degree to which these treatments induce cognitive changes, as predicted by the CBT model, and whether cognitive changes are associated with outcome changes.

4.2 Specific Hypotheses

Hypothesis 1:

There will be no significant difference in outcome efficacy between I-CBT and MC-CBT but both these treatments will be significantly more effective than a no-treatment waiting list (WL) condition.

Specifically, it was predicted that, in terms of pre-treatment to post-treatment comparisons, both I-CBT and MC-CBT will be significantly more effective than a Waiting List condition in bringing about the following positive changes on outcome measures:

i) Less headache activity. (lower headache index, lower peak headache intensity rating, and more headache free days).

ii) Lower anxiety and depression.

iii) Better behavioural and functional outcome (more medication-free days, and increase in physical functioning, role functioning, and social functioning).

It was further predicted that treatment gains from both I-CBT and MC-CBT will be equally maintained at 6-month follow-up.

Hypothesis 2:

I-CBT and MC-CBT will induce significantly more cognitive change compared to no-treatment but there will be no significant difference between I-CBT and MC-CBT.

Specifically, it was predicted that, in terms of pre-treatment to post-treatment comparisons, both I-CBT and MC-CBT will be significantly, and equally, more effective than a no-treatment control condition (WL) in bringing about the following positive cognitive changes:

i) Increased perceived self-efficacy

ii) Increased internal locus of control, decreased health professional locus of control, and decreased chance locus of control

iii) Increase in coping self-statements

iv) Decrease in catastrophising self-statements

It was further predicted that treatment gains from both I-CBT and MC-CBT will be equally maintained at 6-month follow-up.

Hypothesis 3:

There will be a significant association between cognitive changes and outcome changes such that more adaptive cognitive changes will be associated with better outcome.

Specifically, it was predicted that pre-treatment to post-treatment change on each cognitive measure (perceived self-efficacy, locus of control, and appraisal self-statements) will significantly correlate with pre-treatment to post-treatment change on each outcome measure (headache activity, affect, and behavioural/functional outcome).

4.3 Method

4.3.1 Research Design

A split-plot design was employed with one between-groups factor (Treatment Group) and one repeated-measures factor (Treatment Phase). The main hypotheses were addressed through a 3 (Treatment Group) X 2 (Treatment Phase) factorial. To minimise experimenter effects in delivering the two formats of CBT treatment, a second therapist, in addition to the researcher, was recruited and trained in the treatment protocol (termed therapist A and therapist B from now on). Counterbalancing across therapists was achieved by randomly allocating patients from each treatment group to therapists A and B. The levels of the independent variables and the broad group of dependent variables are shown in Table 4.1. Detailed definitions are presented later.

Table 4.1 Summary of Research Design for Study 1

<table>
<tr><td colspan="2" rowspan="2">Independent Factor I (Treatment Group)</td><td colspan="2">Independent Factor II (Treatment Phase)</td></tr>
<tr><td>Pre-treatment</td><td>Post-treatment</td></tr>
<tr><td>I-CBT</td><td>Therapist A
Therapist B</td><td colspan="2" rowspan="3">Dependent Variables:
1) Outcome
Headache Activity
Anxiety and Depression
Behavioural Outcome

2) Cognitive Change
Perceived Self-Efficacy
Locus of Control
Appraisal Strategies</td></tr>
<tr><td>MC-CBT</td><td>Therapist A
Therapist B</td></tr>
<tr><td>WL</td><td></td></tr>
</table>

Note on Follow-Up

A third follow-up condition within the Treatment Phase factor was included for the two active treatment conditions. Waiting list controls were not followed up for ethical reasons. Therefore, some of the analyses for longer term treatment effects and

treatment change processes involved within-group comparisons (post to follow-up change) and also between-group differences at follow-up.

4.3.2 Participants

4.3.2.1 Inclusion Criteria

The inclusion criteria were as follows:

(a) An organic cause for the headaches was ruled out by a consultant neurologist.

(b) A diagnosis of tension headache, migraine or combined headache was made by the neurologist and based on the Headache Classification System proposed by the IHS (1988).

(c) Patients had suffered the headache disorder for at least the previous 6 months.

(d) Patients were aged from 18-65 years, inclusive.

(e) Patients were not concurrently involved in any other trial.

(f) Patients did not start any new prescribed medication or treatment regimen from at least 4 weeks before joining this Study and throughout the whole course of their participation (up to and including Follow-up).

(g) Patients suffered headaches for at least 3 days per week.

4.3.2.2 Number of Participants

Sixty consecutive patients with chronic headache were referred to the study from a Neurology outpatients' clinic. The referral outcome of these is summarised in Figure 4.1 Thirty-eight participants fulfilled the inclusion criteria, and of these, one person dropped out of the study. Therefore, the total sample size was 37, representing 61.7% of the potential sample. Comparison with non-responders as to age/sex profile is presented in Table 4.2 (p. 79). Information about sample characteristics is presented in the Results section.

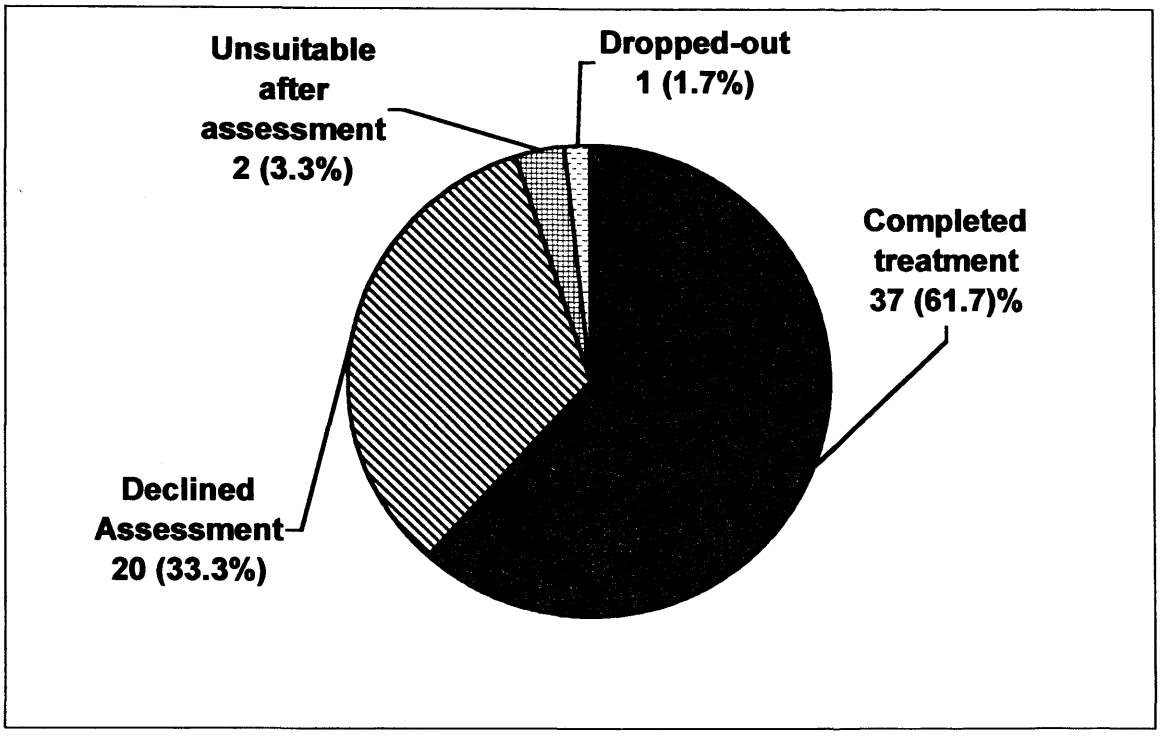


Figure 4.1 Referral Outcome. The pie chart shows the referral outcome for Study 1. Once patients had agreed to participate and went through assessment for the treatment trial, unsuitability and attrition rates were very low. This indicates that those who agreed to participate were, generally, well motivated to complete treatment.

4.3.2.3 Comparison of Participants and Non-Responders

In the current context, "non-responders" are defined as all those who did not complete treatment. From Figure 4.1 it can be seen that there were 23 non-responders (38.3% of the referred patients). No other information, besides age and sex, was available for non-responders, therefore, comparison with participants could only be made on this basis. As can be seen in Table 4.2, participants and non-responders were reasonably matched in terms of age and sex profile. Statistical tests (Chi-square for sex, and independent t-test for age) confirmed that there were no significant differences ($p > .05$, two-tailed) between participants and non-responders. As is typical in this clinical setting and similar to much previous headache research, far more women than men presented themselves for treatment.

Table 4.2 Comparison of participants and non-responders

		Participants[a] (N=37)	Non-Responders[b] (N=23)	Total (N=60)
Sex:	Males	12 (32%)	9 (39%)	21 (35%)
	Females	25 (68%)	14 (61%)	39 (75%)
Age (years):	Mean	37.73	37.30	37.57
	SD	13.31	11.19	12.45
	Range	19 - 66	19 - 60	19 - 66

[a]Participants are all those who completed their participation in the treatment trial; [b]Non-responders includes those who declined to attend for assessment and three patients who were deemed unsuitable or dropped out subsequently.

Note. No other demographic or clinical information was available for the majority of the non-responders (20/23).

4.3.3 Procedure

1) Patients who attended the neurology clinic and who fully met the inclusion criteria were verbally offered a referral to the study together with a brief explanation by the relevant doctor. If they were interested they were given an Information Leaflet (see Appendix C-1) about the study.

2) The doctor (on confirmation with the relevant Consultant Neurologist) then made a formal written referral to the Medical Psychology Department, using a specially developed and easy to complete Referral Form (see Appendix D-1)

3) The names of referred patients were randomly assigned to the three treatment conditions: (1) Clinically intensive CBT (I-CBT); (2) Minimal-Contact home-based CBT (MC-CBT); and (3) Waiting-List headache monitoring (WL). It was felt that random assignment at the pre-assessment stage was important for two reasons. Firstly, this minimised the likelihood of experimenter bias, based on clinical assessment. Secondly, from an ethical viewpoint, it was important to inform the patients at assessment as to when treatment would begin and what sort of treatment would be offered.

4) Following this random allocation, all patients were offered an initial assessment appointment with the researcher (a trained clinical psychologist). If the patient

failed to respond, a further appointment was offered. If a patient did not respond to the second appointment letter then no further contact was made and the referrer was informed.

5) Patients who attended the initial assessment session were asked to sign a Consent Form (see Appendix C-2). The assessment session consisted of a detailed semi-structured CBT interview lasting approximately one-hour. (see Appendix E-1) This session was intended to be a two-way information gathering exercise to confirm patients' general suitability for CBT treatment, and to establish specific difficulties. The latter were seen to be important in that individually tailored goals could be addressed within the standardised CBT treatment. As part of the baseline data collection, patients were asked to complete self-report questionnaires. They were also instructed to keep a daily Headache Diary (see Appendix E-2) for the next four weeks. This served as final confirmation of suitability for inclusion. Patients who were assessed as being unsuitable after return of baseline diaries, (i.e. who had less than 3 days with headache per week), were offered briefer intervention, which was not part of the study.

6) Patients assigned to the two treatment groups were instructed to bring their completed diaries and questionnaires to the first treatment session. They were given an appointment four weeks from the date of assessment. Waiting-list patients were instructed to mail their completed diaries and questionnaires (stamped addressed envelopes were provided for this). They were told that they would be offered group treatment in approximately 16 weeks time, and that they would need to complete headache diaries and questionnaires again at that time.

7) Patients in the two treatment conditions completed 12 weeks of the relevant therapy followed by 4 weeks of post-treatment headache monitoring and completion of post-treatment self-report questionnaires. A post-treatment review session was also conducted four weeks after treatment ended.

8) Follow-up data collection for the two treatment groups was initiated six months after treatment ended. Patients were contacted initially by telephone to confirm

that they had maintained their consent to complete follow-up information. They were then asked to complete and return the self-report questionnaires and a four-week Headache Diary. Ethical considerations meant that treatment could not be withheld unnecessarily for the waiting-list patients. They were therefore not followed-up at six months and were instead offered group CBT treatment as soon as control data, coinciding with post-treatment for the other two treatment conditions, had been collected.

In order to encourage treatment integrity, in addition to the treatment manual itself, detailed written instructions for both therapists, were used. These, together with regular cross-checking of treatment sessions enabled a high level of consistency to be maintained between therapists, and ensured that therapist variables were kept to a minimum. These procedures also fostered the maintenance of treatment integrity over time and across each treatment condition.

The number of participants per treatment group and therapist is shown in Table 4.3. These figures represent all those for whom pre-treatment to post-treatment data was complete.

Table 4.3 Number of participants per treatment group and therapist in Study 1

	Treatment Group		
	I-CBT	MC-CBT	WL
Therapist A	6	7	
Therapist B	6	5	
TOTAL[a]	12 (12)	12 (10)	13

[a] Figures in parentheses are number of participants for whom 6-month follow-up data was available

All treatment was conducted on an outpatients basis within a Medical Psychology Department at a General Hospital. Usual clinical protocols were observed and assessment, progress, discharge, and follow-up reports were sent to referring

consultants as well as the patient's General Practitioner. Immediate clinical and professional accountability was to the Head of the Medical Psychology Department. A summary of the procedure is presented in Figure 4.2 (also see Appendix D-2).

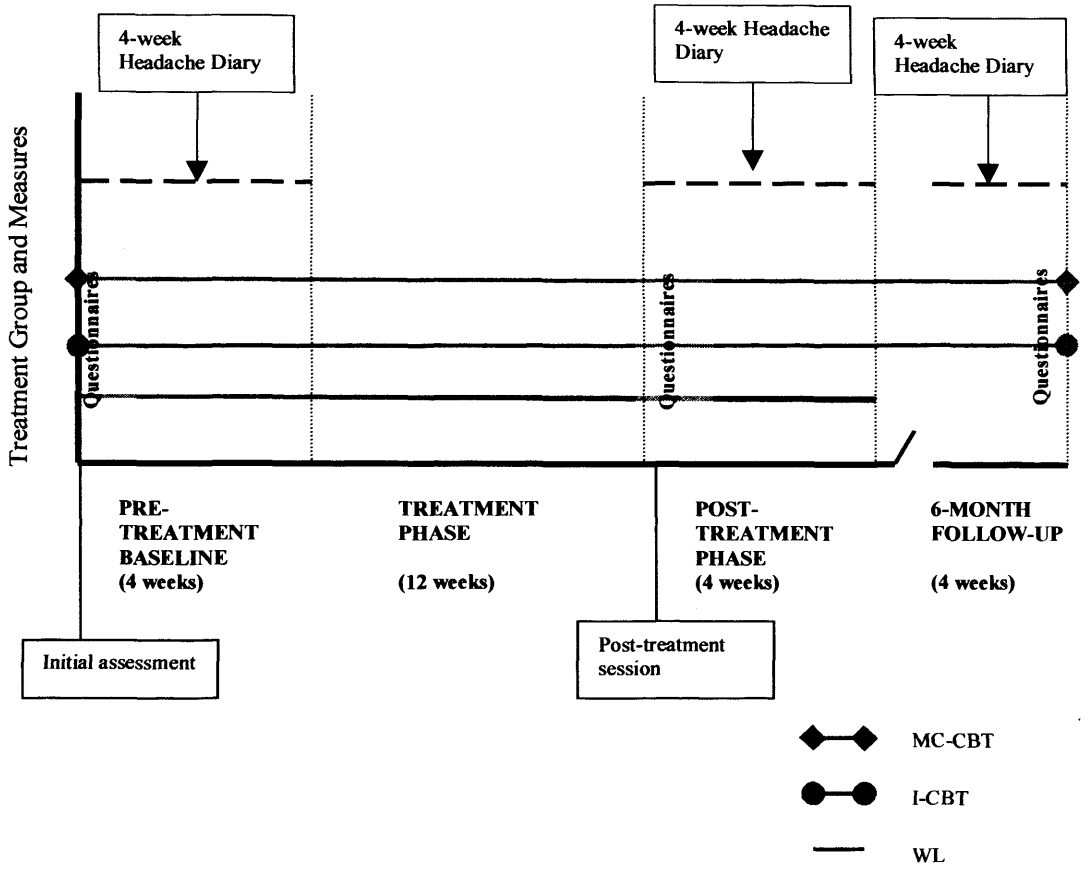


Figure 4.2 Summary of procedure for Study 1. MC-CBT = minimal-contact cognitive-behavioural treatment; I-CBT = intensive conventional format cognitive-behavioural treatment; WL = waiting list controls.

4.3.4 Treatment

The general CBT treatment approach used in the present study, was based on an amalgamation of the approach to cognitive therapy developed by Beck and colleagues (e.g. Beck, *et al.*, 1979) and Meichenbaum (1977). As discussed in the literature review, the application of CBT to the treatment of headaches has taken two routes: the stress-coping approach, advocated by Blanchard's group (e.g. Blanchard & Andrasik, 1985), and the headache-related distress approach, advocated by Bakal and colleagues (e.g.

Bakal, 1982). In the present study, both these approaches were felt to be important and complimentary rather than incompatible. They were therefore incorporated in the overall treatment package.

Both CBT treatment formats were standardised to include the following features:

a) Education (e.g. interplay of physiological, cognitive, emotional, and behavioural systems; gate-control theory etc.)
b) Individually tailored goals and goal planning
c) Pain control strategies
d) Stress management
e) Emphasis on active patient participation (through homework tasks and application of strategies)

4.3.4.1 Clinically Intensive CBT (I-CBT)

In this format, CBT was administered as weekly sessions for a period of twelve weeks. In practice, not all patients attended all twelve sessions but a mean of 9 sessions and range of 7 - 12 sessions was obtained. Each session after the first was structured to include review of homework, the introduction and demonstration of a new strategy, review of general well-being, and the setting of new homework assignments. This format represented conventional multi-session CBT that has been widely used in both psychiatric and physical health settings.

4.3.4.2 Minimal-Contact CBT (MC-CBT)

In this format of CBT, treatment was offered primarily through a specially developed self-management treatment manual (Laher, 1995). The twelve-week treatment was split into three major blocks, each of four weeks duration: (1) Introduction, education, goal-setting and relaxation training; (2) Cognitive restructuring and attention-diversion training; (3) Stress management and pain behaviour.

Just three clinical treatment sessions were offered and these were scheduled to introduce each of the four-week treatment blocks. Patients were given each part of the treatment manual in stepwise fashion in accordance with the clinical introduction of

each treatment block. Time was spent reviewing the previous block of treatment at each clinical session. A post-treatment review session was also held four weeks after the last block was introduced.

To encourage patient compliance, the treatment manual was structured to have clear, week by week instructions. No major problems were reported, by patients, in following the instructions within the treatment manual.

A summary of the treatment content is presented in Table 4.4.

Table 4.4 Summary of treatment content and scheduling for Study 1

BLOCK	WEEK	TOPICS	TREATMENT	
			I-CBT	MC-CBT
One	**Week 1**	Understanding Headaches Setting Goals Breathing Retraining	Session 1	Session 1 and treatment manual
	Week 2	Progressive Muscular Relaxation	Session 2	
	Week 3	Autogenic Relaxation	Session 3	
	Week 4	Applying Shortened Relaxation	Session 4	
Two	**Week 5**	Identifying Negative Thinking	Session 5	Session 2 and treatment manual
	Week 6	Challenging Negative Thinking	Session 6	
	Week 7	Attention Diversion Training	Session 7	
	Week 8	Overall Management of Thoughts and Mood	Session 8	
Three	**Week 9**	Understanding and Identifying Stress	Session 9	Session 3 and treatment manual
	Week 10	General Stress Management Plan	Session 10	
	Week 11	Exercise, Diet, and Sleep	Session 11	
	Week 12	Changing Your Headache Behaviour and Communication Involving Family and Friends	Session 12	
	Week 16	Post-treatment review	Review	Review

4.3.5 Measures Used and Review

The multidimensionality of chronic pain and headache has been well supported by a number of established composite questionnaires. These include the McGill Pain Questionnaire (MPQ, Melzack, 1975, 1987), the Headache Scale (Hunter, 1983)), the Headache Assessment Questionnaire (HAQ, Bakal, 1982), and West Haven-Yale Multidimensional Pain Inventory (WHYMPI, Kerns, Turk, & Rudy, 1985). The psychometric properties of these composite instruments have stood up well to empirical tests, and this, together with their clinical utility, has encouraged the wide use of such instruments (Jahanshahi, Hunter, & Philips, 1986; Melzack & Katz, 1992; Penzien, Holroyd, Holm, & Hursey, 1985b; Turk & Rudy, 1990). However, composite measures, while having the advantage of yielding useful information in one questionnaire, cannot offer a detailed assessment of each of the different components of pain: sensory, cognitive, affective, and behavioural. Furthermore, growing research and theoretical developments have indicated that even these different components can be split into sub-constructs that might require more sensitive assessment scales. For these reasons, and the stated interest in examining cognitive factors in detail, the researcher opted to use several specialist measures rather than a composite instrument.

Most of the measures used in this study were also used in a previous unpublished study of migraine patients (Laher, 1994) in which the measures proved satisfactory from a psychometric and practical perspective. Nevertheless, all measures were also piloted in the current study with a group of five chronic headache patients who matched the sample in terms of referral source (Neurology), headache activity, and demographic characteristics.

4.3.5.1 Headache Diary

The headache diary, in which patients rate the intensity of headache on a numerical scale regularly throughout the day, for several days or weeks, has established itself as the 'gold-standard' in headache outcome measurement. Various versions of the headache diary have been developed following Budzynsky *et al.*'s (1970) original design of a six point scale (0 = no headache) which the patient was required to complete hourly. A daily headache score or *headache index* was derived from the average of the 24 hourly ratings. Budzynski *et al.*(1973) added brief verbal descriptors

for each of the ratings and this system was incorporated by Epstein and Abel (1977) who developed a four times per day format of the headache diary. Collins and Martin (1980) found that these reduced number of ratings yielded the same information as more frequent ratings. These types of findings together with the greater simplicity of its use have meant that Epstein and Abel's diary format has gained the widest use in headache research. Blanchard, Andrasik, Neff, Jurish, and O'Keefe (1981) found that headache diary ratings made by patients correlated significantly with ratings made by significant others. This suggests that the headache diary is a valid and reliable indicator of real clinical change. Blanchard & Andrasik (1985) described the four-times-per-day format of the Headache Diary in detail and their recommendations formed the basis of the Headache Diary that was used in the current study.

The Headache Diary was presented on an A4 card which was folded up to 'pocket-size' format for easy portability (see Appendix E-2). Each card was designed to last for two weeks. Two sets of data were derived from the Headache Diary: (1) Headache Activity; (2) Medication Consumption.

Headache Activity

Participants were asked to rate their headaches four times per day (waking, midday, evening, and bedtime) on the following scale:

0 = No headache
1 = Very mild headache (aware only when attending to it)
2 = Mild headache (can be ignored at times)
3 = Moderate headache (pain noticeably present)
4 = Severe headache (cannot concentrate, can do undemanding tasks)
5 = Extremely intense headache (incapacitated)

In each of the treatment phases - pre-treatment, post-treatment, and 6-month follow-up - headache diaries were completed for four weeks. This is well above the minimum of 1-2 week monitoring period that has been found to yield reliable data (Blanchard, Hillhouse, Appelbaum, & Jaccard, 1987). Piloting of the diary in the current study, suggested that patients found it simple and non time-consuming.

In conformity with the majority of headache literature the following three headache parameters were derived from the headache diary:

1) *Headache Index (HI).* This is a mean total headache rating per day, for each week. This composite measure is assumed to give information about the frequency, intensity and duration of headaches. The headache index has a range of scores from 0 to 20 and is derived from the following formula:

$$\frac{\text{28 ratings for week}}{7}$$

2) *Headache-Free Days (HFD)* per week. This is simply the number of days with a total score of zero.

3) *Peak Headache Intensity Rating (PK).* This is the highest daily total per week (i.e. range of scores: 0 – 20).

For each of the above three parameters, a mean four-week score was derived for the pre-treatment, post-treatment, and 6-month follow-up phases.

Medication Consumption

The headache diary has also been used to record headache-specific medication consumption. To allow for variations in the potency of different medications, a scaled system has been developed and this has been used as the basis of a measure termed the Medication Index (i.e. average daily consumption based on a scaled potency multiplied by dosage) (Blanchard & Andrasik, 1985; Coyne, Sargent, Segerson, and Obourn, 1976). In the current study, piloting of this measure yielded very unreliable data. Furthermore, a consultation with a pharmacist suggested that the Coyne *et al.* scaling was outdated and not easily applicable to the current medications available in the UK market. However, the pilot research indicated that some patients were keen to monitor medication consumption with a view to reducing it. Therefore, it was decided to retain medication monitoring, but, for the purposes of analyses, the simpler measure of Medication Free Days per week (MFD) was used. While, not as sophisticated as the medication index, this measure was considered to be clinically meaningful. Again, a mean MFD score was derived for each of the three treatment phases.

4.3.5.2 Cognitive Measures

A number of instruments have been developed that wholly or partly measure cognitive aspects of the pain experience. Of the composite instruments, the Coping Strategies Questionnaire (CPQ) developed by Rosentiel and Keefe (1983) has received the most attention and has generally been supported as a useful clinical tool to assess a number of cognitive and behavioural coping strategies. However the CSQ is not easy to interpret as several different cognitive/ coping dimensions are measured. This complexity is reflected in inconsistent findings from factor-analytic studies (Degood and Shutty, 1992). Similar limitations apply to other well-known instruments such as the Survey of Pain Attitudes (SOPA, Jensen, Karoly, & Huger, 1987) and the Pain Beliefs and Perceptions Inventory (PBAPI, Williams & Thorn, 1989). The approach favoured in the current study was to use cognitive measures that have been developed to assess more specific aspects of cognition. This is consistent with the theme (see literature review) that well worn constructs such as 'cognition' can themselves be divided into meaningful sub-constructs.

Perceived Self-Efficacy

Much research has asserted the crucial role of self-efficacy beliefs in pain patients' level of functioning and response to treatment. However, measurement of self-efficacy has often been undertaken through ad-hoc and non-standardised scales or through items on standardised composite questionnaires that also measure several other things. Set against this, the 20-item Arthritis Self-Efficacy Scale (ASE, Lorig, Chastain, Ung, Shoor, & Holman, 1989) was the first instrument that was specifically developed to measure perceived self-efficacy for coping in a chronic pain population. The ASE has been shown to have sound reliability and validity (Lorig *et al.*, 1989). In an effort to develop a self-efficacy scale for the general chronic pain population, Anderson, Dowds, Pelletz, Edwards, and Peeters-Asdourian (1995) modified the ASE to form the Chronic Pain Self-Efficacy Scale (CPSS). The reliability and construct validity of the CPSS was assessed to be satisfactory. While both the ASE and CPSS have been shown to be sensitive within a chronic pain population, their content was considered to be inappropriate for assessment of self-efficacy specifically within a chronic headache population. A more appropriate instrument in the current context is

the Headache Self-Efficacy Scale (HSES) developed by N.J. Martin, Holroyd, & Rokiki (1993).

The HSES is a 51-item scale that assesses headache sufferers' beliefs that they can successfully perform coping strategies in the face of personally relevant aggravating factors. N.J. Martin *et al.* (1993) developed the HSES by collecting questionnaire and interview data from one-hundred and ninety college students who were diagnosed as suffering from recurrent headache (either tension-headache, migraine or mixed). While the use of a non-clinical population may appear to be a limitation of the HSES, it can be argued that such a population affords a broader distribution of self-efficacy scores for the purposes of psychometric analysis. A purely clinical group may show a floor-effect in the distribution of scores.

The 51 items comprised a list of the most commonly reported aggravating factors pertaining to such things as feelings, work-related stress, people's reactions, food and chemicals, and the environment. These items were drawn from an initial pool of 78 items identified by N.J. Martin *et al.*'s (1993) sample. Test-retest reliability over a three-week period was stable (r = .67). Predictive validity was tested by correlating self-efficacy scores with scores on a range of other established measures. It was found that, as predicted, lower self-efficacy correlated significantly with higher depression, higher anxiety, greater physical symptoms, and use of passive coping strategies. Partial correlations, with pain severity controlled, revealed that HSES scores could still significantly predict scores on the other measures (N.J. Martin *et al.*, 1993)

The format of the scale first requires respondents to tick all the items that are relevant to them. Then, each ticked item is rated by the sufferer in terms of the degree of confidence in being able to take action towards averting a headache. Ratings are done according to a five-point Likert scale: 1 = 'very confident', 2 = 'pretty confident', 3 = 'somewhat confident', 4 = 'little confidence', 5 = 'no confidence'. Items that are not endorsed are rated as 0.

For the purposes of this study, a composite score entitled perceived self-efficacy (PSE) was derived from the HSES. This weighted measure was derived by summing all the ratings on the endorsed items and expressing this as a percentage of the total

score possible for that number of items. E.g. if 23 items were endorsed, then the maximum self-efficacy score would be 23 x 5 = 115. Summed ratings would then be expressed as percentage out of 115. The scaling was reversed such that higher scores indicated higher self-efficacy ratings This measure takes into account the individual variation in the number of aggravating factors endorsed while enabling a comparison of total self-efficacy scores.

Locus of Control

The Multidimensional Health Locus of Control Scale (MHLCS, Wallston, Wallston, & DeVellis, 1978) has been shown to be a useful measure of the internality-externality of control beliefs in many health-related problems. However, its generalised content has not always been appropriate in the measurement of disorder-specific control beliefs, particularly pain (Main & Waddell, 1991). This has led to the emergence of several modified locus of control scales for different disorders. Pain-related scales that have focused on locus of control beliefs (LOC) have usually been part of more composite measures such as the SOPA (Jensen *et al.*, 1989) or the CSQ (Rosentiel & Keefe, 1983). However, the Beliefs in Pain Control Questionnaire (BPCQ, Skevington, 1990) and the Arthritis Helplessness Index (AHI, Nicassio, Wallston, Callahan, Herbert, & Pincus, 1985) are two measures that have reasonably robust psychometric properties and that offer detailed assessment of LOC beliefs in pain patients (Harkapaa *et al.*, 1996). Because of its specificity to headaches, good psychometric properties and ease of administration and scoring, the current study used the Headache-Specific Locus of Control Scale (HSLCS, N.J. Martin, Holroyd, & Penzien, 1990), which will now be described.

The HSLCS is a 33-item scale designed to assess headache sufferers' beliefs about the extent to which headaches can be managed by themselves (Internal locus), whether headaches are due to fate or chance (External-Chance), and whether control is in the hands of health professionals (External-Health-Professional). There are eleven items for each of the three loci. The items are statements which reflect commonly held beliefs about the determinants of headache occurrence or headache relief e.g. "Following my doctor's medication regimen is the best way for me not to get laid-up with a headache" (external-health-professional). The rating format for all items is in the form of a five-point Likert type scale: 1 = "'strongly disagree", 2 = "moderately

disagree", 3 = "neutral", 4 = "moderately agree", and 5 = "strongly agree". Item order is alternated between each of the three loci to minimise a response-set effect. The total score on each of the three subscales is derived by summing the eleven ratings for that subscale - higher score indicating stronger beliefs. N.J. Martin *et al.* (1990) tested the HSLCS on 207 clinically-diagnosed, recurrent headache sufferers, drawn from a college population. A principle components analysis yielded the hypothesised three-factors of Internal (I), Health Professional (HP), and Chance (C), with minimal correlations between the scales. Reliability was also found to be strong for each of the subscales with alpha coefficients of .88 (I), .86 (HP) , and .84 (C); and three-week stability coefficients of .75 (I), .78 (HP), and .72 (C). In terms of criterion validity, statistically significant correlations were found in the predicted direction between the scores on the HSLCS and measures of headache activity, affect, disability, and treatment preferences. That is, higher internal locus of control was associated with better adjustment and preference for self-regulation treatment. VandeCreek, Min, and O'Donnell (1992) evaluated the psychometric properties of the HSLCS on a headache clinic population and found this instrument to be as reliable and valid as Martin *et al.*'s original study concluded.

For the purposes of the current study the HSLCS was shortened to a 15-item scale by taking only the five items with the highest factor loadings on each subscale (based on data provided by Martin *et al.*, 1990). It is acknowledged that the shortened scale may not have the same reliability as the full scale. However, a reliability analysis indicated that the respective alpha coefficients were all above .75. Scoring was according to the format described above. Therefore, three scores were obtained for each person with the range being 5 - 25 on each scale:

1) *Internal Locus of Control (I-LOC)*
2) *Health Professional Locus of Control (HP-LOC)*
3) *Chance Locus of Control (C-LOC)*

Appraisal Self-Statements

The usual approach to developing pain cognition measures has been to factor analyse the cognitions of pain groups to see if (a) any meaningful factors emerge and (b) whether such factors are consistent with the CBT model. Not surprisingly, therefore,

much work has gone into distinguishing between 'adaptive cognitions' and 'maladaptive cognitions' and the effect of these on pain coping. Lefebvre (1981) using the Cognitive Errors Questionnaire (CEQ) found some support that the cognitive distortions described by Beck *et al.* (1979), such as 'catastrophising' and 'over-generalising', predicted depression and disability in a back pain group. However, later findings with the CEQ have been inconsistent and it is also a cumbersome instrument to use (DeGood & Shutty, 1992). As for other measures - limitations of the CSQ (Rosentiel & Keefe, 1983) in the current context have already been discussed. The Cognitive Evaluation Questionnaire (CevQ, Philips, 1989), The Pain Cognitions Questionnaire (PCQ, Boston, Pearce, and Richardson, 1990) and the Inventory of Negative Thoughts in Response to Pain (INTRP, Gil, *et al.*, 1990) have all been shown to be robust measures of pain cognition. However, due to its strong psychometric properties, good face validity with regard to the cognitive targets in CBT treatment, and ease of administration and scoring, a further instrument, the Pain-Related Self-Statements Scale (PRSS, Flor, *et al.*, 1993) was chosen for the current study.

The PRSS is an 18-item scale that assesses situation-specific aspects of patients' appraisal self-statements for pain that either promote or hinder coping (Flor, *et al.*, 1993). Half the items give a Catastrophising self-statements subscale score while the other half give a Coping self-statements subscale score. Flor *et al.* developed the PRSS on a sample of 377 chronic pain patients (a mixture of rheumatic, lumbar, and temporomandibular pain) and 38 healthy controls. Exploratory factor analysis confirmed that the PRSS consists of two 9-item subscales relating to Catastrophising and Coping respectively (all factor loadings were > .50). Absence of a correlation between the two subscales suggested that separate subscale scores rather than a composite score were more appropriate. The convergent and discriminant validity of the two subscales was well supported in further factor analysis using theoretically related and unrelated measures (e.g. WHYMPI, Kerns *et al.*, 1985). One-week test-retest reliability yielded stability coefficients of 0.87 (PRSS-Catastrophising) and 0.77 (PRSS-Coping). Impressive internal consistency was also found, Cronbach's alpha being 0.92, and 0.88 for the Catastrophising and Coping subscales respectively. Flor *et al.* also found that the PRSS subscales could discriminate between clinical patients and healthy controls. Regression analyses showed that the subscales accounted for a

large part of the variance in terms of pain interference and severity - the relationships being consistent with the predictions of a CBT model of pain. Finally, The PRSS subscales were shown to be sensitive to CBT treatment change - again in the predicted direction.

In terms of scoring, the PRSS items are introduced as statements that reflect the 'typical thoughts of a person in pain' e.g. ' I cannot stand this pain'. The respondent is required to rate on a 6-point Likert scale according to how often such thoughts entered their mind when they experienced severe pain (0 = never to 5 = always). For the purposes of this study, the word 'pain' was replaced by 'headache' on all relevant items. The following two measures were then derived as in the original scale:

1) *Catastrophising Self-Statements (CAT).* This was simply the sum of the ratings for the nine Catastrophising items (range of scores from 0 - 45). Higher scores indicated greater catastrophising.
2) *Coping Self-Statements (COP).* This was simply the sum of the ratings for the nine Coping items (range of scores from 0 - 45). Higher scores indicated greater coping thoughts.

Catastrophising and Coping items were randomly spread out to minimise a 'response set' effect and enhance the encoding of each item.

4.3.5.3 Affect

In recognition of the importance of emotional factors in the CBT model of chronic headache, most headache research has included some measurement of affect. Affect has usually been measured in one of four ways, with some researchers combining these: (1) Through personality scales such as the MMPI and the EPQ (2) As an item or number of items on broader measures of pain and coping such as the WHYMPI (Kerns *et al.*, 1983) and the Sickness Impact Profile (SIP, Bergner, Bobbitt, Carter, & Gilson, 1981); (3) Through broad based screening questionnaires for psychiatric symptoms such as the Symptom Checklist 90-R (SCL-90-R, Derogatis, 1983) and the General Health Questionnaire (Goldberg, 1972); (4) Through specialised instruments for anxiety and depression such as the Speilberger State-Trait Anxiety Inventory (STAI, Spielberger, Gorsuch, & Luschene, 1970), and the Beck Depression Inventory

(BDI, Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). However, these instruments were felt to be inappropriate for the current study for two main reasons. First, the need was for a quickly administered questionnaire to measure anxiety and depression, that would be sensitive to short-term change, rather than a long and multicomponent instrument that assesses many other symptoms and traits. Secondly, most of the specialist measures were developed for psychiatric populations and, while their suitability for pain patients has been demonstrated (see Turk & Melzack, 1992), a pervasive problem pertains to symptom overlap between affective and pain distress. This makes interpretation of some items (e.g. fatigue, which is common to both depression and headache) problematic. A measure which addresses these limitations is the Hospital Anxiety and Depression Scale (HADS) developed by Zigmond and Snaith (1983). This was used in the current study.

The HADS is a 14-item self-report scale with seven items tapping depression and seven items relating to anxiety. Based on clinical observation, Zigmond and Snaith (1983) included only those items which were thought to reliably indicate anxiety and depression symptoms. Although, the two subscales were originally based on clinical observation, later factor analytic studies have confirmed the two factors as well as establishing the internal reliability of the subscales (Clark & Fallowfield, 1986; Moorey, Greer, Watson, Gorman, Rowden *et al.*, 1991). Reliability and validity of the HADS concerning its use with chronic pain patients has also been well-supported (Tyrer, 1992).

Each item on the HADS represents common self-statements relating to anxiety and depression. Some items are worded to reflect positive statements e.g. " I look forward with enjoyment to things" and others concern negative feelings e.g. "I get sudden feelings of panic". Four alternative responses are given for each item, in rank order of how closely they reflect how the person has felt over the past week. The respondent is instructed to tick the response that applies on each item. Each item is scored on a scale of 0 - 3 with the scale reversed for some items such that higher scores always indicate greater distress. Anxiety and depression items are alternated to minimise response bias.

Consistent with usual scoring of the HADS the following two scores were derived for the purposes of this study:

1) Anxiety scale score (*HAD-A*, range = 0 - 21)
2) Depression scale score (*HAD-D*, range = 0 - 21)

Snaith and Zigmond (1994) have suggested that in terms of clinical norms, the following interpretations can be applied to each subscale score: normal = 0 -7; mild = 8 -10; moderate = 11-14; severe = 15 - 21.

4.3.5.4 Behavioural and Functional Outcome

From the patient's and the clinician's perspective, the impact of chronic headache on lifestyle is a meaningful indicator of overall adjustment to headache. In addition, within a multidimensional CBT model of chronic headache, the assessment of behavioural outcome measures is an important consideration. Assessment of behavioural outcome has been undertaken at two levels: (1) assessment of specific pain behaviours; and (2) assessment of overall quality of life. While pain behaviour and quality of life have traditionally been treated as separate constructs, they can also be viewed as different aspects of behavioural outcome if a less restrictive definition of 'behavioural' is applied. Pain behaviours have been assessed through questionnaires such as the Pain Behaviour Checklist (PBC, Philips & Hunter, 1981; Philips & Jahanshahi, 1986) while overall quality of life in chronic pain or headache groups has been assessed through instruments such as the Sickness Impact Profile (SIP, Bergner, *et al.*, 1981; Turner & Clancy, 1988), and the Nottingham Health Profile (NHP, Hunt, McEwen, & McKenna, 1985; Jenkinson, 1990).

For the purposes of this study, it was felt that the PBC yielded information that was too specific while the SIP and the NHP were considered to be too lengthy. Therefore, this study used what was seen to be a promising alternative: The Medical Outcomes Study Short Form-20 Health Survey Questionnaire for Headache (MOSH, Solomon, Skobieranda, & Gragg, 1993). Solomon *et al.* (1993) derived the MOSH from an almost identical instrument aimed at the general patient population as a general health outcome/ quality of life measure that was validated by Stewart, Hays, & Ware (1988).

The instrument proposed by Stewart *et al.* was itself part of a whole battery of health outcome measures developed in the USA as part of the Rand Health Insurance Study and the Medical Outcomes Study (MOS) (see Bowling, 1997 for review).

The MOSH is a 20-item self-administered questionnaire. Six health outcome measures can be obtained. These are Physical Functioning (6 items), Role Functioning (2 items), Social Functioning (1 item), Mental Health (5 items), Health Perception (5 items), and Pain (1 item). Solomon *et al.* (1993) tested the MOSH on a group of 208 chronic headache patients with mixed diagnoses. Internal consistency of the multi-item scales was found to be high with Cronbach's alpha ranging from 0.42 to 0.83 ($p < 0.001$). These reliability figures were comparable to those reported by Stewart *et al.* (1988).

The scoring system is in accordance with that described by Stewart *et al.* (1988) i.e. scores for each subscale are linearly transformed to a 0-100 scale with higher scores indicating better outcome/health. A composite score is not obtained. For the purposes of this study the Mental Health, Health perception, and Pain scales were omitted as that information was collected through other measures. The following measures were used for this study as indicators of Behavioural Outcome:

1) *Physical Functioning (PF)*

   Extent to which headaches interfere with a variety of activities (e.g. vigorous activities such as running, moderate activities such as moving a table etc.). These were rated on a three point scale: 'limited a lot', 'limited a little', and 'not limited at all'. These anchors were scored 0, 50, and 100 respectively. A mean PF score out of 100 was obtained for the six items.

2) *Role Functioning (RF)*

   Extent to which headaches interfere with usual daily activities. Each of the two items are rated as either 'Yes' (score 0) or 'No' (score 100) according to whether there is interference from headaches. A mean score out of 100 is obtained.

3) *Social Functioning (SF)*

Extent to which headaches interfere with normal social activities. This item asks ' have your headaches limited your social activities (like visiting friends or close relatives)?' Rating is done on a six point Likert scale ranging from 'all of the time' (score 0) to 'none of the time' (score 100).

Participants were asked to rate the above according to how things have been over the past 4 weeks (this is a modification from Solomon *et al.*'s (1993) format in which respondents were asked to rate over the past 8 weeks).

4.3.5.5 Demographic Information

In addition to the self-report measures, and in order to compare the profile of patients in the different treatment conditions, the following information was derived from the clinical assessment interview: sex, age, headache chronicity (years), general health (no other health problem vs. at least one other major health problem), ethnicity, marital status, and employment status:

A summary of the self-report measures used is presented in Table 4.5. (*Note.* Due to copyright restrictions, the actual inventories are not attached in the appendices)

Table 4.5 Summary of self-report measures used

OUTCOME			
Dependent Variable	**Specific Measures**	**Range of Scores**	**Scale Used**
Headache Activity	Headache Index (HI)	0 - 20	Headache Diary[a]
	Headache-Free Days per Week (HFD)	0 - 7	Headache Diary[a]
	Peak Headache Intensity (PK)	0 - 20	Headache Diary[a]
Affect	Anxiety	0 - 21	HADS-A[b]
	Depression	0 - 21	HADS-D[b]
Behavioural Outcome	Physical Functioning (PF)	0 - 100	MOSH[c]
	Role Functioning (RF)	0 - 100	MOSH[c]
	Social Functioning (SF)	0 - 100	MOSH[c]
	Medication-Free Days per Week (MFD)	0 - 7	Headache Diary[a]
COGNITION			
Dependent Variable	**Specific Measures**	**Range of Scores**	**Scale Used**
Self-Efficacy	Perceived Self-Efficacy Beliefs (PSE)	0 - 100	HSES[d]
Locus of Control	Internal (I-LOC)	5 - 25	HSLCS[e]
	Health Professional (HP-LOC)	5 - 25	HSLCS[e]
	Chance (C-LOC)	5 - 25	HSLCS[e]
Appraisal Strategies	Catastrophising Self-Statements (CAT)	0 - 45	PRSS[f]
	Coping Self-Statements (COP)	0 - 45	PRSS[f]

[a]Headache Diary (Blanchard & Andrasik, 1985); [b]Hospital Anxiety and Depression Scale (Zigmund & Snaith, 1983); [c]Medical Outcomes Study Scale for Headaches (Solomon *et al.*, 1993); [d]Headache-Specific Self Efficacy Scale (N.J. Martin *et al.*, 1993); [e]Headache-Specific Locus of Control Scale (Martin *et al.*, 1990);[f]Pain-Related Self-Statements Scale (Flor *et al.*, 1993).

4.4 Results

4.4.1 Overview of Analyses

All data were inputted into, and analysed through, SPSS Release 8 for Windows 95. Most of the data were analysed through parametric statistical tests. This was based on a preliminary exploration of the raw data for each dependent measure which suggested that the three main criteria for parametric tests were met, i.e. a normal distribution of scores, homogeneity of variance, and scores that are derived from a measure that can be treated as intervally scaled as a minimum (Howell, 1997).

Kolmogorov-Smirnov tests for normality indicated that the distribution of scores on most of the dependent measures did not deviate significantly from a normal distribution ($p > .05$). Tests for skewness and kurtosis were more variable, but no extreme distributions were obtained. However, on three of the measures, namely, headache-free days, medication-free days, and role functioning, there was in fact significant deviation from normality. For these measures an equivalent non-parametric test was used. Tests for homogeneity of variance of the treatment groups, using the Levene statistic, were non-significant in the vast majority of statistical analyses. Where the Levene's test was significant, an adjusted test statistic was computed by SPSS for the purposes of significance testing on the given measure. Finally, as all the questionnaires have been standardised to some degree, their derived composite scores can be treated as interval data despite individual items being measured on an ordinal scale (Howell, 1997).

To conform with the general convention in the headache outcome literature, and to allow for meaningful comparisons with previous empirical work, the following analyses were undertaken:

1) Split-plot analyses of variance (ANOVAs) to test for general treatment effects, and for interactions between Group X Phase (Pre vs. Post).
2) Pairwise t-tests (using the Bonferroni method, Howell, 1997) on post-treatment scores to establish, more clearly, as to where the treatment effects occurred. Specifically, it was planned to test the following predictions relating

to treatment outcome: no significant difference between I-CBT and MC-CBT; significant difference between I-CBT (better outcome) and WL; significant difference between MC-CBT (better outcome) and WL. As a specific direction was predicted in the latter two comparisons, a one-tailed significance test was used, whereas a two-tailed test was used in the I-CBT vs. MC-CBT comparison as no direction was predicted. To minimise the likelihood of Type I error due to several comparisons, the family-wise alpha level was kept to p = .05 by dividing by the number of comparisons (Kinnear & Gray, 1999). Therefore, for three comparisons on a given dependent measure: I-CBT vs. MC-CBT, I-CBT vs. WL, and MC-CBT vs. WL, alpha was set at p = .017.

3) Chi-square analyses relating to between-group comparisons of number of patients that have achieved clinically significant improvement on the headache index.

4) Related *t*-tests to examine any change within each of the two active treatment groups from post-treatment to 6-month follow-up (WL controls were not followed up). Maintenance of treatment gains at follow-up would be indicated by a non-significant *t*-test result for each treatment group. No significant increment or decrement, on given measures, was predicted and it was expected that treatment gains would be maintained in both treatment groups. Again, to retain an overall alpha level of p = .05, for each pair of related *t*-tests on every dependent measure, alpha was set at p = .025.

5) Correlational analyses of change scores on outcome measures with change scores on hypothesised moderating variables (e.g. self-efficacy). Due to the sample size, it was decided to explore these relationships through bivariate Pearson's Product-Moment correlations. However, exploratory multiple regression analyses was also conducted to see if prediction of outcome change on a given measure could be improved by considering a number of cognitive variables collectively.

4.4.2 Sample Characteristics

4.4.2.1 Demographic Profile

The average age of the total sample was 37.73 years (SD =13.31) and participants ranged in age from 19 to 66 years. Overall, there were approximately twice as many females as males (32.4% males, 67.6% females). In terms of ethnicity, most participants were White European (78.4%) and the rest were from an Asian ethnic background (21.6%). The demographic profile of the total sample and the breakdown across the three treatment groups is shown in Table 4.6. A one-way ANOVA for Age, and Chi-square tests for the categorical variables confirmed that there were no significant differences between the three treatment groups. Similar tests confirmed that there were no major differences concerning profile of patients allocated to each of the two therapists, therefore supporting the validity of the counterbalancing procedure across therapists.

Table 4.6 Demographic profile of the sample (Study 1)

		Treatment Group			TOTAL
Demographic Variable		**I-CBT (n=12)**	**MC-CBT (n=12)**	**WL (n=13)**	SAMPLE (N=37)
Age (years)	Mean	35.0	42.67	35.69	37.73
	SD	16.48	11.80	10.88	13.31
	Range	19 - 66	21 - 57	22 - 55	19 - 66
Sex	Males	3 (25%)	7 (58.3%)	2 (15.4%)	12 (32.4%)
	Females	9 (75%)	5 (41.7%)	11 (84.6%)	25 (67.6%)
Ethnicity	White European	9 (75%)	10 (83.3%)	10 (76.9%)	29 (78.4%)
	Asian	3 (25%)	2 (16.7%)	3 (23.1%)	8 (21.6%)
Marital Status	Single/ Divorced	5 (41.7%)	5 (41.7%)	4 (30.8%)	14 (37.8%)
	Married/ Co-habiting	7 (58.3%)	7 (58.3%)	9 (69.2%)	23 (62.2%)
Employment Status	Working	7 (58.3%)	9 (75%)	9 (69.2%)	25 (67.6%)
	Studying/ training	4 (33.4%)	1 (8.3%)	3 (23.1%)	8 (21.6%)
	Retired	1 (8.3%)	1 (8.3%)	0	2 (5.4%)
	Homemaker	0	1 (8.3%)	1 (7.7%)	2 (5.4%)

4.4.2.2 Clinical Status

The clinical status of the sample at pre-treatment is shown in Table 4.7. The three treatment groups were comparable with regard to headache activity, affect, and general functioning. Anxiety scores were more elevated than depression scores for all groups and were within the moderate clinical range. Depression scores were within the non-clinical range but slightly elevated. This pattern is consistent with that found by other researchers who have investigated chronic headache or pain. Daily Headache Index scores averaged around 10. This figure is much higher than that generally reported in the literature (around 5) for pre-treatment headache activity and confirms that the sample came from a population of chronic, high frequency, high intensity headache sufferers.

One-way ANOVAs for each of the variables shown in Table 4.7 and for all other dependent variables (including cognitive measures) confirmed that there were no significant differences between the Treatment Groups at pre-treatment. Again, similar tests showed that the between-therapist difference in terms of clinical status of patients at pre-treatment was non-significant.

All patients reported experiencing a mixture of migraine and tension-type symptoms with the latter being more frequent.

Table 4.7 Clinical status of participants at pre-treatment.

Clinical Status		Treatment Group I-CBT (n=12)	Treatment Group MC-CBT (n=12)	Treatment Group WL (n=13)	TOTAL SAMPLE (N=37)
Other Health Problems	None	8 (66.7%)	9 (75%)	9 (69.2%)	26 (70.3%)
	Some	4 (33.3%)	3 (25%)	4 (30.8%)	11 (29.7%)
Headache Chronicity (years)	Mean	11.0	7.50	9.69	9.41
	SD	9.17	7.33	10.09	8.64
	Range	1 - 30	2 - 25	1 - 30	1 -30
Headache Index	Mean	10.40	10.16	10.23	10.27
	SD	2.76	2.07	1.76	2.16
Headache-Free Days (per week)	Mean	0.94	0.69	0.75	0.79
	SD	1.03	0.90	0.93	0.94
Medication-Free Days (per week)	Mean	2.42	4.69	3.83	3.65
	SD	2.91	2.55	2.51	2.75
HAD Anxiety	Mean	9.50	9.00	12.00	10.22
	SD	3.73	4.07	3.81	3.99
HAD Depression	Mean	3.75	6.75	6.62	5.73
	SD	2.30	2.96	4.15	3.46
Physical Functioning[a]	Mean	49.33	50.58	57.08	52.46
	SD	18.77	18.17	23.32	20.05
Role Functioning[a]	Mean	41.67	41.67	46.15	43.24
	SD	41.74	35.88	43.11	39.37
Social Functioning[a]	Mean	26.67	35.00	40.00	34.05
	SD	15.57	15.08	25.82	19.92

[a] Measured through the MOSH scale.

4.4.3 Results for Hypothesis 1

There will be no significant difference in outcome efficacy between I-CBT and MC-CBT but both these treatments will be significantly more effective than a no-treatment waiting list (WL) condition.

4.4.3.1 Overall Headache Activity

Table 4.8 shows the pre-treatment to post-treatment outcome on headache activity. Overall, consistent with the prediction within Hypothesis 1, the reduction in headache activity within the two active treatment groups was significantly greater than that within controls and there were no significant differences in outcome between I-CBT and MC-CBT. A more detailed discussion of the individual headache outcome measures follows below.

Table 4.8 Treatment outcome on headache activity measures

Variable	Treatment Group[a]	Treatment Phase				F (d.f.)	
		Pre		Post			
		Mean	SD	Mean	SD	Phase	Group X Phase
Headache Index	I-CBT	10.40	2.76	5.26	3.24	53.85** (1, 34)	7.42* (2, 34)
	MC-CBT	10.16	2.07	6.25	3.54		
	WL	10.23	1.76	9.24	1.93		
Peak Headache Intensity	I-CBT	14.27	1.78	9.65	3.18	43.93** (1,34)	3.55* (2, 34)
	MC-CBT	13.75	2.45	10.21	3.89		
	WL	13.52	2.17	12.02	3.10		
Headache -Free days (week)	I-CBT	0.94	1.03	1.28	0.95	(not applicable)[b]	
	MC-CBT	0.69	0.91	0.80	0.82		
	WL	0.80	0.97	1.00	0.80		

*$p < .05$; **$p < .01$; [a] n = 12, 12, and 13 for I-CBT, MC-CBT, and WL respectively, except for HFD where for WL, n =12.; [b] Parametric ANOVA not conducted due severe violation of normality and homogeneity assumptions. *Note.* Means are based on four-week scores in each phase.

4.4.3.2 *Headache Index*

Table 4.8 shows that there was a significant main effect for phase ($F(1, 34) = 53.85$, $p < .01$) indicating that headache index scores decreased substantially over time (i.e. from pre-treatment to post-treatment). A significant Group x Phase interaction was also found ($F(2, 34) = 7.42$, $p < .05$), supporting the observed superiority of the two active treatment groups in comparison with waiting-list controls (WL).

Pairwise comparisons confirmed that there was no significant difference between I-CBT and MC-CBT at post-treatment ($t(22) = -0.71$, $p = .48$, 2-tailed) but that the WL group's mean headache index score was significantly higher than that of either the I-CBT group ($t(23) = -3.77$, $p < .017$) or the MC-CBT group ($t(23) = -2.65$, $p < .017$). Figure 4.3 gives a visual representation of the treatment effect and shows that headache index scores were reduced more substantially in the two active treatment groups from pre- to post-treatment while the drop for patients in the WL group was far less. Within-group *t*-tests confirmed that there was significant pre-post change within the I-CBT group ($t(11) = 7.04$, $p < .017$) and also the MC-CBT ($t(11) = 3.75$, $p < .017$) group but not within the WL group ($t(12) = 1.8$, $p > .017$).

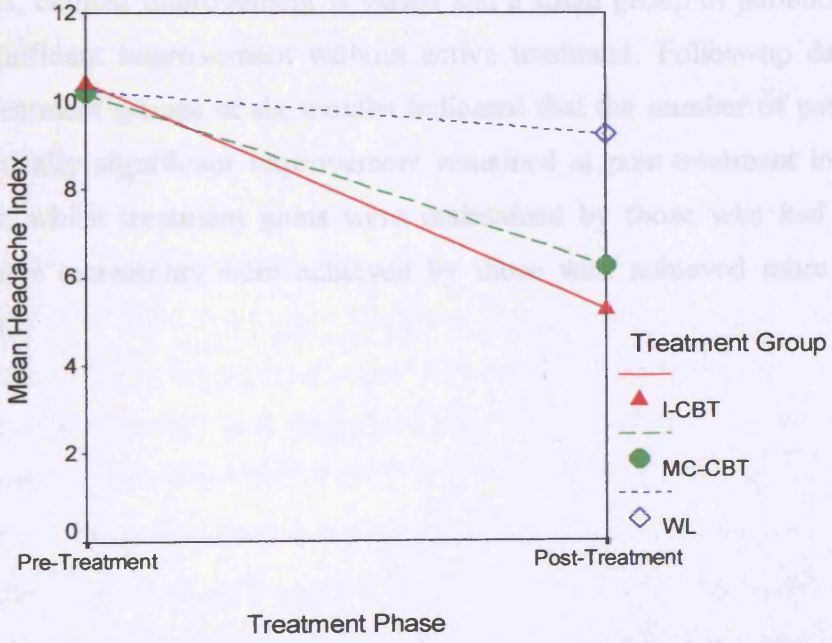


Figure 4.3 Treatment outcome on daily headache index. Higher headache index scores indicate worse headache activity. The graph shows that headache activity decreased more substantially in the two active treatment groups (I-CBT and MC-CBT). There does not appear to be a large difference in outcome between I-CBT and MC-CBT.

Data at 6-month follow-up for the I-CBT group and the MC-CBT group showed that the post-treatment outcome was maintained (I-CBT, n = 12, M = 4.93, SD = 3.24; MC-CBT, n = 10, M = 5.65, SD = 3.08). Within-group *t*-tests (post to follow-up, with alpha set at .01) were non-significant for both treatment groups.

4.4.3.3 Clinically Significant Change

As discussed previously, clinically significant change is a more stringent test of efficacy than the usual requirement for statistically significant differences. A reduction in the headache index of at least 50% is required before a patient can be classified as achieving clinically significant change. Figure 4.4 shows that the proportion of patients who achieved clinically significant change was greatest within the I-CBT group (58%) and least within the Waiting List group (23%). However, the observed between-group differences were not statistically significant (χ^2 (d.f. = 2, N = 37) = 3.227, $p > .05$). This Chi Square result itself needs to be treated with caution as two of the cells had expected frequencies below five. Nevertheless, the trend indicates that while CBT treatment was effective in achieving clinically significant change for many patients, clinical improvement is varied and a small group of patients achieved clinically significant improvement without active treatment. Follow-up data for the two active treatment groups at six months indicated that the number of patients who achieved clinically significant improvement remained at post-treatment levels. This suggests that, whilst treatment gains were maintained by those who had improved most, no major increments were achieved by those who achieved more moderate improvement.

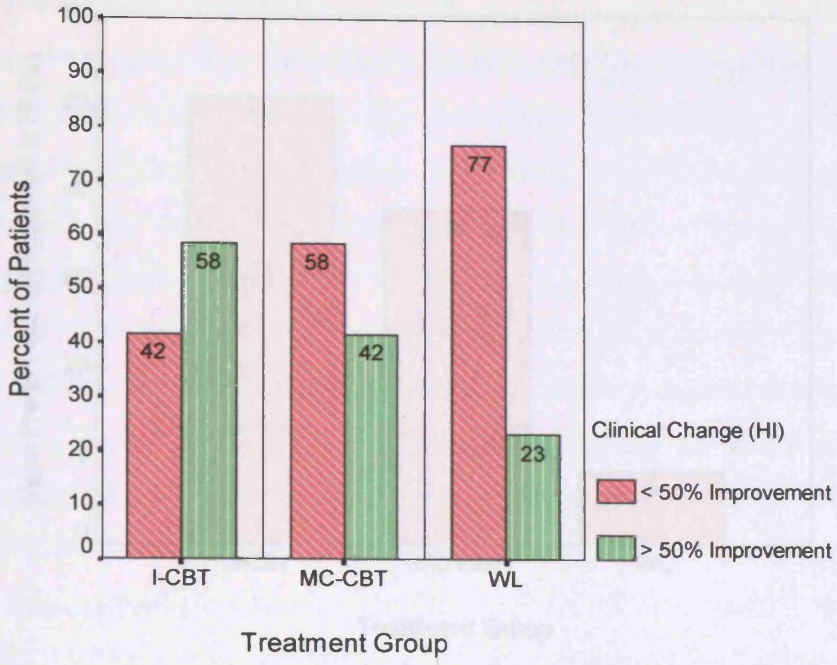


Figure 4.4 Clinically significant change on headache index. Percentage clinical change on the headache index is measured by dividing the pre to post treatment difference in HI by the pre-treatment score and multiplying by 100. The bar chart shows the per cent of patients who achieved clinically significant improvement on the headache index (i.e. greater than 50% decrease in HI) vs. the per cent of patients who failed to reach this cut-off, in each treatment group. The I-CBT group had biggest proportion of patients who achieved clinically significant improvement whilst the WL controls had the lowest proportion.

The actual mean pre-post change scores on the headache index were –5.14 (51%) for the I-CBT group, -3.92 (38%) for the MC-CBT group, and –1.00 (8%) for the WL group. These are shown in Figure 4.5.

A more accurate reflection of treatment effects can be shown by taking into account the distribution of change scores per treatment group, rather than the mean change scores. The box and whisker plots in Figure 4.6 show the distribution of change scores per treatment group together with their respective medians. While the overall range of scores is greater in the MC-CBT group, the median and the interquartile range is similar to the I-CBT group: the middle 50% of range of scores in these two groups is clearly superior to the equivalent range in the WL group. In fact, the graph shows that if a cut-off of at least –2 points change in the headache index is considered, then about 75% of patients achieved this in each of the two active treatment groups whilst only 25% of patients achieved this in the WL group.

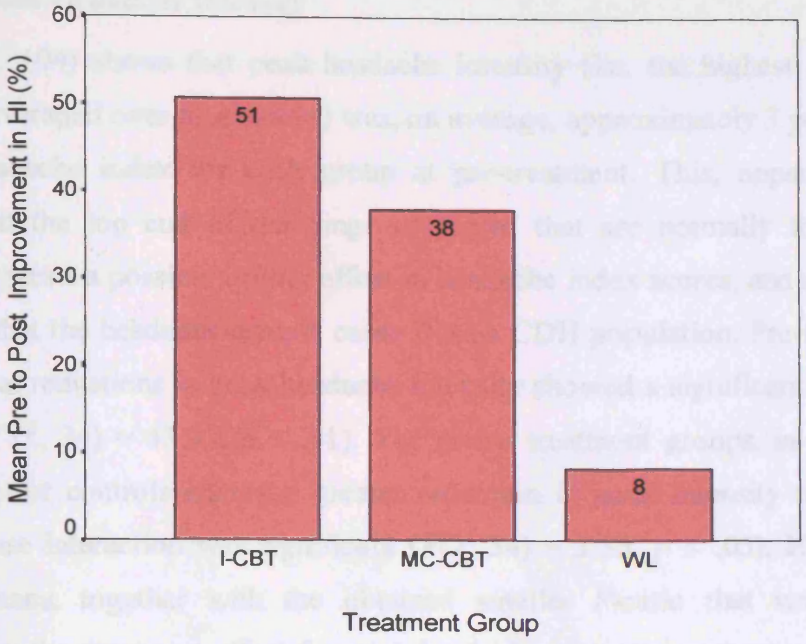


Figure 4.5 Mean improvement on headache index. The bar chart shows the mean pre-treatment to post-treatment headache index improvement (i.e. decrease in HI) per treatment group expressed as a percentage change from pre-treatment headache index scores.

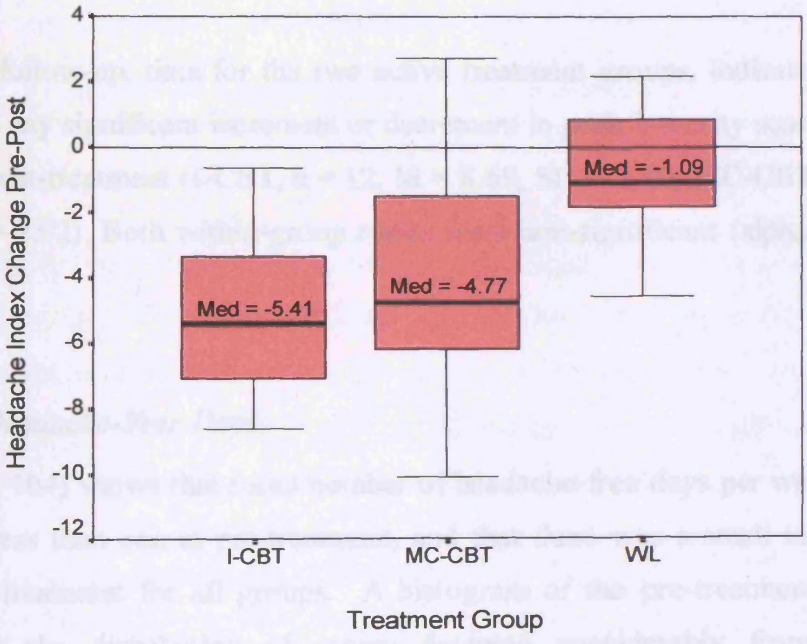


Figure 4.6 Distribution of headache index changes. The box and whisker plots show the range of headache index changes (based on the actual difference between pre-treatment and post-treatment HI scores) achieved by patients within each treatment group. Minus scores indicate reduction in headache activity from pre-treatment levels. Bigger reductions in HI were achieved by more I-CBT and MC-CBT patients compared to WL controls. Med = median; the boxes show the interquartile frequency range for change scores.

4.4.3.4 *Peak Headache Intensity*

Table 4.8 (p. 104) shows that peak headache intensity (i.e. the highest daily rating each week, averaged over four weeks) was, on average, approximately 3 points higher than the headache index for each group at pre-treatment. This, apparently small differential at the top end of the range of scores that are normally found in the literature, suggests a possible ceiling effect in headache index scores, and supports the assumption that the headache sample came from a CDH population. Pre-treatment to post-treatment reductions in peak headache intensity showed a significant main effect for phase (F (1, 34) = 43.93, $p < .01$). The active treatment groups, in comparison with waiting-list controls achieved greater reduction in peak intensity ratings. This Group x Phase interaction was significant (F(2, 34) = 3.55, $p < .05$). However, the observed means, together with the obtained smaller F-ratio that was obtained, suggested that the treatment effect for peak headache intensity was not as marked as that for mean headache index. This was confirmed through between-group pairwise comparisons on post-treatment scores, using independent t-tests; there were no statistically significant differences between any of the groups.

At 6-month follow-up, data for the two active treatment groups, indicated that there had not been any significant increment or decrement in peak intensity scores that were reported at post-treatment (I-CBT, n = 12, M = 8.69, SD = 2.60; MC-CBT, n = 10, M = 9.58, SD = 2.92). Both within-group t-tests were non-significant (alpha set at .025, two-tailed).

4.4.3.5 *Headache-Free Days*

Table 4.8 (p. 104) shows that mean number of headache-free days per week, for each group, was less than one at pre-treatment, and that there was a small increase from pre- to post-treatment for all groups. A histogram of the pre-treatment diary data showed that the distribution of scores deviated considerably from a normal distribution. This was confirmed through a Kolmogorov-Smirnov test, which showed a significant deviation from normality. Therefore, it was considered that a parametric ANOVA was inappropriate to test for treatment effects. Instead, a non-parametric one-way analysis of variance test (Kruskal-Wallis) was conducted on change scores between pre-treatment and post-treatment. No significant treatment effect was found

(χ^2 (d.f. = 2, N = 36) = 0.183, $p > .05$). Figure 4.7 shows that the percentage of headache-free days increased steadily for each treatment group but remained generally low (below 20% in the four-week post-treatment period).

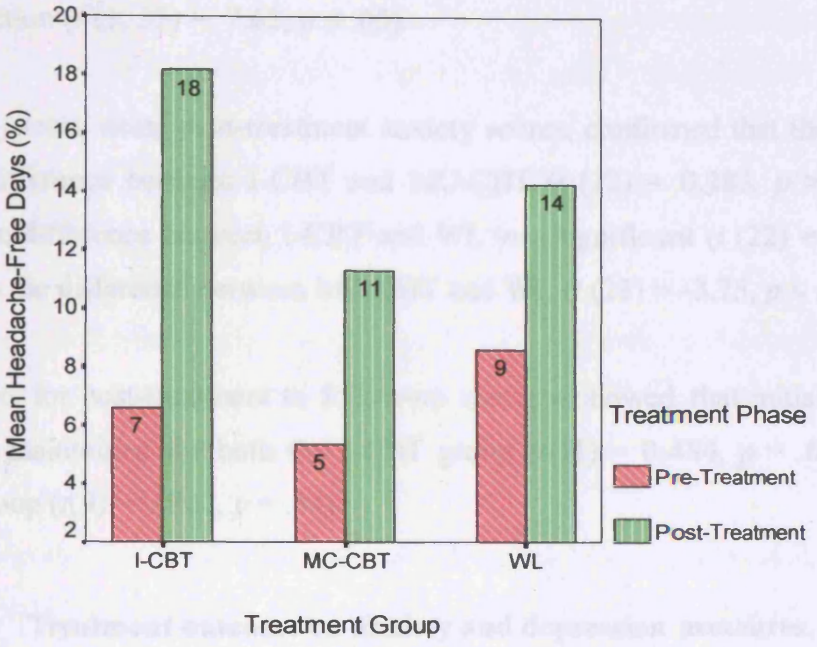


Figure 4.7 Per cent of headache-free days at pre- and post-treatment.

At 6-month follow-up, the average percentage of headache-free days went up only slightly in both the I-CBT group (to 20%) and the MC-CBT group (to 12%). This, together with the small magnitude of pre-treatment to post treatment changes suggests that increase in the number of headache-free days might be a more difficult target for CBT intervention compared with other outcome measures.

4.4.3.6 Anxiety and Depression Outcome

Anxiety scores for all groups were in the mild clinical range at pre-treatment and there was a significant decrease by post-treatment, suggesting a main effect for time ($F(1, 34) = 10.60$, $p < .05$). A look at the means in Table 4.9 shows that the two active treatment groups appeared to show greater improvement. However, the Group x Phase interaction, was not significant ($F(2, 34) = 2.11$, $p > .05$). This might be partly

explained by the fact that the WL group started off with more elevated anxiety scores than the other two groups. Though, this pre-treatment difference was non significant following a one-way ANOVA test ($F(2, 34) = 2.18, p = .129$), it was decided to carry out a 2-way analysis of co-variance (ANCOVA) with pre-treatment anxiety scores as the co-variate. The ANCOVA revealed that there was, in fact, a significant Group x Phase interaction ($F(1, 33) = 7.62, p < .05$).

Independent *t*-tests, using post-treatment anxiety scores, confirmed that there was no significant difference between I-CBT and MC-CBT (t (22) = 0.283, $p > .017$). As predicted, the difference between I-CBT and WL was significant (t (22) = -3.91, $p < .017$), as was the difference between MC-CBT and WL (t (23) = -3.75, $p < .017$).

Related *t*-tests for post-treatment to follow-up changes showed that initial treatment effects were maintained for both the I-CBT group ($t(11) = 0.484, p = .64$) and the MC-CBT group ($t(9) = 0.802, p = .44$).

Table 4.9 Treatment outcome on anxiety and depression measures.

Variable	Treatment Group[a]	Treatment Phase				F (d.f.)	
		Pre		Post			
		Mean	SD	Mean	SD	Phase	Group X Phase
HAD-A	I-CBT	9.50	3.73	6.67	2.39	10.60* (1, 34)	2.11 (2, 34)
	MC-CBT	9.00	4.07	6.33	3.31		
	WL	12.00	3.81	11.77	3.90		
HAD-D	I-CBT	3.75	2.30	2.58	1.56	13.67** (1, 34)	3.89* (2, 34)
	MC-CBT	6.75	2.96	3.75	2.42		
	WL	6.62	4.15	6.31	3.86		

* $p < .05$; ** $p < .01$

Note. HAD-A = Anxiety score and HAD-D = Depression score (HADS scale).

As can be seen in Table 4.9 depression scores were in the non-clinical range for all three groups at pre-treatment. However, there was a significant main effect for time (F (1, 34) = 13.67, $p < .01$) suggesting that there was still scope for there to be reductions in non-clinical levels of depression. A significant Group x Phase interaction was also found ($F(2,34) = 3.89$, $p < .05$), showing that the treatment group factor was important. Pairwise analysis of post-treatment depression scores showed that, as predicted, there was no significant difference between I-CBT and MC-CBT (t (22) = -1.40, $p > .017$). Contrary to predictions, the difference between I-CBT and WL, and that between MC-CBT and WL was also not significant in each case. This is partly explained by the 'floor-effect' in depression scores.

Related t-tests for within group changes from post-treatment to 6-month follow-up showed that there was no significant change in either the I-CBT group ($t(11) = 0.80$, $p = .44$) or the MC-CBT group ($t(9) = -0.16$, $p = .88$).

The overall results for anxiety and depression indicate that, whilst such symptoms were not excessive, particularly with regard to depression, CBT treatment (in either form) was superior to no treatment, and that MC-CBT was just as effective as I-CBT. Figure 4.8 shows the magnitude of changes in each treatment group. Whilst these changes are marginal, they may have some important implications concerning patients' confidence in being able to manage headaches; these implications are discussed later.

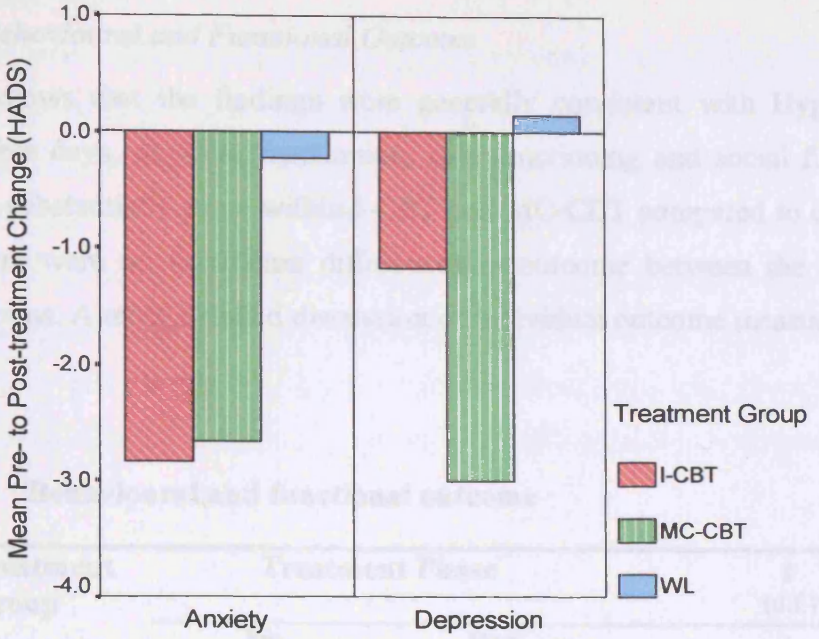


Figure 4.8 Treatment change on anxiety and depression. The bar chart shows the actual mean pre-treatment to post-treatment changes on the HAD scales per treatment group.

4.4.3.7 *Behavioural and Functional Outcome*

Table 4.10 shows that the findings were generally consistent with Hypothesis 1: medication-free days, physical functioning, role functioning and social functioning, all increased substantially more within I-CBT and MC-CBT compared to controls. In addition, there were no significant differences in outcome between the two active treatment groups. A more detailed discussion of individual outcome measures follows Table 4.10.

Table 4.10 Behavioural and functional outcome

Variable	Treatment Group	Treatment Phase				F (d.f.)	
		Pre		Post			
		Mean	SD	Mean	SD	Phase	Group X Phase
MFD (week)	I-CBT	2.42	2.90	3.79	2.65	(not applicable)[a]	
	MC-CBT	4.69	2.55	5.65	1.95		
	WL	4.00	2.55	4.38	2.57		
PF	I-CBT	49.33	18.77	70.92	10.20	15.50** (1, 34)	6.13** (2, 34)
	MC-CBT	50.58	18.17	64.50	17.27		
	WL	57.08	23.32	55.08	20.97		
RF	I-CBT	41.67	41.74	70.83	33.43	(not applicable)[a]	
	MC-CBT	41.67	35.89	66.67	32.57		
	WL	46.15	43.12	57.69	40.03		
SF	I-CBT	26.67	15.57	45.00	15.08	27.52** (1, 34)	4.48* (2, 34)
	MC-CBT	35.00	15.08	56.67	16.70		
	WL	40.00	25.82	43.08	25.62		

* $p < .05$; ** $p < .01$

[a]Parametric ANOVA not conducted due to severe violation of normality and homogeneity assumptions.

Note. MFD = Medication-Free Days, means are based on four week diaries at each treatment phase; PF = Physical Functioning, RF = Role Functioning, SF = Social Functioning

Medication-free days

Table 4.10 shows that medication-free days per week increased slightly for all treatment groups by post-treatment. A non-parametric analysis of variance test for pre to post change scores (Kruskal-Wallis) showed that the between-groups difference was not significant (χ^2 (d.f. = 2, N = 37) = 4.851, $p > .05$). Data for medication-free days needs to be treated with caution as most patients in each group were not on any medication at the start of treatment and/or had no intention of taking any. This is shown in Figure 4.9. Follow-up of the two active treatment groups at six months showed that the pattern of medication-free days was maintained in both groups.

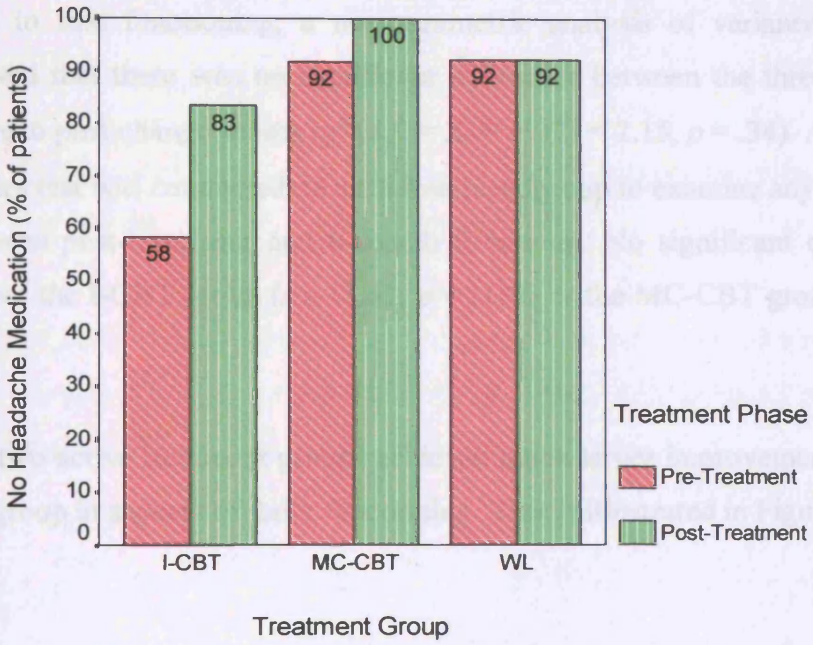


Figure 4.9 Per cent of patients on no headache medication

Physical, Role, and Social Functioning

With regard to general functioning, Table 4.10 (p.114) shows that the mean improvement on physical functioning and social functioning were particularly marked in the two active treatment groups. For physical functioning, there was a significant Group x Phase interaction ($F(2, 34) = 6.13$, $p < .01$) as well as a main effect for treatment phase ($F(1, 34) = 15.50$, $p < .05$). Pairwise t-tests confirmed that there was no significant difference in post-treatment scores between the I-CBT and MC-CBT groups ($t(22) = 1.11$, $p > .017$). As predicted, I-CBT patients were significantly more

improved than WL controls ($t(23) = 2.37, p < .017$). However, contrary to prediction, the difference between MC-CBT and WL was not significant ($t(23) = 1.22, p > .017$).

Table 4.10 shows that for social functioning, there was a significant main effect for phase of treatment ($F(1, 34) = 27.52, p < .01$) as well as a significant Group x Phase interaction ($F(2, 34) = 4.48, p < .05$). Pairwise comparisons of post-treatment scores supported the prediction of no significant difference between I-CBT and MC-CBT ($t(22) = -1.78, p = .28$). However, the predicted significant difference between WL and I-CBT was not found ($t(23) = 0.23, p > .017$), neither was that between WL and MC-CBT ($t(23) = 1.56, p > .017$).

With regard to role functioning, a non-parametric analysis of variance (Kruskal-Wallis) showed that there was no significant difference between the three treatment groups on pre to post change scores (χ^2 (d.f. = 2, N = 37) = 2.15, $p = .34$). A Wilcoxon paired samples test was conducted on each treatment group to examine any significant change between post-treatment and 6-month follow-up. No significant change was found in either the I-CBT group ($z = -1.41, p = .157$) or the MC-CBT group ($z = 0, p = 1$).

Overall, the two active treatment groups achieved much larger improvements than the waiting-list group in aspects of daily functioning. This is illustrated in Figure 4.10.

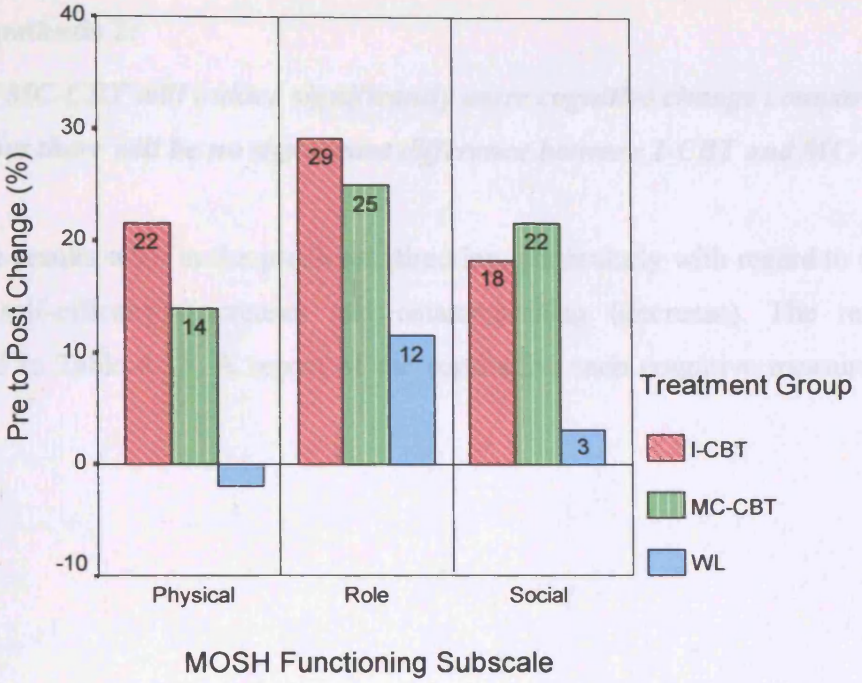


Figure 4.10 Treatment effects on functional outcome. Higher positive percentage change scores indicate greater improvement

For both I-CBT and MC-CBT, within-group change from post-treatment to follow-up was found to be non-significant (based on related *t*-tests with alpha set at p = .025) with regard to both physical functioning and social functioning.

4.4.4 Hypothesis 2:

I-CBT and MC-CBT will induce significantly more cognitive change compared to no-treatment but there will be no significant difference between I-CBT and MC-CBT.

Overall, the results were in the predicted direction, particularly with regard to change in perceived self-efficacy (increase) and catastrophising (decrease). The results are summarised in Table 4.11. A report of the results for each cognitive measure follows Table 4.11.

Table 4.11 Outcome on Cognitive Measures

Variable	Treatment Group	Treatment Phase				F (d.f.)	
		Pre		Post			
		Mean	SD	Mean	SD	Phase	Group X Phase
PSE	I-CBT	35.25	17.25	50.09	17.26	18.31** (1, 34)	5.64** (2, 34)
	MC-CBT	36.67	10.63	46.54	16.09		
	WL	30.58	13.66	30.26	13.72		
I-LOC	I-CBT	15.08	4.66	19.33	3.45	47.67** (1, 34)	5.79** (2, 34)
	MC-CBT	16.58	3.94	20.92	2.19		
	WL	16.23	3.98	17.23	4.19		
HP-LOC	I-CBT	12.75	3.49	14.58	2.19	5.52* (1, 34)	0.31 (2, 34)
	MC-CBT	15.25	4.65	16.00	4.77		
	WL	13.08	4.01	14.38	4.11		
C-LOC	I-CBT	16.17	5.13	10.25	2.53	28.08** (1, 34)	2.33 (2, 34)
	MC-CBT	14.50	4.36	10.08	4.38		
	WL	17.77	5.04	15.85	6.22		
CAT	I-CBT	29.00	7.59	17.50	2.65	34.42** 1, 34)	9.04** (2, 34)
	MC-CBT	24.17	6.31	16.58	5.09		
	WL	28.38	7.92	28.08	7.12		
COP	I-CBT	20.83	5.52	30.33	3.82	80.97** (1, 34)	8.40** (2, 34)
	MC-CBT	22.25	5.59	29.58	5.12		
	WL	19.85	6.32	22.38	7.87		

* $p < .05$; ** $p < .01$

Note. PSE = Perceived Self-Efficacy (HSES); I-LOC, HP-LOC, and C-LOC all refer to locus of control for headache: Internal, Health Professional, and Chance respectively (HSLCS); CAT = Catastrophising Self-Statements; COP = Coping Self-Statements (PRSS)

4.4.4.1 *Perceived Self-Efficacy*

Table 4.11 (p.119) shows that mean perceived self-efficacy increased far more in the two active treatment groups compared to WL controls. This Group x Phase interaction was highly significant ($F(1, 34) = 5.64$, $p < .01$). These large increases within I-CBT and MC-CBT not surprisingly, contributed to a significant main effect for phase ($F(1, 34) = 18.31$, $p < .01$). The treatment effect can be more clearly seen in Figure 4.11.

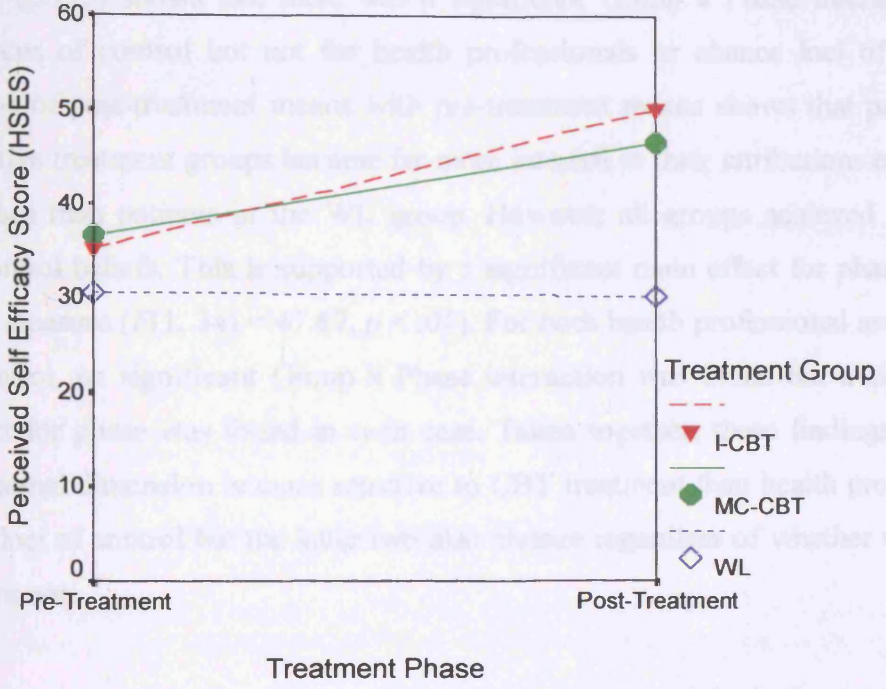


Figure 4.11 Change in perceived self-efficacy. Higher scores indicate a greater sense of self-efficacy. The graph shows that there is a very clear improvement in perceived self-efficacy within the two active treatment groups (I-CBT and MC-CBT) compared to no change in the control (WL) group.

Independent *t*-tests on post-treatment perceived self-efficacy scores supported the prediction of no significant difference between I-CBT and MC-CBT ($t(22) = 0.522$, $p = .61$, two-tailed). On the other hand, and as predicted, comparison with WL controls showed that each of the two active treatment groups achieved significantly greater perceived self-efficacy (for WL vs. I-CBT, $t(23) = 3.19$, $p < .017$; for WL vs. MC-CBT, $t(23) = 2.73$, $p < .017$).

At 6-month follow-up, the initial treatment gains were maintained in both the I-CBT group (n = 12, M = 53.20, SD = 14.91) and the MC-CBT group (n = 10, M = 45.84, SD = 11.64). This was confirmed through within-group *t*-tests which showed no significant decrease or increase in self-efficacy from post-treatment to 6-month follow-up.

4.4.4.2 Locus of Control

Table 4.11 (p.119) shows that there was a significant Group x Phase interaction for internal locus of control but not for health professionals or chance loci of control. Comparison of post-treatment means with pre-treatment means shows that patients in the two active treatment groups became far more internal in their attributions of control of headaches than patients in the WL group. However all groups achieved a rise in internal control beliefs. This is supported by a significant main effect for phase on the internality measure ($F(1, 34) = 47.67, p < .01$). For both health professional and chance loci of control, no significant Group x Phase interaction was found but a significant main effect for phase was found in each case. Taken together, these findings suggest that the internal dimension is more sensitive to CBT treatment than health professional or chance loci of control but the latter two also change regardless of whether treatment is offered or not.

Pairwise comparisons on post-treatment scores relating to each loci of control yielded mixed results. Firstly, for internal locus of control, there was no significant difference between I-CBT and MC-CBT as predicted. However, with regard to comparison with WL controls, the MC-CBT group, as predicted, were significantly more internal in their control beliefs ($t(23) = 2.72, p < .017$) but the I-CBT group were not significantly more internal.

With health professional locus of control, the difference between I-CBT and MC-CBT was non-significant, as predicted. However, the prediction that control beliefs in health professional will diminish significantly as a result of treatment was not borne out. In fact health professional locus of control increased slightly in each group. At post-treatment, as predicted, belief in chance or fate was significantly lower in each of the active treatment groups compared with WL controls (for I-CBT vs. WL, $t(23) = -2.90, p$

< .017; for MC-CBT vs. WL, $t(23) = -2.66$, $p < .017$) whilst there was no significant difference between the active treatment groups. These treatment effects can be more clearly seen in Figure 4.12.

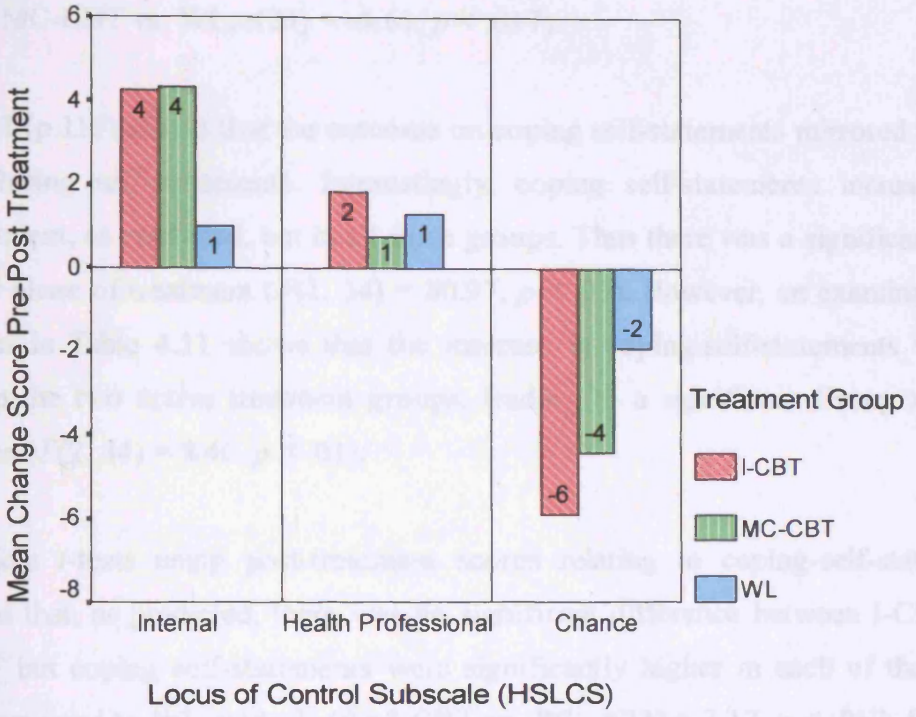


Figure 4.12 Change in locus of control beliefs. Greater improvement is indicated by negative change scores on the catastrophising scale and positive change scores on the coping scale.

At 6-month follow-up, no significant changes had taken place in the locus of control beliefs of I-CBT patients or MC-CBT patients (within-group *t*-tests on all measures were non-significant, with alpha set at .025).

4.4.4.3 Appraisal Statements

Table 4.11 (p.119) shows that, as predicted, catastrophising self-statements decreased significantly within the two active treatment groups but did not change significantly within the WL controls. Hence, a significant Group x Phase interaction was found ($F(2, 34) = 9.04$, $p < .01$). A significant main effect for phase of treatment was also found, but a glance at the means in Table 4.11 clearly shows that this was almost entirely due to decreases within the two active treatment groups.

Pairwise contrasts using post-treatment scores relating to degree of catastrophising self-statements confirmed that there was no significant difference between I-CBT and MC-CBT but that catastrophising self-statements were significantly lower in these two groups when compared with WL controls (for ICBT vs. WL, $t(23) = -4.84$, $p < .017$; for MC-CBT vs. WL, $t(23) = -4.61$, $p < .017$).

Table 4.11 (p.119) shows that the outcome on coping self-statements mirrored that for catastrophising self-statements. Interestingly, coping self-statements increased by post-treatment, as predicted, but in all three groups. Thus there was a significant main effect for phase of treatment ($F(1, 34) = 80.97$, $p < .01$). However, an examination of the means in Table 4.11 shows that the increase in coping-self-statements was far greater in the two active treatment groups, leading to a significant Group x Phase interaction ($F(2, 34) = 8.40$, $p < .01$).

Independent *t*-tests using post-treatment scores relating to coping-self-statements confirmed that, as predicted, there was no significant difference between I-CBT and MC-CBT but coping self-statements were significantly higher in each of these two groups compared to WL controls (for I-CBT vs. WL, $t(23) = 3.17$, $p < .017$; for MC-CBT vs. WL, $t(23) = 2.69$, $p < .017$).

The treatment effect with regard to catastrophising and coping self-statements can be more clearly seen in Figure 4.13. The treatment effect on catastrophising self-statements was more marked than that for coping-self-statements.

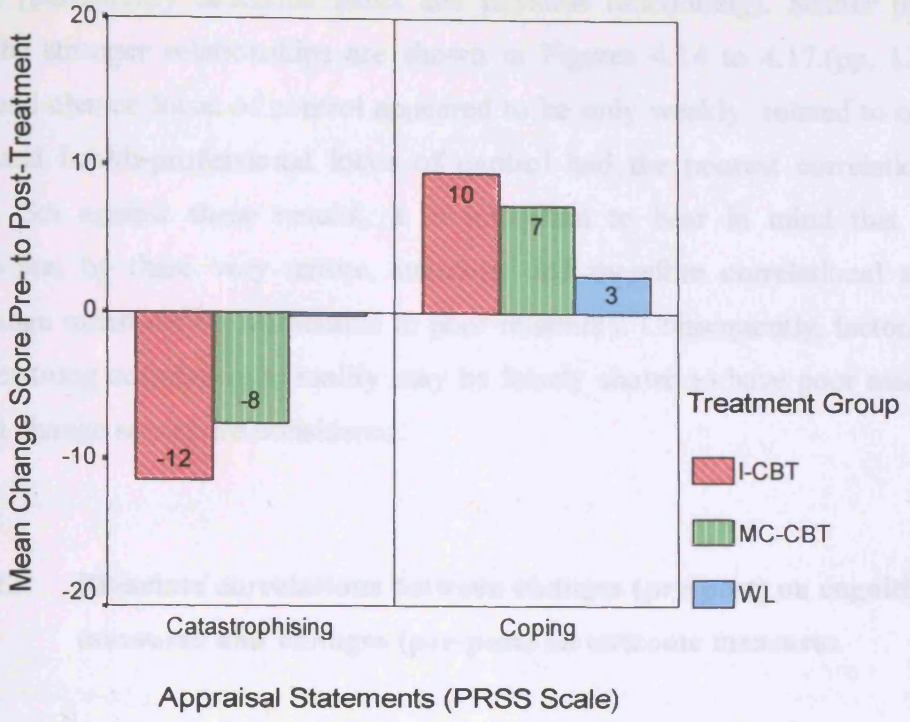


Figure 4.13 Change in appraisal self-statements

At 6-month follow-up, mean scores for both the I-CBT group (n = 12, Cat. M = 18.67, SD = 4.56; Cop. M = 28.25, SD = 3.19) and MC-CBT group (n = 10, Cat. M = 17.30, SD = 5.42; Cop. M = 29.00, SD = 4.52) showed that no significant changes had taken place in either catastrophising or coping self-statements suggesting that initial treatment gains were well maintained (using related *t*-tests with alpha set at $p = .025$).

4.4.5 Results for Hypothesis 3

There will be a significant association between cognitive changes and outcome changes. More adaptive cognitive changes will be associated with better outcome.

Table 4.12 shows that the findings concerning Hypothesis 3 were mixed. Firstly, the size of the correlations clearly indicate that a strong relationship between changes on specific cognitive measures and changes on outcome measures was not found. On the other hand, a number of moderately strong and significant correlations were found between changes on some cognitive measures (particularly, perceived self-efficacy and, catastrophising and coping self-statements) and changes on some outcome

measures (particularly headache index and physical functioning). Scatter plots for four of the stronger relationships are shown in Figures 4.14 to 4.17.(pp. 126-127) Internal and chance locus of control appeared to be only weakly related to outcome change, and health-professional locus of control had the poorest correlation with outcome. Set against these results, it is important to bear in mind that change measures are, by there very nature, unstable, and therefore correlational analyses using change measures are vulnerable to poor reliability. Consequently, factors which may have strong correlation in reality may be falsely shown to have poor association when just change scores are considered.

Table 4.12 Bivariate correlations between changes (pre-post) on cognitive measures and changes (pre-post) on outcome measures

	Cognitive Measures					
Outcome	**PSE**	**I-LOC**	**HP-LOC**	**C-LOC**	**CAT**	**COP**
HI	-.51**	-.20	.13	.24	.47**	-.43**
HAD-A	-.28*	-.33*	.08	.19	.52**	-.42**
HAD-D	-.22	-.32*	.03	.10	.29*	-.32*
PF	.57**	.34*	-.10	-.36*	-.56**	.53**
RF[a]	.44**	.24	-.14	-.43*	-.39**	.43**
SF	.33	.30*	-.04	-.12	-.49**	.41**

*$p < .05$; **$p < .01$ (both alpha levels are one-tailed);

[a]Spearman's rank correlations (r_s) computed as data for RF deviated severely from parametric test requirements

Note. PSE = perceived self-efficacy (HSES); I-LOC, HP-LOC, and C-LOC refer to internal, health professional, and chance locus of control, respectively (HSLCS); CAT and COP refer to catastrophising and coping self-statements respectively (PRSS); HI = headache index; HAD-A and HAD-D refer to anxiety and Depression respectively (HADS); PF, RF, and SF refer to physical, role, and social functioning respectively (MOSH).

Figure 4.14 The graph shows the extent to which change, from pre-treatment to post-treatment, in headache index (range 0 – 20) covaries with change in perceived self-efficacy scores (range: 0 – 100; scale: HSES)). As predicted, there appears to be an inverse relationship such that positive gains in perceived self-efficacy are associated with reductions in headache index scores ($r(37) = -.51$, $p < .01$). A best-fit regression line is shown for comparison.

Figure 4.15 The graph shows the extent to which change, from pre-treatment to post-treatment, in physical functioning (range: 0 –100; scale: MOSH) covaries with change in perceived self-efficacy (range: 0-100; scale HSES);. As predicted, there appears to be a positive relationship such that patients who achieved greater increases in physical functioning, also experienced greater increases in perceived self-efficacy ($r(37) = .57$, $p < .01$). A best-fit regression line has been plotted for comparison with the scatter plot.

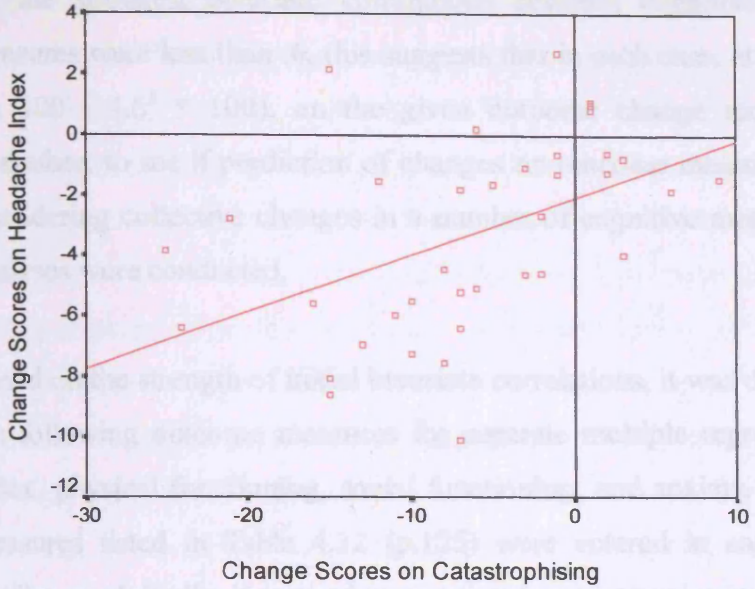


Figure 4.16 The graph shows the extent to which change, from pre-treatment to post-treatment, in the headache index (range: 0 – 20) covaries with change in catastrophising self-statements (range: 0 – 45; scale: PRSS). As predicted, there appears to be a positive relationship such that patients who achieved greater reductions in the headache index also achieved greater reductions in the strength of catastrophising self-statements ($r(37) = .47$, $p < .05$). A best-fit regression line has been plotted to allow comparison.

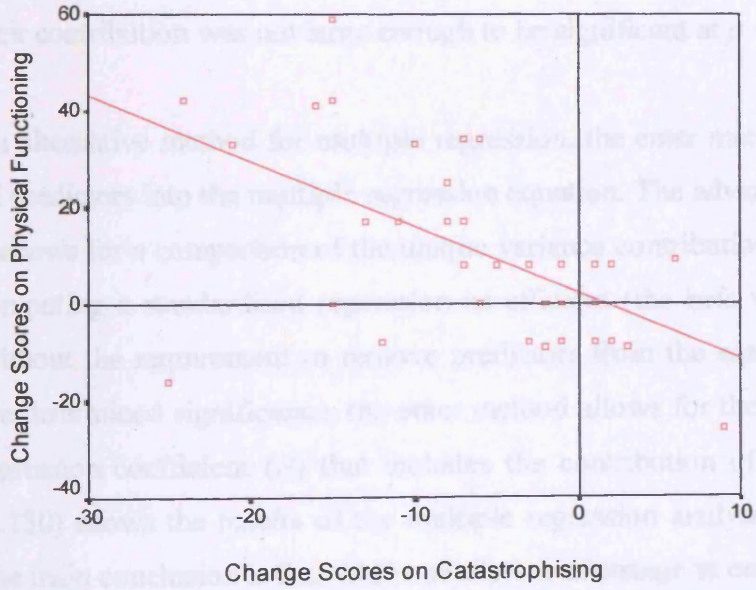


Figure 4.17 The graph shows the extent to which change, from pre-treatment to post-treatment, in physical functioning (range 0 – 100; scale MOSH) covaries with change in catastrophising self-statements (range: 0 – 45; scale: PRSS). As predicted, there appears to be an inverse relationship such that patients who achieved greater increases in physical functioning also achieved greater reductions in the strength of catastrophising self-statements ($r(37) = -.56$, $p < .01$). A best-fit regression line has been plotted to allow comparison.

As the strongest bivariate correlations between cognitive measures and outcome measures were less than .6, this suggests that in each case, at least 64% of the variance i.e. $100 - (.6^2 * 100)$, on the given outcome change score was unaccounted for. Therefore, to see if prediction of changes on outcome measures could be improved by considering collective changes in a number of cognitive measures, multiple regression analyses were conducted.

Based on the strength of initial bivariate correlations, it was decided to consider each of the following outcome measures for separate multiple regression analyses: headache index, physical functioning, social functioning, and anxiety. Changes on the cognitive measures listed in Table 4.12 (p.125) were entered in each multiple regression as predictors. Initially, it seemed appropriate to use stepwise multiple regression, so that a hierarchical analysis of the incremental variance contributed by each independent variable could be undertaken. However, the stepwise procedure for each dependent measure showed that no significant incremental variance was found beyond the zero-order correlations reported in Table 4.12. (alpha set at $p < .05$). That is, for each separate dependent measure, all independent variables, beyond the one which accounted for the greatest variance, were removed from the equation (or not entered) as their contribution was not large enough to be significant at $p < .05$.

An alternative method for multiple regression, the enter method, simultaneously enters all predictors into the multiple regression equation. The advantage of this method is that it allows for a comparison of the unique variance contribution of all predictors through computing a standardised regression co-efficient (the beta weight) for each predictor. Without the requirement to remove predictors from the equation if they do not reach pre-determined significance, the enter method allows for the computation of a multiple regression coefficient (R) that includes the contribution of all predictors. Table 4.13 (p.130) shows the results of the multiple regression analyses using the enter method. The main conclusion is that whilst prediction of change in outcome is enhanced through considering the cognitive predictors collectively, the increment from this is not huge. Therefore, other factors need to be taken into consideration.

Firstly, there may be some overlap between the variance contributions of different cognitive measures. Secondly, other variables apart from the ones considered here

appear to be important in mediating outcome change. A glance at the R^2 column shows that at least 50% of the variance on each outcome change measure is not explained by the cognitive change predictors considered here. The standardised regression coefficients (column headed β) indicate that for headache index change, perceived self-efficacy was the strongest predictor (though the beta weight only approached significance (t = -1.92, p = .064). For decrease in anxiety, decrease in catastrophising self-statements was the strongest predictor. From all the outcome measures, improvement in physical functioning was found to be most strongly associated with adaptive change on cognitive measures with 49% of the variance being accounted for. Of the cognitive change predictors, perceived self-efficacy contributed most to the variance. In the case of social functioning, catastrophising self-statements was found to be the strongest predictor.

Table 4.13 Multiple regression analyses for prediction of change in headache outcome from change on cognitive variables (N= 37)

Outcome Measure	Cognitive Predictors[a]	B	*SE* B	β	Pt	R	R^2
Headache Index	PSE	-0.09	0.05	-.35	-.33	.60	.36*
	CAT	0.103	0.10	.26	.19		
	COP	-0.15	0.13	-.24	-.20		
	C-LOC	-0.08	0.15	-.12	-.10		
	I-LOC	0.04	0.17	.04	.05		
	HP-LOC	-0.07	0.16	-.07	-.08		
HAD Anxiety	CAT	0.29	0.11	.63*	.44	.62	.38*
	C-LOC	-0.30	0.16	-.40	-.32		
	COP	-0.15	0.15	-.22	-.19		
	I-LOC	-0.23	0.19	-.19	-.21		
	PSE	0.02	0.05	.08	.09		
	HP-LOC	-0.03	0.18	-.03	-.03		
Physical Functioning	PSE	0.57	0.24	.37*	.39	.70	.49*
	COP	0.94	0.72	.25	.23		
	CAT	-0.60	0.51	-.25	-.21		
	HP-LOC	0.77	0.86	.13	.16		
	I-LOC	0.34	0.92	.06	.07		
	C-LOC	0.04	0.78	.01	.01		
Social Functioning	CAT	-1.13	0.53	-.50*	-.36	.59	.35*
	C-LOC	1.60	0.82	.43	.34		
	COP	1.07	0.75	.31	.25		
	I-LOC	0.83	1.00	.15	.16		
	HP-LOC	0.44	0.89	.08	.09		
	PSE	0.04	0.25	.03	.03		

*$p < .05$; [a]predictors listed in order of size of beta weight (β)

Note. PSE = perceived self-efficacy (HSES); I-LOC, HP-LOC, and C-LOC refer to internal, health professional, and chance locus of control, respectively (HSLCS); CAT and COP refer to catastrophising and coping self-statements respectively (PRSS).

B = unstandardised regression coefficient; *SE* B = standard error of B; β = standardised regression coefficient; Pt = partial correlation coefficient; *R* = Multiple regression coefficient

Overall, the tentative conclusion from the correlational analyses is that there is some association between cognitive change and outcome change in the predicted direction. That is, more adaptive cognitive change is associated with greater improvements in outcome. However, this conclusion must be seen as very tentative as the larger proportion of variance in outcome change for each dependent measure was

unaccounted for by the cognitive variables considered here. From the cognitive variables, perceived self-efficacy and appraisal statements (particularly, catastrophising) were most predictive of outcome change whilst locus of control did not seem to add very much to the predictive model. From the outcome variables, improvement in physical functioning was found to most strongly associated with adaptive cognitive change (particularly an increase in perceived self-efficacy). The small improvement in the regression prediction from a bivariate model to a multiple regression model suggests that there may be some overlap in the cognitive measures. On the other hand, the large differences in the beta weights and also in bivariate coefficients would appear to support the uniqueness of each cognitive variable.

4.4.6 Summary of Results

1. As predicted, comparisons with controls indicated that I-CBT and MC-CBT were both significantly effective on a range of outcome measures in the treatment of patients with CDH, and the treatment gains were well maintained at 6-month follow-up. The biggest improvements in outcome were observed in the headache index, anxiety, and physical functioning. Peak headache intensity rating seemed more resistant to change, as was the case with headache-free days. Depression levels were generally low and non-clinical to start with but small improvements were still achieved.
2. As predicted, both I-CBT and MC-CBT were significantly more effective than a no-treatment control condition, in bringing about adaptive cognitive changes. However, the strength of this treatment effect was variable with regard to the type of cognitive measure that that was considered. Perceived self-efficacy for headache management (increase), and catastrophising self-statements (decrease) were particularly improved in both treatment groups, whereas changes in health-professional locus of control and chance locus of control were not as marked. Changes in coping self-statements and strength of internal locus of control beliefs were intermediate.
3. As predicted, no significant differences were found between I-CBT and MC-CBT with regard to outcome changes or cognitive changes.

4. There was only tentative support for an association between the extent of cognitive change and degree of outcome change. The strength of the relationship between cognitive change and outcome change varied according to the specific cognitive and outcome measures that were paired within bivariate correlations. Overall, the correlations were strongest if they included either perceived self-efficacy or catastrophising from the cognitive measures and either the headache index or physical functioning from the outcome measures. The finding that even the largest correlations were below r =.6 prompted further analysis through multiple regression, to see if prediction of outcome changes could be improved by considering cognitive variables collectively. Only modest improvements in predictive power were obtained, and for each outcome measure, after partialling out the variance due to cognitive changes, at least 64% of the variance remained unaccounted for.

Chapter 5

Study 2: The Role of the Cognitive Component in Minimal-Contact Psychological Treatment for Chronic Daily Headache

5.1 Introduction

This study investigated the relative importance of giving patients explicit training in cognitive strategies within a minimal-contact CBT treatment package for headache. The efficacy of MC-CBT was supported in Study 1 and there was also evidence that cognitive changes were induced as a result of treatment. However, it was not clear whether treatment worked because it consisted of a well-structured and credible package that induced non-specific cognitive changes or whether there was a substantive and specific effect resulting from the inclusion of explicit cognitive coping strategies. It was felt that this could be tested by experimentally manipulating the presence or absence of just the explicit cognitive treatment component while leaving the rest of the minimal-contact treatment as identical. This was the focus of Study 2.

In the treatment condition in which the four-week block of treatment, dealing explicitly with cognitive strategies, was omitted, another treatment component was added instead. This replacement component was termed 'positive coping skills', and the aim of this was to induce cognitive change through non-specific means. This minimal-contact treatment, *without* an explicit cognitive treatment block and *with* the *positive coping skills*, was termed MC-PCS.

It was hypothesised that MC-CBT works primarily through explicit teaching of cognitive coping strategies rather than through non-specific treatment mechanisms. Therefore, it was predicted that while positive outcome and cognitive changes are possible in 'non-cognitive' treatments such as MC-PCS, the depth and long-term sustainability of such changes may require a more explicit cognitive based treatment such as MC-CBT.

5.2 Specific Hypotheses

Hypothesis 4:

MC-CBT and MC-PCS will be equally effective in terms of post-treatment outcome but MC-CBT will be significantly more effective in terms of longer-term (6-month) outcome.

Specifically it was predicted that, at 6-month follow-up MC-CBT patients will show significantly better outcome than MC-PCS patients, as indicated by the following positive changes on outcome measures.:

i) Less headache activity (lower headache index, lower peak headache intensity rating, more headache-free days).

ii) Lower anxiety and depression.

iii) Better behavioural and functional outcome (increases in: medication-free days, role functioning, physical functioning, and social functioning).

Hypothesis 5:

At post-treatment, both MC-CBT and MC-PCS will be equally capable of inducing cognitive changes but, over the longer-term (6 months), these changes will be significantly more well-maintained in MC-CBT patients than in MC-PCS patients.

Specifically, it was predicted that, at 6-month follow-up, MC-CBT will show significantly better cognitive change than MC-PCS patients as indicated by the positive changes on cognitive measures listed below:

a) Perceived self-efficacy (increased sense of perceived self-efficacy)

b) Locus of control
   - i) increased internal locus of control
   - ii) decreased health-professional locus of control
   - iii) decreased chance locus of control

c) Appraisal self-statements
   - i) decrease in catastrophising self-statements
   - ii) increase in coping self-statements

5.3 Method

5.3.1 Research Design

A 2 x 3 split-plot design was employed with one between-groups factor consisting of two conditions (MC-CBT and MC-PCS) and one repeated-measures factor consisting of three phases (pre-treatment, post-treatment, and follow-up). To minimise experimenter effects in delivering the two formats of treatment, a second therapist, in addition to the researcher, was recruited and trained in the treatment protocol (termed therapist A and therapist B from now on). Counterbalancing across therapists was achieved by randomly allocating patients from each treatment group to therapists A and B. Definitions for the independent and dependent variables are shown below and a summary of the main research design is presented in Table 5.1

Table 5.1 Summary of research design for Study 2

<table>
<tr><th colspan="2" rowspan="2">Independent Factor I (Treatment Group)</th><th colspan="3">Independent Factor II (Treatment Phase)</th></tr>
<tr><th>Pre-treatment</th><th>Post-treatment</th><th>Follow-up</th></tr>
<tr><td rowspan="2">MC-CBT</td><td>Therapist A
Therapist B</td><td rowspan="4" colspan="3">Dependent Variables:
1) Outcome
Headache Activity
Anxiety and Depression
Behavioural Outcome

2) Cognitive Change
Perceived Self-Efficacy
Locus of Control
Appraisal Strategies</td></tr>
<tr><td rowspan="2">MC-PCS</td><td>Therapist A
Therapist B</td></tr>
</table>

5.3.2 Participants

5.3.2.1 Inclusion Criteria

The inclusion criteria were exactly the same as those for Study 1. The reader is referred to page 77.

5.3.2.2 Number of Participants

Thirty-three consecutive patients with chronic headache were referred to Study 2 from a Neurology outpatients clinic. The referral outcome of these is summarised in Table 5.2 Thirty-one participants fulfilled the inclusion criteria, and of these, eleven people declined to take part. Therefore, the total sample size, who also completed treatment, was 20, representing 60.6% of the potential sample. All of these 20 participants stayed for the full duration of treatment and were also able to provide data at 6-month follow-up. Information about sample characteristics and comparison with non-responders as to the age and sex profile is presented in the Results section.

Table 5.2 Referral outcome for Study 2

Referral Outcome	Number of Patients	Percent
Completed treatment	20	60.6
Declined assessment	11	33.3
Unsuitable (after assessment)	2	6.1
Total	33	100.0

5.3.3 Procedure

The procedure was almost identical to that for Study 1:

1) Patients who attended the neurology clinic and who met all of the above inclusion criteria were verbally offered a referral to the study together with a brief explanation by the relevant doctor. If they were interested they were given an Information Leaflet about the study (see Appendix C-1).

2) The doctor (on confirmation with the relevant Consultant Neurologist) then made a formal written referral to the Medical Psychology Department, using a specially developed and easy to complete Referral Form (see Appendix D-1).

3) The names of referred patients were randomly assigned to the two treatment conditions: (1) MC-CBT; (2) MC-PCS. As in Study 1 It was felt that random assignment at the pre-assessment stage was important in minimising experimenter bias based on clinical assessment and, from an ethical viewpoint, in keeping the patients informed at assessment as to what sort of treatment would be offered.

4) Following this random allocation, all patients were offered an initial assessment appointment with the researcher (a trained clinical psychologist). If the patient failed to respond, a further appointment was offered. If a patient did not respond to the second appointment letter then no further contact was made and the referrer was informed.

5) Patients who attended the initial assessment session were asked to sign a Consent Form (see Appendix C-2). The assessment session consisted of a detailed semi-structured CBT interview lasting approximately one-hour (see Appendix E-1). This session was intended to be a two-way information gathering exercise to confirm patients' general suitability for CBT treatment, and to establish specific difficulties. The latter, were seen to be important in that individually tailored goals could be addressed within the standardised CBT treatment. As part of the baseline data collection, patients were asked to complete self-report questionnaires. They were also instructed to keep a daily Headache Diary (see Appendix E-2) for the next four weeks. This served as final confirmation of suitability for inclusion. Patients who were assessed as being unsuitable after return of baseline diaries i.e. who had less than 3 days with headache per week were offered a briefer intervention, which was not part of the study.

6) Patients assigned to the two treatment groups were instructed to bring their completed diaries and questionnaires to the first treatment session. They were given an appointment four weeks from the date of assessment.

7) Patients in the two treatment conditions completed 12 weeks of the relevant therapy followed by 4 weeks of post-treatment headache monitoring and

completion of post-treatment self-report questionnaires. A post-treatment review session was also conducted four weeks after treatment ended.

8) Follow-up data collection for the two treatment groups was initiated six months after treatment ended. Patients were contacted initially by telephone to confirm that they had maintained their consent to complete follow-up information. They were then asked to complete and return the self-report questionnaires and a four-week Headache Diary.

Table 5.3 Allocation to treatment groups and therapists

	Treatment Group	
	MC-CBT	**MC-PCS**
Therapist A	8	5
Therapist B	2	5
TOTAL[a]	10 (10)	10 (10)

[a]Figures in parentheses are number of participants for whom 6-month follow-up data was available

With regard to enhancing treatment integrity and consistency, experience from Study 1 suggested that regular (weekly) cross-checking and discussion between the two therapists, together with the use of specific written instructions and use of the treatment manual itself, were all very useful. Therefore, these procedures were maintained for Study 2.

All treatment was conducted on an outpatients basis within a Medical Psychology Department at a General Hospital. Usual clinical protocols were observed and assessment, progress, discharge, and follow-up reports were sent to referring consultants as well as the patient's General Practitioner. Immediate clinical and professional accountability was to the Head of the Medical Psychology Department.

5.3.4 Treatment

Minimal-Contact CBT (MC-CBT)

The MC-CBT treatment used here was exactly the same as that used in Study 1 and a more detailed description can be found in Section 4.3.4 (pp. 82-84).

In this format of CBT, treatment was offered primarily through a specially developed self-management treatment manual (Laher, 1995). The twelve-week treatment was split into three major blocks, each of four weeks duration: (1) Introduction, education, goal-setting and relaxation training; (2) Cognitive restructuring and attention-diversion training; (3) Stress management and pain behaviour.

Just three clinical treatment sessions were offered and these were scheduled to introduce each of the four-week treatment blocks. Patients were given each part of the treatment manual in stepwise fashion in accordance with the clinical introduction of each treatment block. Time was spent reviewing the previous block of treatment at each clinical session. A post-treatment review session was also held four weeks after the last block was introduced.

To encourage patient compliance, the treatment manual was structured to have clear, week by week instructions. No major problems, in following the instructions within the treatment manual, were reported by patients.

MC-PCS

This minimal-contact treatment did not include the four-week cognitive block of treatment. In all other respects, MC-PCS was identical to MC-CBT. A credible cognitive component was maintained by encouraging patients to build on any strategies that they found to be positive. No explicit cognitive coping strategies were taught. A summary of the treatment content of MC-CBT and MC-PCS is presented in Table 5.4.

Table 5.4 Treatment content and scheduling for Study 2

Treatment Block	Week	Minimal-Contact Treatment	
		MC-CBT	MC-PCS
Block 1	Week 1:	Understanding Headaches Setting Goals Breathing Retraining	Understanding Headaches Setting Goals Breathing Retraining
	Week 2:	Progressive Muscular Relaxation	Progressive Muscular Relaxation
	Week 3:	Autogenic Relaxation	Autogenic Relaxation
	Week 4:	Applying Shortened Relaxation	Applying Shortened Relaxation
Block 2	Week 5	Identifying Negative Thinking	Activity Pacing
	Week 6	Challenging Negative Thinking	Positive Coping Skills 1
	Week 7	Attention Diversion Training	Positive Coping Skills 2
	Week 8	Overall Management of Thoughts and Mood	Managing Moods
Block 3	Week 9	Understanding and Identifying Stress	Understanding and Identifying Stress
	Week 10	General Stress Management Plan	General Stress Management Plan
	Week 11	Exercise, Diet, and Sleep	Exercise, Diet, and Sleep
	Week 12	Changing Your Headache Behaviour and Communication Involving Family and Friends	Changing Your Headache Behaviour and Communication Involving Family and Friends
	Week 16	Post-treatment Review	Post-treatment Review

5.3.5 Measures

Measures were identical to those used in Study 1 and the reader is referred to the detailed review and scoring procedures covered in Section 4.3.5 (pp.85-98). In summary, the following measures were important:

Outcome Measures:

Headache Activity: headache index, peak headache intensity rating, and headache-free days per week.

Affect: anxiety and depression.

Behavioural and Functional Outcome: medication-free days, physical functioning, role functioning, and social functioning.

Cognitive Measures:

Perceived Self-Efficacy for headache management.

Locus of Control for headaches: internal, health professional, chance.

Appraisal Self-Statements related to headaches: catastrophising and coping.

Demographic profile measures from the structured clinical interview were again identical to those used in Study 1. In summary, data on the following was collected: sex, age, headache chronicity (years), general health (no other health problem vs. at least one other major health problem), ethnicity, marital status, and employment status.

5.4 Results

5.4.1 Overview of Analyses

As with Study 1, all analyses were carried out within SPSS Release 8 for Windows 95, and initial exploration of the data showed that parametric assumptions were met for all dependent variables except headache-free days, medication-free days, and role functioning. Non-parametric tests were therefore used for these three dependent measures. On the parametric tests of differences, the Levene's test was used to test for equality of variances. In most cases, variance within compared groups did not differ significantly. An adjusted test statistic was used where the Levene's test was significant.

The format of the results and the rationale for the different tests is very similar to that used for Study 1. Therefore, the reader is referred to Section 4.4.1 (pp. 99-100). The main differences within Study 2 were as follows:

(1) ANOVAS for treatment group x phase effects consisted of only two levels in the treatment group factor (MC-CBT and MC-PCS) but three levels on the phase factor (pre-treatment, post-treatment, and follow-up).

(2) Planned comparisons looked at the between-group difference on post-treatment scores (no significant difference predicted) and again on follow-up scores (MC-CBT outcome predicted to be significantly better than that of MC-PCS). For each variable, because two separate *t*-tests were conducted, the significance level was set at $p = .025$ (i.e. halving the conventional significance level of $p = .05$).

(3) The hypotheses for Study 2 did not necessitate any correlational analyses.

5.4.2 Sample characteristics

5.4.2.1 Demographic Profile

The demographic profile of the sample is shown in Table 5.5. The MC-CBT group was, on average, slightly older than the MC-PCS group by about nine years. Overall, the age range and mean ages were similar to those reported in Study 1. As expected, more females than males formed the sample and the ratio was almost two females for

every male. The between-group difference in proportion of males and females was not significant. Eighty-five per cent of the total sample were of White European ethnicity. Overall, the demographic profile was very similar to that of the Study 1 sample.

Table 5.5 Demographic profile of participants in Study 2

Demographic Variable		Treatment Group MC-CBT (n = 10)	Treatment Group MC-PCS (n = 10)	TOTAL SAMPLE (N = 20)
Age (years)	Mean	39.30	30.70	35.00
	SD	11.83	9.46	11.32
	Range	26 – 66	18 - 44	18 - 66
Sex	Males	3 (30%)	4 (40%)	7 (35%)
	Females	7 (70%)	6 (60%)	13 (65%)
Ethnicity	White European	9 (90%)	8 (80%)	17 (85%)
	Asian	1 (10%)	2 (20%)	3 (15%)
Marital Status	Single/ Divorced	2 (20%)	4 (40%)	6 (30%)
	Married/ Co-habiting	8 (80%)	6 (60%)	14 (70%)
Employment Status	Working	6 (60%)	8 (80%)	14 (70%)
	Studying/ training	0	0	0
	Retired	1 (10%)	0	1 (5%)
	Homemaker	3 (30%)	1 (10%)	4 (20%)
	Unemployed	0	1 (10%)	1 (5%)

5.4.2.2 Clinical Status

The participants in each treatment group had comparable clinical status at pre-treatment (see Table 5.6). Between-group tests for the quantitative variables (Mann-Whitney test for headache-free days, medication-free days and role functioning, *t*-tests for the remainder) confirmed that there were no significant differences ($p > .05$) between MC-CBT and MC-PCS at pre-treatment. A between-group Chi-Square test for 'other health problems' was also non-significant.

The high mean headache index for both groups, together with a mean of less than one headache-free day per week, confirmed that the sample came from a CDH population. Anxiety scores were in the moderate clinical range whilst depression was in the non-clinical range but slightly elevated. Scores for physical functioning and role

functioning indicated that there was moderate impairment in these areas but the biggest impact appeared to be on social functioning. Mean medication-free days was above three for both groups. Overall, the clinical status of the sample was very similar to that of the Study 1 sample.

As with Study 1, all patients experienced a mixture of tension-type and migrainous symptoms.

Table 5.6 Clinical status of participants in Study 2 at pre-treatment

Clinical Status		Treatment Group MC-CBT (n = 10)	Treatment Group MC-PCS (n = 10)	TOTAL SAMPLE (N=20)
Other Health Problems	None	5 (50%)	8 (80%)	13 (65%)
	Some	5 (50%)	2 (20 %)	7 (35%)
Headache Chronicity (years)	Mean	8.20	7.70	7.95
	SD	8.28	4.06	6.35
	Range	1 - 25	1 - 13	1 - 25
Headache Index[a]	Mean	9.28	10.10	9.69
	SD	0.88	1.17	1.09
Headache-Free Days (per week)[a]	Mean	0.48	0.25	0.36
	SD	0.98	0.53	0.77
Medication-Free Days (per week)[a]	Mean	3.78	3.23	3.50
	SD	2.71	2.69	2.65
Anxiety[b]	Mean	10.60	9.80	10.20
	SD	2.72	3.49	3.07
Depression[b]	Mean	5.90	4.30	5.10
	SD	2.23	2.67	2.53
Physical Functioning[c]	Mean	48.30	43.40	45.85
	SD	20.82	18.20	19.20
Role Functioning[c]	Mean	45.00	45.00	45.00
	SD	43.78	43.78	42.61
Social Functioning[c]	Mean	32.00	30.00	31.00
	SD	21.50	14.14	17.74

[a]Headache Diary; [b]Hospital Anxiety and Depression Scale; [c]Medical Outcomes Study Scale for Headaches.

5.4.3 Results for Hypothesis 4:

MC-CBT and MC-PCS will be equally effective in terms of post-treatment outcome but MC-CBT will be significantly more effective in terms of longer-term (6-month) outcome.

5.4.3.1 Headache Activity

Treatment effects on headache activity are shown in Table 5.7. Overall, the means indicate that both treatments were successful in bringing about some improvement by post-treatment. However, at six-month follow-up, the gains were maintained far better in the MC-CBT group whilst there was some relapse in the MC-PCS group. Generally, Hypothesis 4 was well-supported. The details for each variable are discussed below.

Table 5.7 Headache activity outcome

Variable	Group	Treatment Phase						F (d.f., 2, 17)	
		Pre		Post		Follow-up		Phase	Group x Phase
		Mean	SD	Mean	SD	Mean	SD		
Headache Index	MC-CBT	9.28	0.88	3.86	2.44	3.40	2.44	43.73**	7.13**
	MC-PCS	10.10	1.17	4.99	2.46	7.24	2.35		
Peak Headache Intensity	MC-CBT	12.10	0.59	6.80	3.16	6.90	3.17	30.97**	5.93*
	MC-PCS	12.48	0.96	8.03	2.42	10.88	2.79		
Headache -Free days (week)	MC-CBT	0.48	0.98	1.10	1.20	2.00	1.14	Not applicable	
	MC-PCS	0.25	0.53	0.73	1.05	0.50	0.71		

*$p < .05$; **$p < .01$

Headache Index

There was a significant main effect for phase ($F(2, 17) = 43.73$, $p < .01$), indicating that both treatment groups saw a decrease in headache index from pre-treatment levels. The overall improvement in headache appeared to be much greater in the MC-CBT group, and this was supported by a significant Group x Phase interaction. This interaction can be explained by the fact that, by the time of 6-month follow-up, headache index scores went up again in the MC-PCS group (though not to pre-treatment levels) whilst improvement was maintained and even built on slightly in the MC-CBT group. This effect can be clearly seen in Figure 5.1.

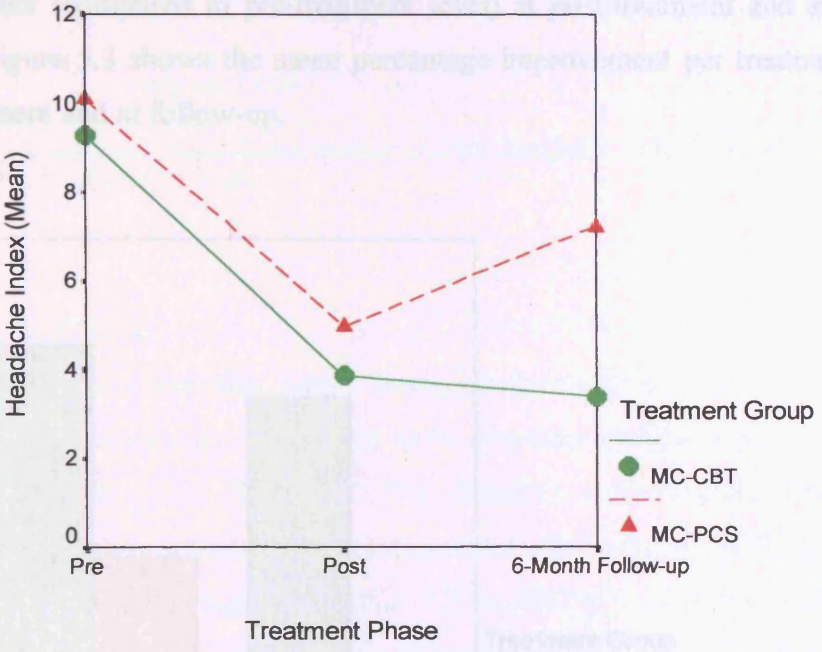


Figure 5.1 Between group changes on the headache index

Between-group *t*-tests at post-treatment and at follow-up showed that there was no significant difference in headache index scores at post-treatment but that MC-CBT patients had significantly lower headache index scores than MC-PCS patients at 6-month follow-up ($t(18) = -3.577$, $p < .025$). This finding supported hypothesis 4.

Clinically Significant Change

At post-treatment, 80% of the MC-CBT group achieved clinically significant headache improvement compared to 40% who did so within the MC-PCS group. This

difference approached statistical significance (χ^2 (d.f. = 1, N = 20) = 3.33, p = .068). At 6-month follow-up, there was a slight decline in the number who were still above the cut-off for clinically-significant improvement. However, the decline was different for the two groups. Within the MC-CBT group this number went down from 8 patients to 7 patients whilst in the MC-PCS group only one patient out of ten was above the cut-off for clinically significant improvement at follow-up. Thus at follow-up MC-CBT was significantly superior to MC-PCS in terms of the proportion of patients who remained clinically improved on the headache index (χ^2 (d.f. = 1, N = 20) = 7.50, $p < .05$). This is illustrated in Figures 5.2 and 5.3. Figure 5.2 shows the number of patients in each group who achieved at least 50% improvement in the headache index (compared to pre-treatment level) at post-treatment and at 6-month follow-up. Figure 5.3 shows the mean percentage improvement per treatment group, at post-treatment and at follow-up.

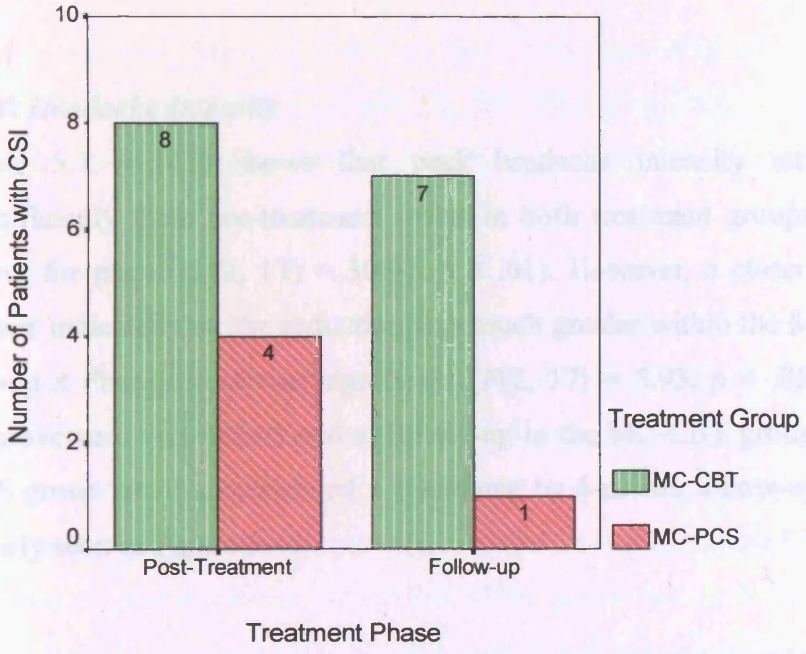


Figure 5.2 Clinically significant improvement on the headache index. Graph shows number of patients who achieved at least 50% reduction in the headache index from pre-treatment levels.

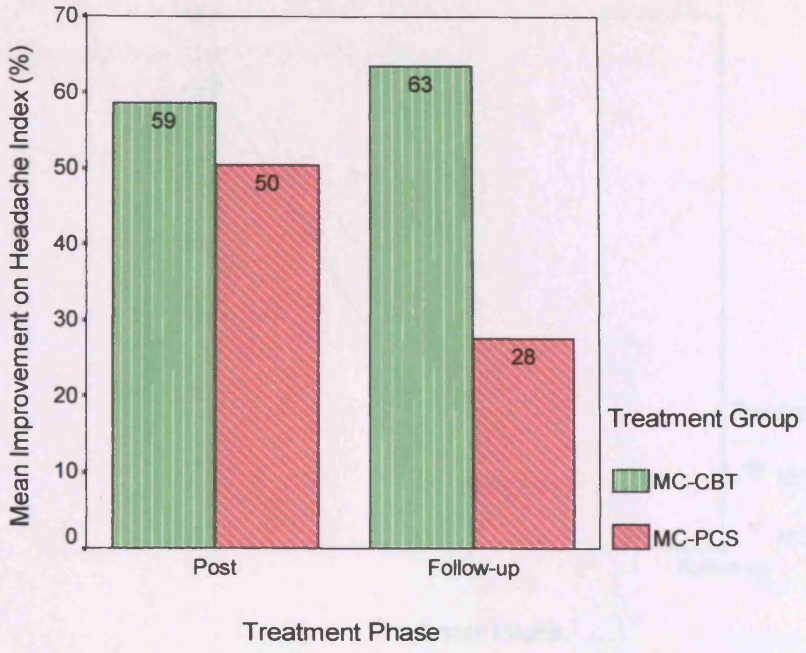


Figure 5.3 Mean percentage improvement on the headache index. Graph shows mean improvement from pre-treatment levels.

Peak Headache Intensity

Table 5.7 (p.145) shows that peak headache intensity ratings were reduced significantly from pre-treatment levels in both treatment groups, leading to a main effect for phase ($F(2, 17) = 30.97$, $p < .01$). However, a closer examination of the means indicates that the reduction was much greater within the MC-CBT group. This Group x Phase effect was significant ($F(2, 17) = 5.93$, $p < .05$). As predicted, the improvement was maintained at follow-up in the MC-CBT group but not in the MC-PCS group which experienced a worsening by 6-month follow-up. This effect can be clearly seen in Figure 5.4.

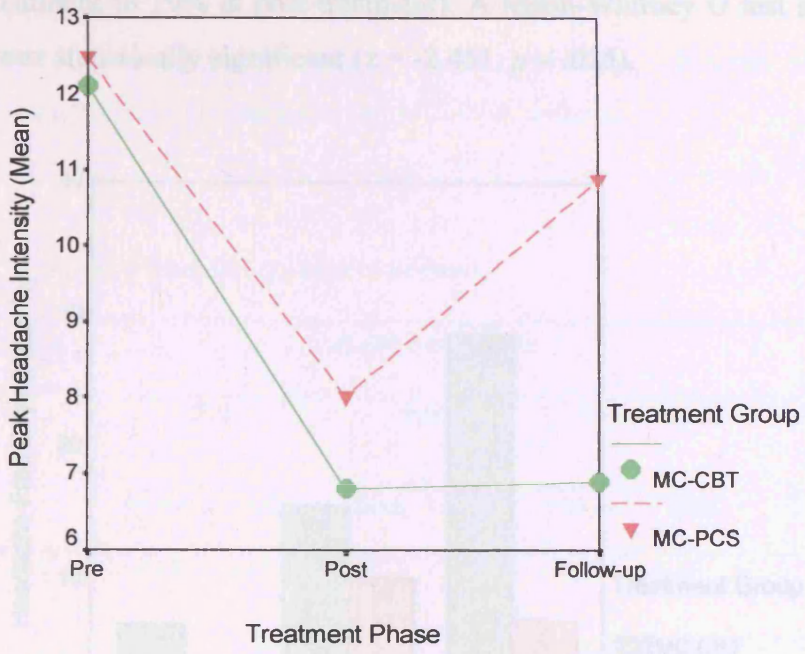


Figure 5.4 Change in peak headache intensity rating

A between-group *t*-test on post-treatment scores showed no significant difference, but at 6-month follow-up, as predicted, the MC-CBT group had a significantly lower peak headache intensity rating than the MC-PCS group ($t(18) = -2.97$, $p < .025$).

Headache-Free Days

Table 5.7 (p.145) shows that mean number of headache-free days at pre-treatment was less than 0.5 within each treatment group. As was found in Study 1, this measure seemed to be less sensitive to treatment than some of the other outcome variables. However, in the MC-CBT group, headache-free days per week went steadily up, to about one at post-treatment and to two days at 6-month follow-up. There was a small increase at post-treatment in the MC-PCS group, but at follow-up the amount of headache-free days was slightly down such that it was significantly lower than that for the MC-CBT group (Mann-Whitney U test, $z = -2.76$, $p < .01$). This pattern is reflected in Figure 5.5 which shows the mean percentage of headache-free days per treatment group at each phase of treatment. Change from pre-treatment to post-treatment was moderate but of a similar degree in each group. However, if pre-treatment to follow-up change is considered then the MC-CBT group clearly experienced a substantial mean increase in percentage of headache-free days (from

7% at pre-treatment to 29% at post-treatment). A Mann-Whitney U test showed that this change was statistically significant ($z = -2.451$, $p < .025$).

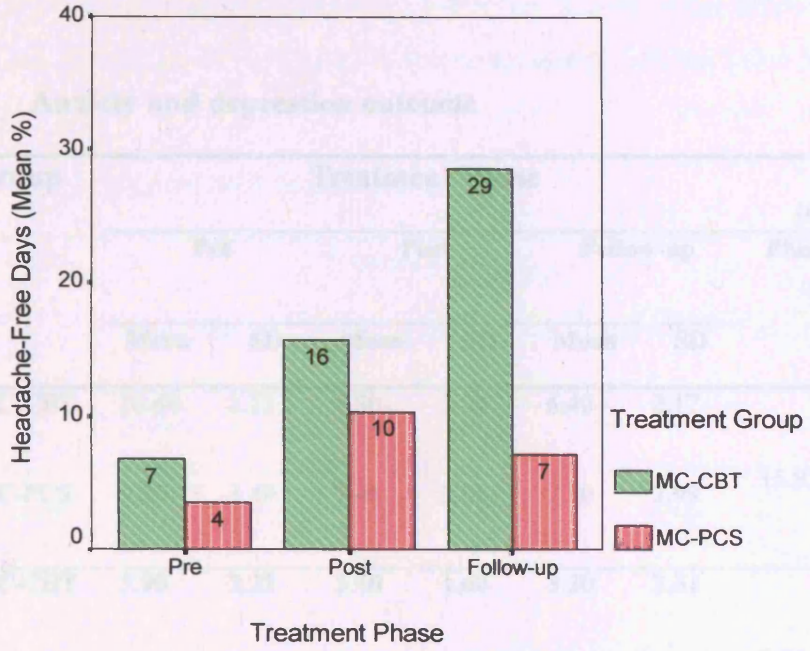


Figure 5.5 Percentage of headache-free days during each treatment phase

5.4.3.2 Affect

Table 5.8 shows that anxiety levels reduced significantly in both treatment groups, leading to a significant main affect for phase ($F(2, 17) = 15.91$, $p < .01$). However a Group x Phase interaction was not found. Independent *t*-tests on post-treatment scores and on follow-up scores confirmed that there was no significant difference in anxiety levels between MC-CBT and MC-PCS. Therefore, the prediction that MC-CBT would be superior at follow-up was not supported.

Depression levels were in the low range for both treatment groups at pre-treatment but the means at post-treatment and follow-up suggest that further scope for reduction was possible. However, the main affect for phase was not statistically significant ($p > .05$). An examination of the means showed that whilst depression scores reduced in both groups at post-treatment, at follow-up they went up again in the MC-PCS group but had reduced further in the MC-CBT group. This Group x Phase interaction was

found to be significant ($F(2, 17) = 9.68$, $p < .05$). At both post-treatment and follow-up, independent *t*-tests showed that there was no significant difference between the depression levels of MC-CBT patients and MC-PCS patients.

Table 5.8 Anxiety and depression outcome

Variable	Group	Treatment Phase						F (d.f. 2, 17)	
		Pre		Post		Follow-up		Phase	Group x Phase
		Mean	SD	Mean	SD	Mean	SD		
HADS-A	MC-CBT	10.60	2.72	8.30	3.20	6.40	2.17	15.91**	1.86
	MC-PCS	9.80	3.49	7.40	3.06	7.50	2.99		
HADS-D	MC-CBT	5.90	2.23	5.10	1.60	3.30	2.31	3.06	9.68*
	MC-PCS	4.30	2.67	3.00	1.56	4.60	2.84		

*$p < .05$; **$p < .01$

5.4.3.3 Behavioural and Functional Outcome

Table 5.9 summarises the findings with regard to behavioural and functional outcome. Overall, Hypothesis 4 was supported, in that there were substantial improvements within both treatment groups but gains were better maintained in the MC-CBT group. A more detailed discussion of individual outcome measures follows Table 5.9.

Table 5.9 Behavioural and functional outcome

Variable	Group	Treatment Phase						F (d.f. 2, 17)	
		Pre		Post		Follow-up		Phase	Group x Phase
		Mean	SD	Mean	SD	Mean	SD		
MFD	MC-CBT	3.77	2.71	5.20	2.14	5.33	2.00	(Not applicable)[a]	
	MC-PCS	3.23	2.69	4.48	2.81	3.83	2.34		
PF	MC-CBT	48.30	20.82	68.30	14.43	65.20	17.67	25.60**	0.508
	MC-PCS	43.40	18.20	62.50	15.18	55.80	15.21		
RF	MC-CBT	45.00	43.78	70.00	34.96	70.00	25.82	(Not applicable)[a]	
	MC-PCS	45.00	43.78	65.00	33.75	60.00	39.44		
SF	MC-CBT	32.00	21.50	50.00	19.44	56.00	18.38	8.63*	1.97
	MC-PCS	30.00	14.14	38.00	17.51	38.00	17.51		

*$p < .05$; **$p < .01$

[a]Parametric ANOVA not conducted due to severe violation of normality and homogeneity assumptions.

Note. MFD = Medication-Free Days, means are based on four week diaries at each treatment phase; PF = Physical Functioning, RF = Role Functioning, SF = Social Functioning (all from the MOSH scale).

Medication-Free Days

Table 5.9 shows that medication-free days per week started from a mean of around three days for each treatment group and increased by post-treatment to 5.20 days and 4.48 in the MC-CBT and MC-PCS groups respectively. However, at 6-month follow-up, the increase was maintained in the MC-CBT group but dropped back in the MC-PCS group. Nevertheless, non-parametric Friedman tests showed that overall improvement over the three treatment phases was significant within both treatment groups: for MC-CBT, χ^2 (d.f. = 2) = 10.89, $p < .01$; for MC-PCS, χ^2 (d.f. = 2) = 8.73, $p < .025$. Between-group differences at post-treatment and at follow-up were non-significant (using Mann-Whitney U tests, $p > .05$). Figure 5.6 shows the mean percentage of medication-free days per treatment phase for each group. As with the Study 1 sample, medication consumption was not excessive.

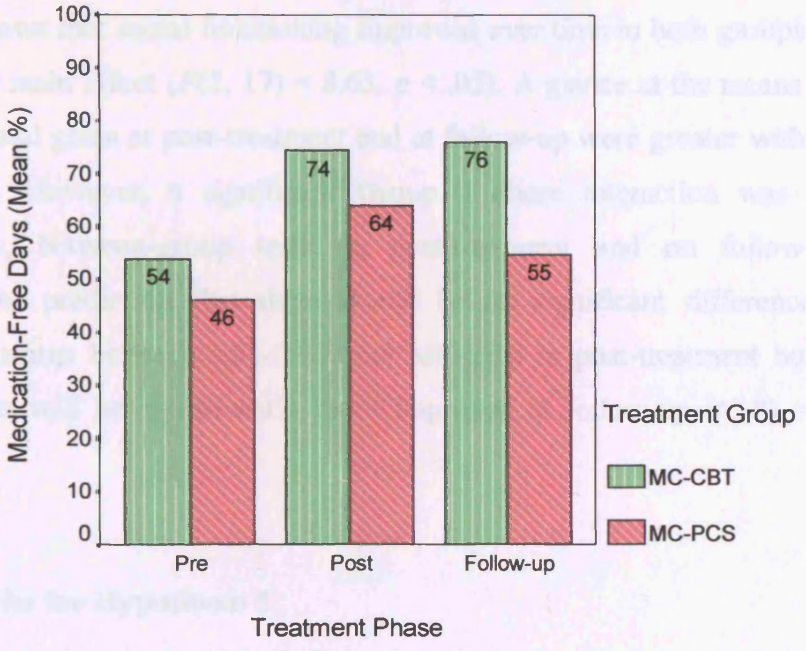


Figure 5.6 Percentage of medication-free days per treatment phase.

Physical Functioning

Table 5.9 shows that the level of physical functioning improved in both treatment groups from pre-treatment through to follow-up. This main effect for phase was significant ($F(2, 17) = 25.60$, $p < .05$). A Group x Phase interaction was not found. Between-group differences at post-treatment and at follow-up were non-significant.

Therefore, the prediction that MC-CBT would be superior to MC-PCS at follow-up was not supported.

Role Functioning

Table 5.9 shows that role functioning improved considerably in both treatment groups and that treatment gains were reasonably well maintained at 6-month follow-up. Friedman tests showed that within MC-CBT, this improvement was significant (χ^2 (d.f. = 2) = 8.33, $p < .025$) whilst the change was not significant within the MC-PCS group (χ^2 (d.f. =2) = 5.20, $p = .074$). Mann-Whitney U tests for between-group differences in role functioning at post-treatment and at follow-up were non significant ($p > .025$). Therefore, the prediction that MC-CBT would be significantly more effective in terms of follow-up status was only partially supported.

Social Functioning

Table 5.9 shows that social functioning improved over time in both groups, leading to a significant main effect ($F(2, 17) = 8.63, p < .05$). A glance at the means shows that the incremental gains at post-treatment and at follow-up were greater within the MC-CBT group. However, a significant Group x Phase interaction was not found. Nevertheless, between-group tests on post-treatment and on follow-up scores supported the prediction that there would be no significant difference in social functioning status between MC-CBT and MC-PCS at post-treatment but that MC-CBT patients will be significantly more improved at follow-up ($t(18) = 2.42, p < .025$).

5.4.4 Results for Hypothesis 5

At post-treatment, both MC-CBT and MC-PCS will be equally capable of inducing cognitive changes but, over the longer-term (6 months), these changes will be significantly more well-maintained in MC-CBT patients than in MC-PCS patients.

Table 5.10 summarises the treatment effects with regard to cognitive variables. Overall, Hypothesis 5 was supported, particularly on the measures of perceived self-

efficacy and catastrophising self-statements. A more detailed discussion of the findings for each variable follows Table 5.10.

Table 5.10 Treatment effects on cognitive variables

Variable	Group	Treatment Phase						F (d.f. 2, 17)	
		Pre		Post		Follow-up		Phase	Group x Phase
		Mean	SD	Mean	SD	Mean	SD		
PSE	MC-CBT	34.28	7.32	49.44	9.80	55.20	11.97	7.67*	3.27
	MC-PCS	41.03	10.80	46.70	11.80	46.32	9.26		
I-LOC	MC-CBT	16.60	3.27	20.60	1.65	22.0	1.89	16.32**	8.48*
	MC-PCS	18.40	2.88	20.50	1.90	19.00	2.54		
HP-LOC	MC-CBT	12.40	3.47	12.60	2.95	12.61	3.44	0.97	0.64
	MC-PCS	12.40	3.31	13.40	2.84	14.30	2.83		
C-LOC	MC-CBT	16.00	3.27	9.70	3.20	7.60	2.22	18.90**	0.99
	MC-PCS	15.70	5.38	10.60	4.20	10.70	2.71		
CAT	MC-CBT	30.40	6.69	18.70	4.06	18.00	3.56	36.10**	3.70*
	MC-PCS	26.70	6.00	20.40	4.81	23.20	3.99		
COP	MC-CBT	19.60	7.07	29.40	4.17	29.10	2.81	17.91**	2.30
	MC-PCS	21.40	7.28	27.30	4.16	23.70	3.16		

*$p < .05$; **$p < .01$

Note. PSE = Perceived Self-Efficacy; I-LOC, HP-LOC, and C-LOC all refer to locus of control for headache: Internal, Health Professional, and Chance respectively; CAT = Catastrophising Self-Statements; COP = Coping Self-Statements.

5.4.4.1 *Perceived Self-Efficacy*

Table 5.10 (p.155) shows that perceived self-efficacy improved within both treatment groups and that treatment gains were maintained at 6-month follow-up. This contributed to a significant main effect for phase ($F(2, 17) = 7.67$, $p < .05$). An examination of the mean scores clearly suggests that most of this main effect was due to greater improvements within the MC-CBT group. However, the Group x Phase interaction only approached significance ($F(2, 17) = 3.27$, $p = .063$). On the other hand, within-group repeated-measures ANOVAs confirmed that there was significant improvement within the MC-CBT group ($F(2, 8) = 10.28$, $p < .01$) but not within the MC-PCS group ($F(2, 8) = 0.86$, $p = .46$). Independent t-tests showed that, as predicted, there was no significant difference in level of perceived self-efficacy between the two groups at post-treatment but by follow-up the MC-CBT group had edged ahead with further gains. However, the between-group difference at follow-up was not significant. Figure 5.7 illustrates the finding that the MC-CBT group achieved a much greater improvement in perceived self-efficacy from pre-treatment levels and that perceived self-efficacy continued to increase after treatment had ended.

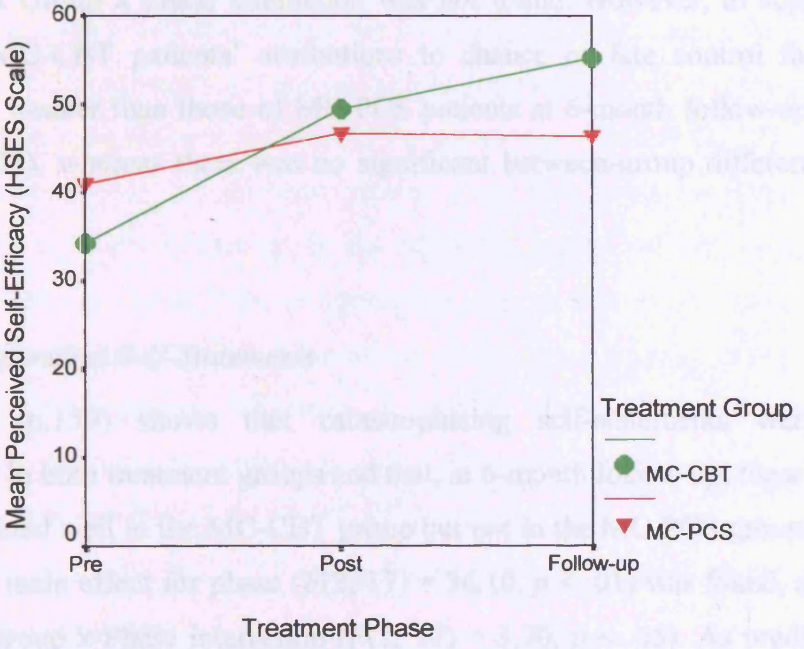


Figure 5.7 Changes in perceived self-efficacy for headaches across treatment phases. Higher scores indicate greater perceived self-efficacy.

5.4.4.2 *Locus of Control*

Table 5.10 (p.155) shows that patients in each treatment group, achieved greater internal locus of control for headaches, the main effect for phase being significant ($F(2, 17) = 16.32$, $p < .01$). A significant Group x Phase interaction was also found ($F(2, 17) = 8.48$, $p < .05$) and a look at the mean scores for internal locus of control indicates that treatment gains were greater within the MC-CBT group. As predicted, there was no significant difference in strength of internal locus of control beliefs between the two groups at post-treatment but the MC-CBT group had significantly higher internal locus of control scores at 6-month follow-up ($t(18) = 3.00$, $p < .01$).

With regard to health professionals locus of control beliefs, as found in Study 1, the strength of these beliefs was unaffected by treatment in both groups. No significant main effect or interaction was found (see Table 5.10).

Control beliefs in chance or fate decreased substantially in both treatment groups by post-treatment and this adaptive change was well maintained at 6-month follow-up. This was supported by a significant main effect for phase ($F(2, 17) = 18.90$, $p < .05$). A significant Group x Phase interaction was not found. However, in support of the prediction, MC-CBT patients' attributions to chance or fate control factors were significantly weaker than those of MC-PCS patients at 6-month follow-up ($t(18) = -2.80$, $p < .025$), whereas there was no significant between-group difference at post-treatment.

5.4.4.3 *Appraisal Self-Statements*

Table 5.10 (p.159) shows that catastrophising self-statements were reduced substantially in both treatment groups and that, at 6-month follow-up, these reductions were maintained well in the MC-CBT group but not in the MC-PCS group. Therefore a significant main effect for phase ($F(2, 17) = 36.10$, $p < .01$) was found, as well as a significant Group x Phase interaction ($F(2, 17) = 3.70$, $p < .05$). As predicted, there was no significant difference between the two groups at post-treatment, but at 6-month follow-up MC-CBT patients were significantly less catastrophic in their headache-related self-statements than MC-PCS patients ($t(18) = -3.07$, $p < .01$). Figure 5.8 illustrates the treatment effect for catastrophising self-statements.

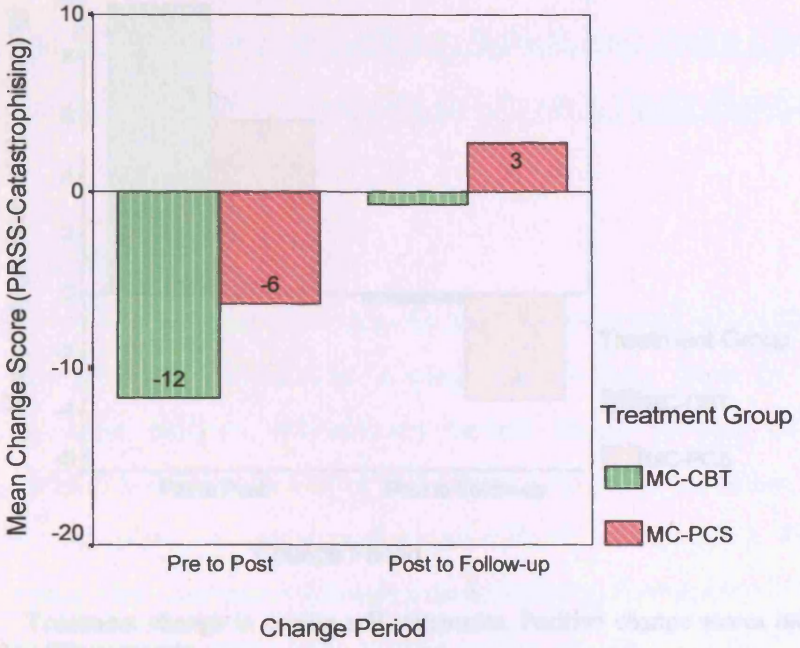


Figure 5.8 Treatment change in catastrophising self-statements. Negative change scores indicate less catastrophising.

With regard to coping self-statements, the treatment effect was again in the predicted direction, with the strength of coping beliefs increasing in both groups following treatment. Thus, a significant main effect for phase was found ($F(2, 17) = 17.91$, $p < .01$). The mean scores for different phases show that treatment gains were well maintained at 6-month follow-up in the MC-CBT group but not in the MC-PCS group. However, the Group x Phase interaction was not significant. Some support for an interaction was provided by independent *t*-tests which showed that there was no significant between-groups difference in coping self-statements scores at post-treatment but, at 6-month follow-up, MC-CBT patients' beliefs in coping were significantly stronger than those of MC-PCS patients ($t(18) = 4.04$, $p < .001$). Figure 5.9 shows the trend for MC-CBT being better at achieving positive treatment gains and also maintaining these.

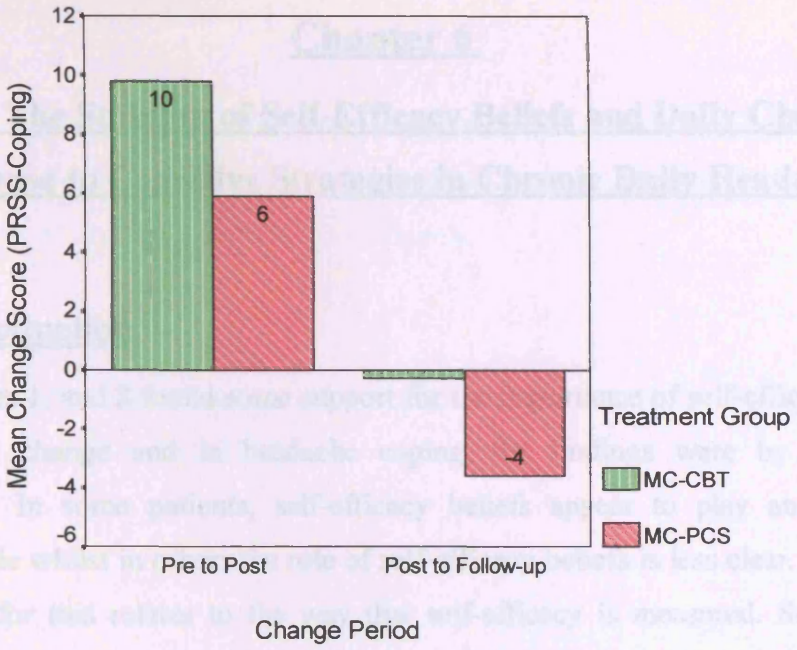


Figure 5.9 Treatment change in coping self-statements. Positive change scores indicate greater amount of coping self-statements.

5.4.5 Summary of Results

1. With regard to positive outcome changes, as predicted, both MC-CBT and MC-PCS were equally effective at post-treatment but at 6-month follow-up, the gains within the MC-CBT group were significantly better maintained. This treatment effect was more marked for headache index, anxiety, and the functional outcome measures. Headache-free days seemed more resistant to change whilst medication-free days were generally quite high to begin with so there was little scope for further change. Depression levels were in the non-clinical range from the outset but a trend similar to the major treatment effects was obtained.
2. In terms of adaptive cognitive changes, as predicted, both MC-CBT and MC-PCS were equally effective at post-treatment but MC-CBT was significantly more effective in maintaining treatment gains at 6-month follow-up. This treatment effect was particularly marked for perceived self-efficacy (increase), and catastrophising self-statements (decrease).

Chapter 6

Study 3: The Stability of Self-Efficacy Beliefs and Daily Change in Response to Cognitive Strategies in Chronic Daily Headache

6.1 Introduction

Whilst studies 1, and 2 found some support for the importance of self-efficacy beliefs in treatment change and in headache coping, the findings were by no means unequivocal. In some patients, self-efficacy beliefs appear to play an important mediating role whilst in others the role of self-efficacy beliefs is less clear. A possible explanation for this relates to the way that self-efficacy is measured. Self-efficacy beliefs have usually been measured through a questionnaire, the assumption being that such beliefs are reasonably stable over several weeks yet are sensitive to treatment-induced change. However, it is possible that standardised self-efficacy questionnaires are not sensitive to daily fluctuations in patients' beliefs. Such daily fluctuations may impact on headache coping and use of treatment strategies.

An interesting clinical observation that emerged from Studies 1 and 2 suggested that daily fluctuations in self-efficacy beliefs were common. A number of patients commented, after completing the Headache Self-Efficacy Scale (HSES), that in fact their 'confidence' in being able to do something, successfully, towards managing their headaches varied widely from one day to the next and sometimes even on the same day. In the context of the current research programme, two crucial questions follow from this. Firstly, to what extent do the daily fluctuations in self-efficacy correlate with fluctuations in headache activity? Secondly, is the explicit cognitive component within minimal-contact CBT treatment (MC-CBT) more effective than other treatment strategies in substantially improving daily self-efficacy beliefs?

6.2 Specific Hypotheses

Hypothesis 6:

There will be a significant inverse correlation between daily headache activity and a daily measure of self-efficacy in patients with CDH. Higher self-efficacy will be associated with lower headache activity.

Hypothesis 7:

Increase in daily self-efficacy will be greatest during the explicit cognitive treatment block within MC-CBT.

6.3 Method

6.3.1 Research Design

A single-case research design was employed for Study 3. This methodology is ideally suited to measurement of daily-level change while allowing for experimental manipulation of intervention components. To test the specific effects of the cognitive treatment block, without depriving patients of this component, required a treatment design whereby the order of presentation of the cognitive component could be varied vis-à-vis the other treatment blocks.

MC-CBT was presented as a step-wise build-up of three major four-week treatment blocks. In their original order, these were: 1) Relaxation training; 2) Cognitive restructuring and attention-diversion training; 3) Stress management training. Therefore, based on guidelines proposed by Barlow and Hersen (1984), six single-case experiments were designed so that all possible orders could be covered. This is shown in the matrix in Table 6.1.

Table 6.1 Research design for Study 3

Participant	Treatment Order[a]
Single-Case 1	A-B-BC-BCD
Single-Case 2	A-B-BD-BDC
Single-Case 3	A-C-CB-CBD
Single-Case 4	A-C-CD-CDB
Single-Case 5	A-D-DB-DBC
Single-Case 6	A-D-DC-DCB

A = Baseline Monitoring
B = Relaxation Training
C = Cognitive Restructuring and Attention Diversion Training
D = Stress Management Training

[a]MC-CBT treatment involved the continuation of strategies learnt in each previous treatment component. Therefore, a reversal design would not have been practicable due to carryover effects

6.3.2 Participants

Participants were six patients with CDH referred by a neurology clinic. Inclusion criteria were the same as those for Study 1 and Study 2 (see section 4.3.2., p. 77).

6.3.3 Measures

Almost all of the measures were identical to those used in Study 1, and the reader is referred to the detailed review and scoring procedures covered in Section 4.3.5 (pp. 85-98). In summary, the following measures were important:

Outcome Measures:

Headache Activity: headache index, peak headache intensity rating, and headache-free days per week.

Affect: anxiety and depression.

Behavioural and Functional Outcome: medication-free days, physical functioning, role functioning, and social functioning.

Cognitive Change Measures:

Daily Self-Efficacy (see below).

Perceived Self-Efficacy for headache management.

Locus of Control for headaches: internal, health professional, chance.

Appraisal Self-Statements related to headaches: catastrophising and coping.

6.3.3.1 Daily Self-Efficacy Ratings

A once per day rating for self-efficacy was incorporated on the Headache Diary. Participants were instructed to enter their daily self-efficacy ratings when entering the last of their four headache ratings (usually before bedtime). The ratings was based on the following question:

How confident do you feel in being able to apply the headache management strategies that you have so far been taught on this programme? Participants were asked to rate on a scale of 0 -10 (0 = "Not at all confident", 10 = "Completely confident").

6.3.3.2 Other Measures

Profile measures from the structured clinical interview were again identical to those used in Study 1. In summary, data on the following was collected: sex, age, headache chronicity (years), general health (no other health problem vs. at least one other major health problem), ethnicity, marital status, and employment status.

6.3.4 Procedure

The procedure was almost identical to that for Studies 1 and 2 (see section 4.3.3, p. 79) except it was not necessary to allocate to treatment groups. Each of the first six patients who met all of the inclusion criteria and who attended were randomly assigned to one of the six experimental treatment orders.

Patients completed 12 weeks of MC-CBT with the order of the three treatment blocks being varied for each patient, according to the planned experiments. There followed four weeks of post-treatment monitoring of headache activity, daily self-efficacy beliefs, and daily outcome-efficacy beliefs. A post-treatment review session was also conducted four weeks after treatment ended. Long-term follow-up was not planned.

All treatment was conducted by the researcher. Treatment was on an outpatients basis within a Medical Psychology Department at a General Hospital. Usual clinical protocols were observed and assessment, progress, discharge, and follow-up reports were sent to referring consultants as well as the patient's General Practitioner. Immediate clinical and professional accountability was to the Head of the Medical Psychology Department.

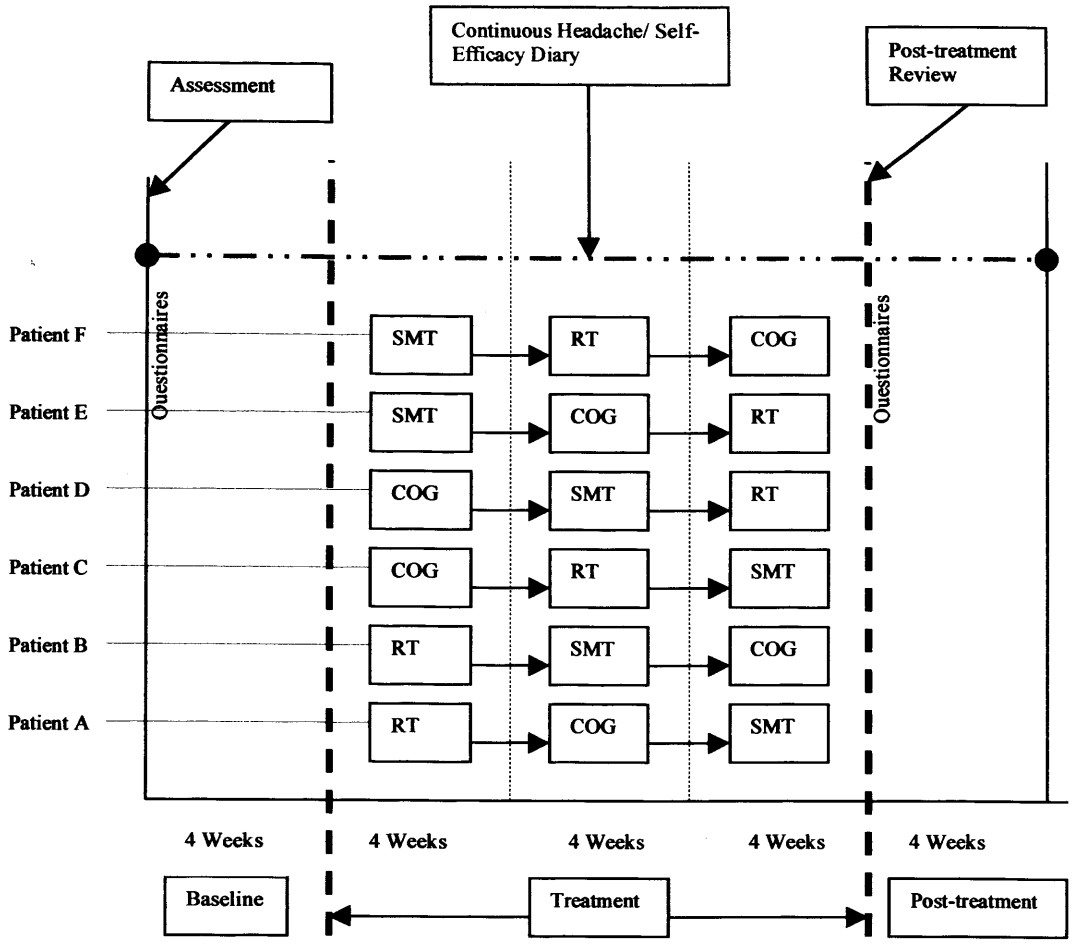


Figure 6.1 Summary of procedure for Study 3 RT = relaxation training treatment block; COG = cognitive treatment block; SMT = stress management treatment block.

Minimal-Contact CBT (MC-CBT)

The content of the MC-CBT treatment used here was exactly the same as that used in Study 1 and a more detailed description can be found in Section 4.3.4 (p. 82-84).

In this format of CBT, treatment was offered primarily through a specially developed self-management treatment manual. The twelve-week treatment was split into three major blocks, each of four weeks duration: (1) Introduction, education, goal-setting and relaxation training; (2) Cognitive restructuring and attention-diversion training; (3) Stress management and pain behaviour.

Just three clinical treatment sessions were offered and these were scheduled to introduce each of the four-week treatment blocks. Patients were given each part of the treatment manual in stepwise fashion in accordance with the clinical introduction of each treatment block. Time was spent reviewing the previous block of treatment at each clinical session. A post-treatment review session was also held four weeks after the last block was introduced.

The order of presenting the treatment blocks was varied across each of the six patients as shown in Figure 6.1. Regardless of the order of the components, the 'Introduction, education, and goal-setting' aspects were always presented at the beginning.

6.4 Results

6.4.1 Overview of Analyses

All data were analysed and graphed through the Microsoft Excel 97 spreadsheet package. As Study 3 was set up primarily as an exploratory investigation which might inform the current and future research programmes, extensive statistical analyses were not planned. Instead, it was felt more appropriate to rely mainly on graphical presentation of data so that the relationships of interest might be observed (Morley & Adams, 1991).

6.4.2 Sample characteristics

6.4.2.1 Demographic Profile

The demographic profile of the six participants is shown in Table 6.2. There were four females and two males. The mean age of the participants was 33.3 years. All participants were of White European ethnicity.

Table 6.2 Demographic profile

Patient	Sex	Age	Ethnicity	Marital Status	Employment Status
A	M	42	WE	Married	Employed
B	F	34	WE	Married	Employed
C	F	37	WE	Separated	Employed
D	F	22	WE	Single	Student
E	M	36	WE	Married	Employed
F	F	29	WE	Married	Homemaker

Note. WE = White European

6.4.2.2 Clinical Status

The clinical status of the six patients at pre-treatment is shown in Table 6.3. All patients reported headache chronicity of at least two years. Headache index scores were variable, with patients B, E, and F reporting far less headache activity than

patients A, C, and D. The headache index of the latter three patients more closely matched that reported by participants in Studies 1 and 2. As found in the previous two studies, anxiety scores tended to be in the moderate clinical range and were higher than depression scores. The MOSH subscale scores for all patients except patient C indicated that general functioning was not severely impaired. Patient C stood out as having the poorest clinical status overall.

Table 6.3 Clinical status

Patient	Headache chronicity (years)	Other health problems?	HI	PK	HFD (%)	HAD-A.	HAD-D	PF	RF	SF
A	5	No	10.36	13.25	0	9	5	42	50	60
B	6	Yes	3.21	6.75	32	8	9	50	50	80
C	8	Yes	12.57	16.25	0	14	14	16	0	20
D	3	No	9.82	11.75	0	12	7	58	50	60
E	4	No	5.89	8.75	18	13	4	42	0	80
F	2	No	5.11	10.50	32	5	3	33	100	40

Note. HI = mean daily headache index; PK = mean weekly peak headache intensity rating; HAD-A and HAD-D are anxiety and depression scores, respectively, on the Hospital Anxiety and Depression Scale; PF = physical functioning, RF = role functioning, SF = social functioning (all subscales from the Medical Outcomes Study Scale foe Headaches). None of the patients were on any headache medication.

6.4.3 Results for Hypothesis 6

There will be a significant inverse correlation between daily headache activity and a daily measure of self-efficacy in patients with CDH. Higher self-efficacy will be associated with lower headache activity.

6.4.3.1 Statistical Correlations

Hypothesis 6 was well supported in all patients except patient B whose relatively high pre-treatment daily self-efficacy rating seemed to leave little scope for further improvement. Table 6.4 shows two correlation coefficients (Pearson's) for each patient: (1) the correlation between mean daily headache index and mean daily self-efficacy; and (2) the correlation between the mean peak headache intensity rating and

mean daily self-efficacy. The overall conclusion is that there is a strong association between daily self-efficacy and daily headache activity.

Table 6.4 Correlations between daily self-efficacy and each of headache index and peak headache intensity rating.

Patient	Correlations (*r*)	
	HI and DSE	**PK and DSE**
A	-.85**	-.77**
B	-.22 (n.s.)	-.29 (n.s.)
C	-.77**	-.66**
D	-.90**	-.86**
E	-.84**	-.87**
F	-.74**	-.80**

**$p < .01$; n.s. = not significant ($p > .05$)

Note. HI = headache index; PK = peak headache intensity rating; DSE = daily self-efficacy rating. All correlations are based on the mean daily score for each of 16 weeks.

6.4.3.2 Graphical Analyses

Figures 6.2 to 6.7 (pp. 170-172) show the changes, across treatment, in the headache index (HI), with changes in daily self-efficacy (DSE) superimposed, for each patient. For each graph, RT = relaxation training block, COG = cognitive treatment block, and SMT = stress management training block. For both HI and DSE, each data point represents the mean from seven days. Therefore, as the four data points per treatment phase represented 28 days, it was felt that further trend analysis was not necessary, and the four points could be taken as a reasonably accurate representation of the trend within that particular treatment phase.

Overall, the single-case graphs show that as treatment progressed, headache activity came down, and daily self-efficacy increased. Headache activity seemed to be more sensitive to treatment change than daily self-efficacy, particularly following the introduction of the cognitive treatment block. The pre-treatment to post-treatment

improvements can be seen more clearly than the changes from one block to another consecutive block.

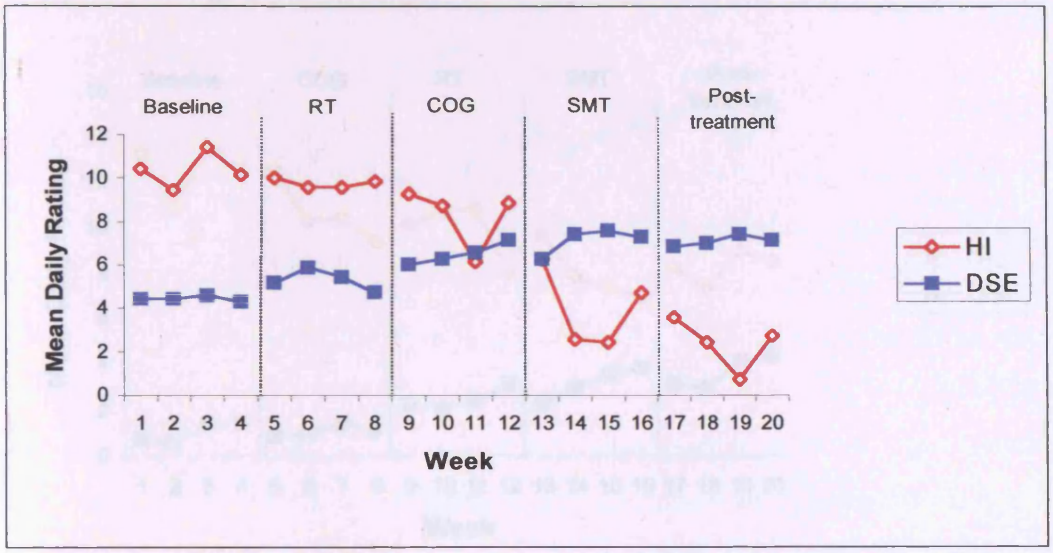


Figure 6.2 **Treatment changes for Patient A.** There was a small and steady rise in daily self-efficacy across the treatment phases. Headache activity showed a more dramatic fall after the introduction of the cognitive and SMT blocks, and treatment gains were maintained in the post-treatment phase.

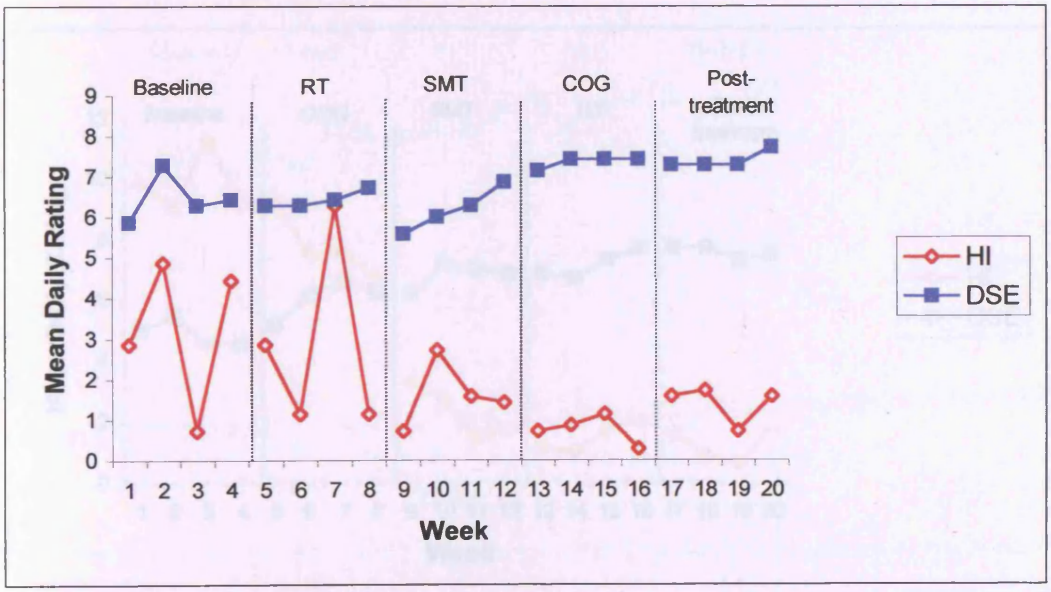


Figure 6.3 **Treatment changes for Patient B.** Daily self-efficacy ratings were high at pre-treatment and there was no substantive change across the treatment phases. Headache activity showed a more clear and stable drop in the latter part of the treatment programme, coinciding with the introduction of the SMT and cognitive blocks respectively.

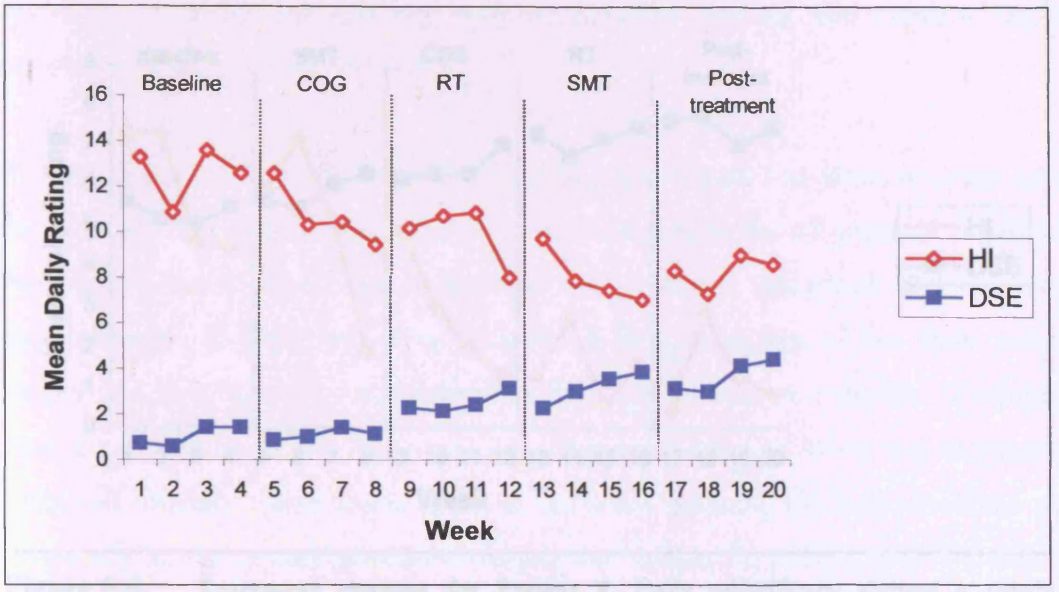


Figure 6.4 **Treatment changes for Patient C.** Daily self-efficacy was very low at baseline and showed a small but steady increase across the treatment programme. Similarly, headache activity was high at baseline but reduced gradually and moderately across the treatment phases.

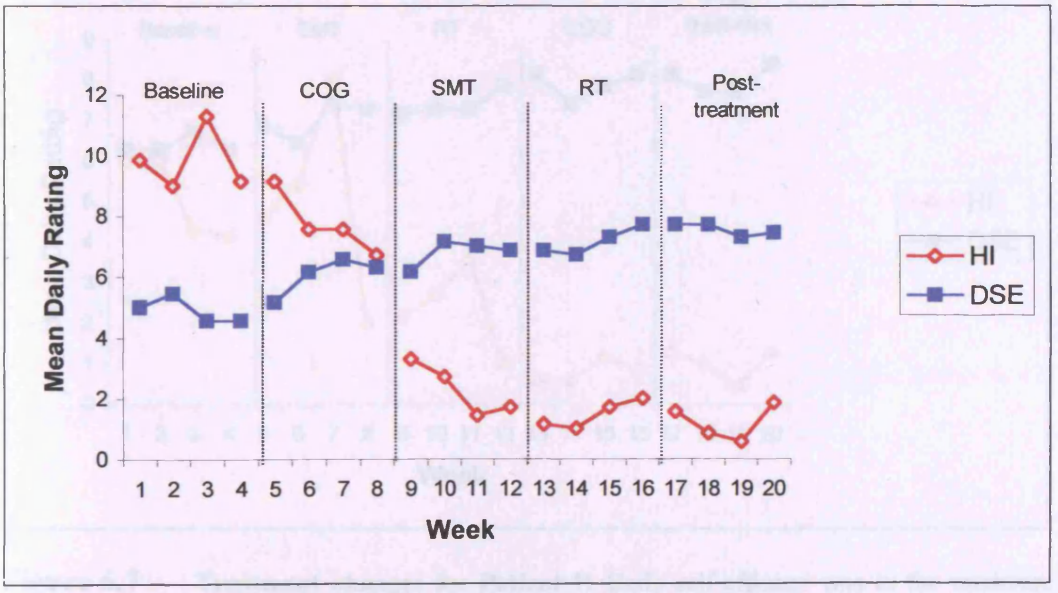


Figure 6.5 **Treatment changes for Patient D.** Daily self-efficacy was at a moderate level during baseline and showed a small and gradual increase over the course of treatment. Headache activity was high at baseline, but then showed a substantive fall during the introduction of the cognitive block followed by an even bigger drop in the latter part of the treatment programme.

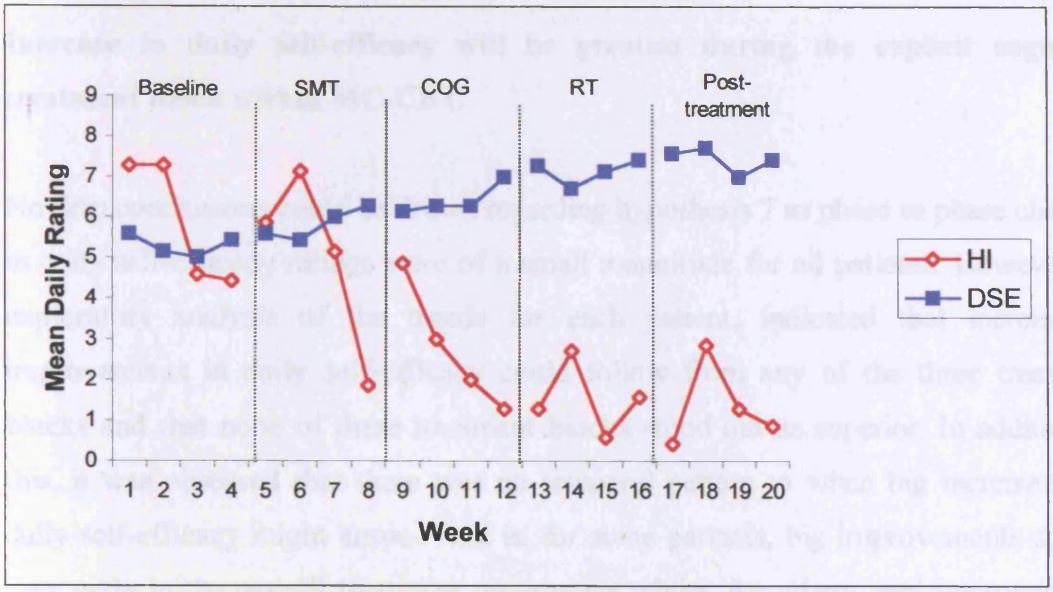


Figure 6.6 **Treatment changes for Patient E.** Daily self-efficacy showed a steady increase, towards the high range, across treatment after being in the moderate range at baseline. Headache activity was unstable during baseline and during the first treatment block but then showed a definite and substantive fall towards the end of the treatment programme.

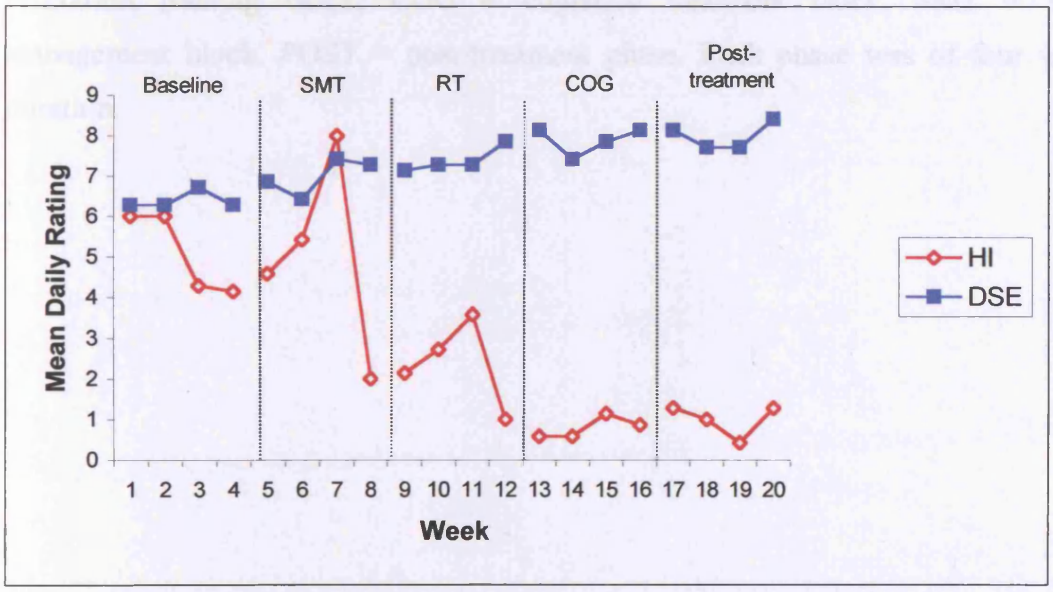


Figure 6.7 **Treatment changes for Patient F.** Daily self-efficacy was in the moderate range at baseline and gradually increased towards the high range across treatment. Headache activity was unstable at baseline and during the first treatment block but then showed a more clear and stable fall in the latter part of treatment, particularly, following the introduction of the cognitive block.

6.4.4 Results for Hypothesis 7

Increase in daily self-efficacy will be greatest during the explicit cognitive treatment block within MC-CBT.

No firm conclusions could be drawn regarding hypothesis 7 as phase to phase changes in daily self-efficacy ratings were of a small magnitude for all patients. However, an exploratory analysis of the trends for each patient, indicated that incremental improvements in daily self-efficacy could follow from any of the three treatment blocks and that none of these treatment blocks stood out as superior. In addition to this, it was observed that there was no temporal pattern to when big increments in daily self-efficacy might ensue. That is, for some patients, big improvements started very early in the overall treatment programme whilst, for others, real improvements came much later in the treatment programme.

Figures 6.8 to 6.13 (pp. 174-176) show the phase by phase incremental changes in daily self-efficacy for headache management for each patient. In each graph, RT = relaxation training block, COG = cognitive treatment block, SMT = stress management block, POST = post-treatment phase. Each phase was of four weeks duration.

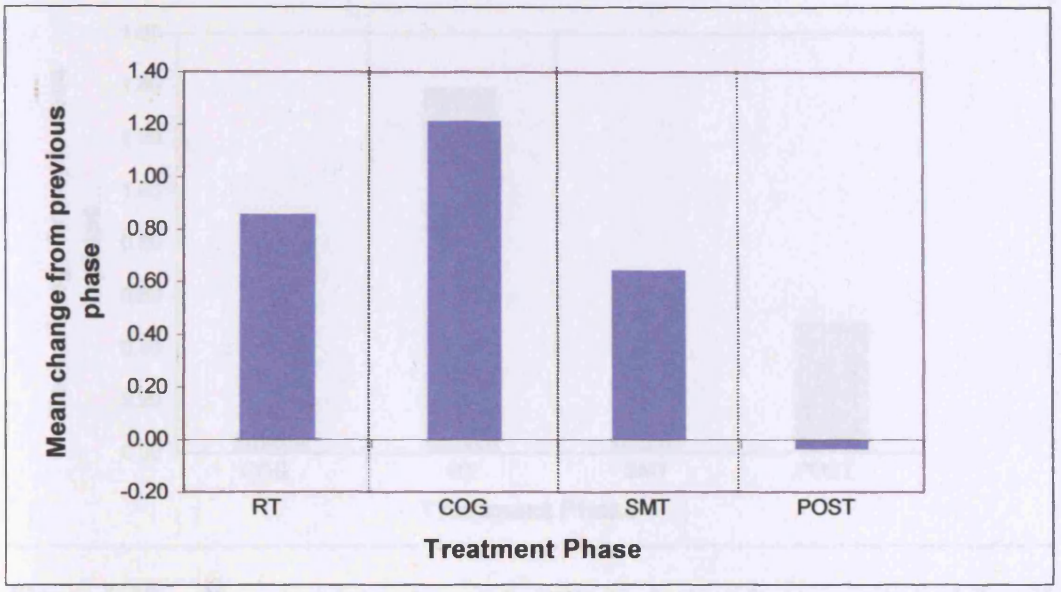


Figure 6.8 **Phase by phase changes in DSE for Patient A.** Each treatment block gave rise to an increment in mean daily self-efficacy for headache management. The cognitive block was associated with the biggest increment whilst in the post-treatment phase, there was no further increment and a slight decrease in daily self-efficacy.

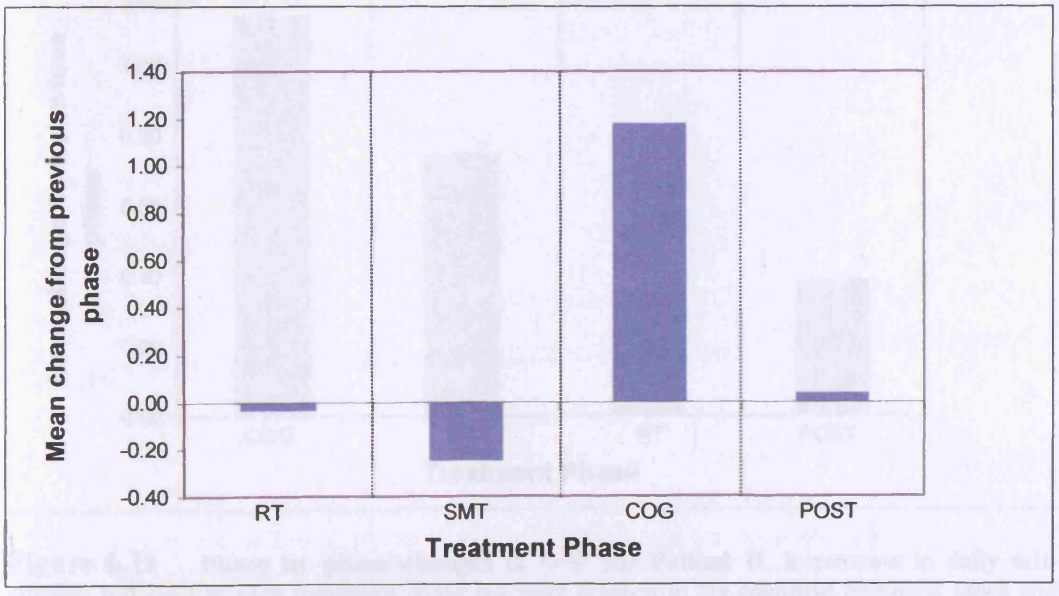


Figure 6.9 **Phase by phase changes in DSE for Patient B.** There was a very clear increment in daily self-efficacy during the cognitive treatment block but in SMT and RT there were slight decreases in daily self-efficacy.

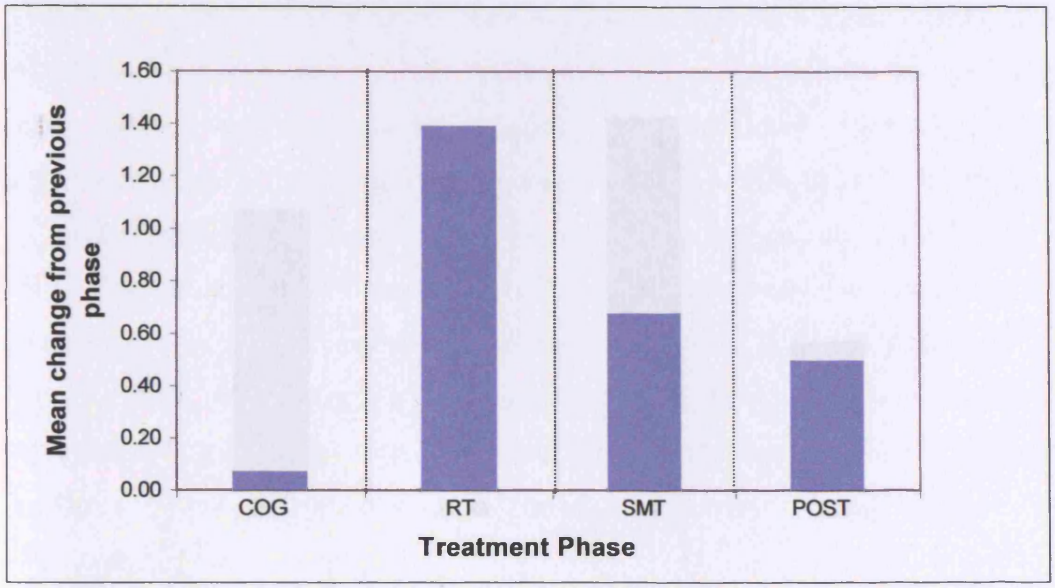


Figure 6.10 **Phase by phase changes in DSE for Patient C.** Increments in daily self-efficacy followed in each treatment phase but were greatest in the RT block and least in the cognitive block.. Some gains continued after treatment had ended.

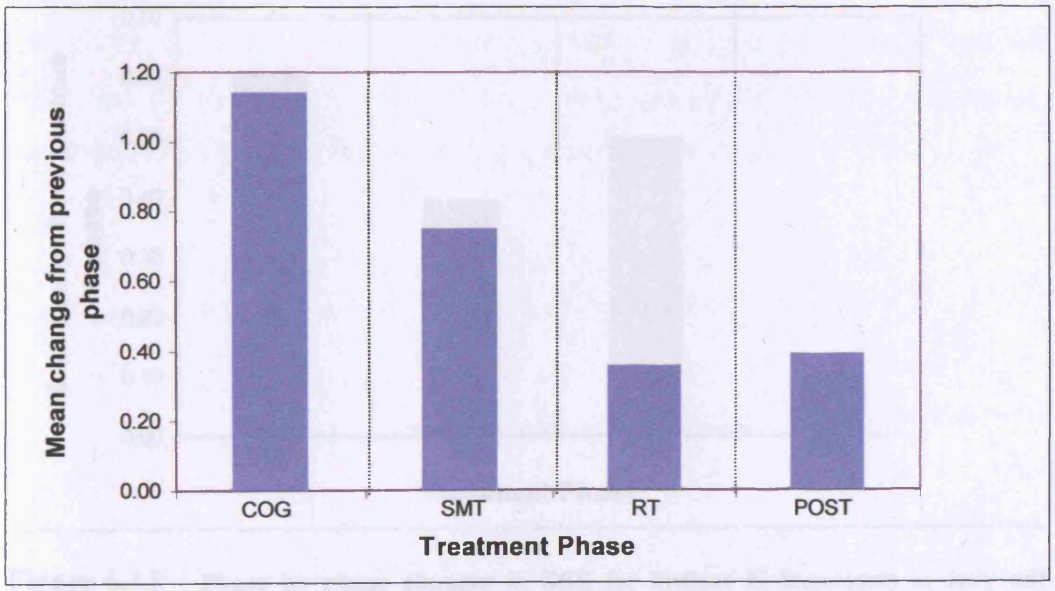


Figure 6.11 **Phase by phase changes in DSE for Patient D.** Increments in daily self-efficacy followed in each treatment phase but were greatest in the cognitive treatment block and least in the RT block. Some gains continued after treatment had ended.

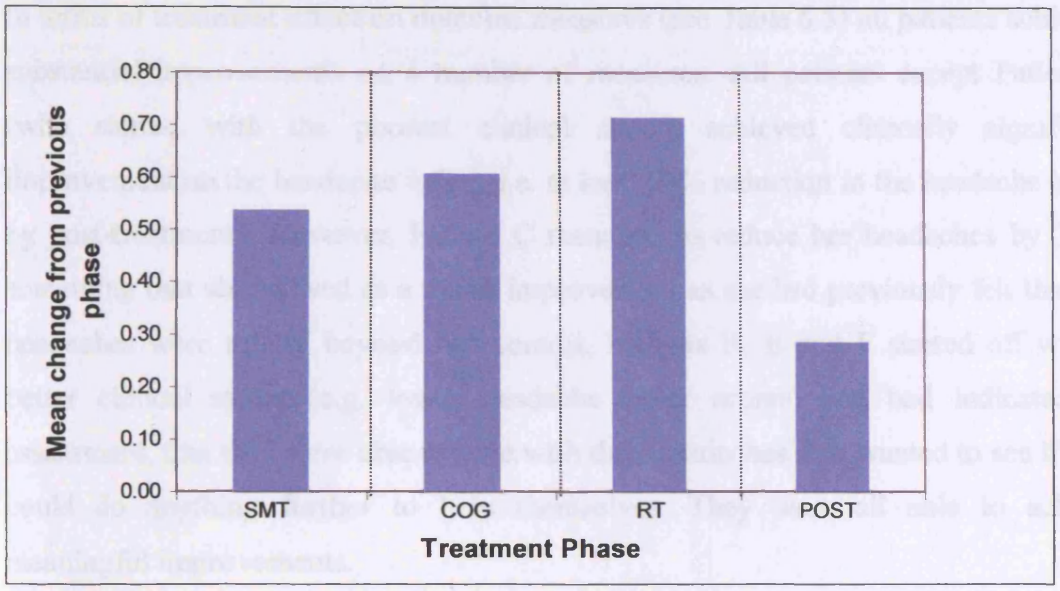


Figure 6.12 **Phase by phase changes in DSE for Patient E.** Increments in daily self-efficacy were of a similar magnitude in each treatment block but greatest within the RT block. Further gains were made in the post-treatment phase.

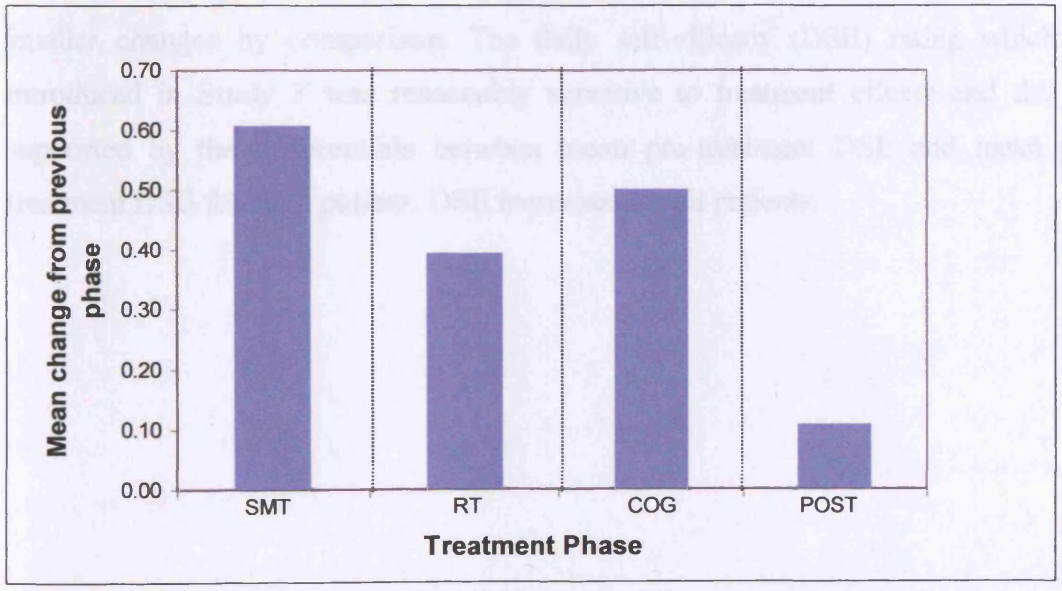


Figure 6.13 **Phase by phase changes in DSE for Patient F.** Increments in daily self-efficacy were of a similar magnitude in each treatment block but were greatest within the SMT block. A small increment was achieved in the post-treatment phase.

6.4.5 Overall Outcome

The overall outcome for each of the six patients is summarised in Tables 6.5 and 6.6. In terms of treatment effect on outcome measures (see Table 6.5) all patients achieved substantial improvements on a number of measures. All patients except Patient C (who started with the poorest clinical status) achieved clinically significant improvement on the headache index (i.e. at least 50% reduction in the headache index by post-treatment). However, Patient C managed to reduce her headaches by 34%, something that she viewed as a major improvement as she had previously felt that her headaches were totally beyond her control. Patients B, E and F started off with a better clinical status (e.g. lower headache index scores) and had indicated, at assessment, that they were able to 'live with their headaches' but wanted to see if they could do anything further to help themselves. They were all able to achieve meaningful improvements.

With regard to treatment effects on cognitive variables (see Table 6.6), the results generally supported the findings from studies 1 and 2. Perceived self-efficacy, catastrophising self-statements and coping self-statements seemed to be the most sensitive to treatment effects whilst the locus of control subscales showed much smaller changes by comparison. The daily self-efficacy (DSE) rating which was introduced in Study 3 was reasonably sensitive to treatment effects and this was supported by the differentials between mean pre-treatment DSE and mean post-treatment DSE for each patient. DSE increased for all patients.

Table 6.5 Treatment effect on outcome measures

Measure	Phase	Patient A	Patient B	Patient C	Patient D	Patient E	Patient F
HI	PRE	10.36	3.21	12.57	9.82	5.89	5.11
	POST	2.36	1.39	8.29	1.21	1.36	1.00
PK	PRE	13.25	6.75	16.25	11.75	8.75	10.50
	POST	5.00	4.75	11.00	3.00	3.5	3.50
HFD (%)	PRE	0	32	0	0	18	32
	POST	9	54	0	46	43	57
HAD-A	PRE	9	8	14	12	13	5
	POST	6	4	9	5	5	4
HAD-D	PRE	5	9	14	7	4	3
	POST	4	4	7	3	2	3
PF	PRE	42	50	16	58	42	33
	POST	58	67	42	83	75	67
RF	PRE	50	50	0	50	0	100
	POST	100	100	50	100	50	100
SF	PRE	60	80	20	60	80	40
	POST	80	80	40	80	80	80

Note. HI = headache index; PK = peak headache intensity rating; HFD (%) = per cent of headache-free days in phase; HAD-A and HAD-D refer to anxiety and depression scores respectively on the HADS; PF, RF, and SF refer to physical functioning, role functioning, and social functioning subscales of the MOSH.

Table 6.6 Treatment effect on cognitive measures

Measure	Phase	Patient A	Patient B	Patient C	Patient D	Patient E	Patient F
DSE	PRE	4.43	6.46	1.04	4.89	5.29	6.39
	POST	7.11	7.36	3.68	7.54	7.43	8.00
PSE	PRE	32	44	2	38	47	47
	POST	42	68	26	47	58	54
I-LOC	PRE	17	16	9	18	15	21
	POST	21	19	11	16	19	22
HP-LOC	PRE	11	9	12	15	12	7
	POST	11	10	12	34	11	9
C-LOC	PRE	5	9	17	7	11	7
	POST	5	7	11	7	9	5
CAT	PRE	18	24	38	22	23	16
	POST	14	16	24	14	17	13
COP	PRE	29	20	14	17	16	26
	POST	38	29	21	32	27	35

Note. DSE = daily self-efficacy rating; PSE = perceived self-efficacy (HSES); I-LOC, HP-LOC, and C-LOC refer to internal, health professional, and chance locus of control respectively (HSLCS); CAT and COP refer to catastrophising and coping self-statements respectively (PRSS).

6.4.6 Summary of Results

1. For most patients, daily self efficacy was found to be strongly and positively correlated with changes in headache activity.
2. The prediction that daily self-efficacy would show the most increase during the cognitive treatment block was not supported.

Chapter 7

Discussion

At the beginning of the research programme, the researcher set himself the broad aim of enhancing the understanding of minimal-contact cognitive behavioural treatment (MC-CBT) for chronic daily headache (CDH). A critical review of the literature indicated that, in contrast to the abundance of outcome studies relating to migraine and tension headache, no work has been done on CBT outcome for CDH, despite this now being a recognised and clinically prevalent headache disorder. In addition, it was argued that the emergence of promising findings relating to the efficacy of minimal-contact formats of CBT require further replication, particularly with difficult-to-treat headache populations such as those with CDH. Finally, despite the wide application and acceptance of CBT treatment approaches, and the assumed primacy of cognitive strategies, it was found that the literature contains major gaps regarding the mechanisms through which CBT might operate. For these reasons, the particular focus of the present research was on two major sets of questions pertaining to: (a) the *efficacy* of CBT and particularly MC-CBT in the treatment of CDH and (b) the *mechanism* through which CBT treatment might operate.

Based on a review of the literature, the broad predictions concerning efficacy was that CBT treatment generally would be effective in the treatment of CDH and that MC-CBT would be equally effective to conventional format clinically intensive CBT (I-CBT). It was predicted that treatment effects would be comprehensive in that improvement would show on a range of outcome measures within the broad areas of headache activity, affect, and, behavioural and functional status. With regard to treatment mechanisms, it was predicted that adaptive changes in cognition were important in the achievement of better outcome and that the explicit teaching of cognitive strategies would be more effective than non-specific expectancy effects in terms of inducing adaptive cognitive changes. Specifically, it was predicted that, with regard to headache management and coping, adaptive cognitive strategies would be indicated by an increased sense of perceived self-efficacy, stronger internal locus of control beliefs, weaker beliefs in health professional and chance loci of control, less catastrophising self-statements and greater coping self-statements.

7.1 Summary of Findings

7.1.1 Treatment Efficacy

With regard to the headache index, the results from Study 1 indicate that MC-CBT is just as effective as I-CBT in reducing headache activity and that both these treatment formats are significantly more effective in comparison to a waiting-list control condition. Mean pre- to post-treatment improvement was 51% in the I-CBT group, 38% in the MC-CBT group, and just 8% for waiting list controls. No significant change was found at 6-month follow-up in the two active treatment groups, and this finding supports the longer-term efficacy of these treatments. The comparative and long-term efficacy of MC-CBT is further supported on the basis of findings from Study 2 in which the control treatment condition was a similar minimal-contact treatment that replaced the explicit cognitive component with a positive coping skills component (MC-PCS). At post-treatment, there was a trend for MC-CBT (mean improvement of 59%) to be superior to MC-PCS (mean improvement of 50%) but this was not statistically significant. However, at 6-month follow-up the gains were significantly better maintained, and even built on, in the MC-CBT treatment group (mean pre- to follow-up improvement of 63% vs. 28% in the MC-PCS group). The findings from the series of single-case experiments within Study 3 add further support to the efficacy of MC-CBT as clear reductions in the headache index were consistently observed following a four-week baseline monitoring period and the subsequent introduction of MC-CBT (mean improvement for the six patients was 68%).

The cross-study pattern of outcomes reported for the headache index were also paralleled by improvements on other outcome measures, namely, self-reported anxiety, and self-ratings of physical functioning, role functioning, and social functioning. These findings tend to support the assumption that a multicomponent treatment such as MC-CBT will lead to wide-ranging outcome effects. However, the findings also suggest that when other outcome measures are considered, namely depression, headache-free days, and medication-free days, the efficacy of MC-CBT and I-CBT is less clear.

7.1.2 Treatment Mechanisms

As argued earlier, the hypothesis that the mechanism of treatment change in CBT treatments is primarily cognitive change requires two related empirical tests before it can be supported. First, CBT treatment must be able to induce significantly greater adaptive cognitive changes than other treatments. Second, adaptive cognitive changes should correlate with positive treatment outcome.

With regard to bringing about adaptive cognitive changes, the findings from Study 1 support the comparative and long-term efficacy of both I-CBT and MC-CBT. However, the strength of this treatment effect was variable with regard to the type of cognitive measure that that was considered. Perceived self-efficacy for headache management (increase), and catastrophising self-statements (decrease) were particularly improved in both treatment groups, whereas changes in health-professional locus of control and chance locus of control were not as marked. Changes in coping self-statements and strength of internal locus of control beliefs were intermediate.

Having established some support for the efficacy of MC-CBT in bringing about adaptive cognitive changes, Study 2 was set up to test whether the explicit cognitive component of treatment was primarily responsible for this. The findings show that whilst a 'non-cognitive' minimal-contact treatment package is just as effective as MC-CBT in the short-term, over the longer term, MC-CBT is significantly more effective. The pattern of treatment effects was similar to that for Study 1 with perceived self-efficacy, and catastrophising self-statements being particularly sensitive to treatment. The conclusion is that explicit cognitive training strategies are required within a minimal-contact treatment package if the adaptive cognitive changes are to be maintained over the longer term. In contrast to the findings of Studies 1 and 2, the single-case experiments within Study 3 showed that when a daily measure of self-efficacy is considered the explicit cognitive training component is no more effective, than other treatment components, in bringing about positive changes. However, there was a trend for the cognitive training component to be more effective, and the overall treatment package led to similar pattern of gains as that reported for Studies 1 and 2.

With regard to the correlation between adaptive cognitive changes and positive outcome effects, the findings were mixed. Findings from Study 1 suggest that whilst there may be a moderate correlation between cognitive changes and outcome change, a large part of the variance on each outcome measure (at least 64%) is explained by factors other than the collective changes on the cognitive variables considered here, namely perceived self-efficacy, locus of control beliefs, and appraisal self-statements. Adaptive changes in perceived self-efficacy (increase) and catastrophising self-statements (decrease) appear to be most predictive of positive outcome effects, particularly on the measures of headache index (decrease) and physical functioning (increase). More convincing support for an association between cognitive change and outcome change was offered by the single-case experiments within Study 3. The findings suggest that, when a daily measure of self-efficacy is considered, this has a strong and significant inverse correlation with daily headache ratings.

Note on Terminology

In the following discussion, the term CBT is used to cover both I-CBT and MC-CBT. The separate terms are used where the discussion concerns differences between the two.

7.2 The Efficacy of MC-CBT Treatment for CDH: Interpretation

The finding that minimal contact cognitive-behavioural treatment (MC-CBT) is just as effective as conventional-format clinically intensive CBT (I-CBT) in the treatment of CDH supports similar findings from studies that have looked at chronic headache sufferers with less frequent headaches (Haddock *et al.*, 1997; Primavera & Kaiser, 1992; Rowan & Andrasik, 1996). In some respects this finding seems surprising given the assumption held by some researchers (e.g. Blanchard *et al.*, 1989) that CDH sufferers may require more intensive psychological input compared to other chronic headache groups. This view essentially emerged from a small body of studies, which showed that established psychological treatments were not effective with CDH. However, in the light of the current findings, a more critical appraisal of the outcome literature is possible. This critique is taken up in the ensuing discussion concerning specific findings from the present research.

7.2.1 Headache Activity

The research programme supported the overall efficacy of CBT treatment for CDH. The treatment gains in terms of headache activity (38% to 65%+ improvement) are of a similar magnitude to those reported by others in the context of cognitive-based treatments for chronic headache (Blanchard, 1992; Gauthier *et al.*, 1996; Martin, 1993). The longer-term efficacy of CBT is also supported and this corroborates previous findings (Knapp, 1982; Sorbi, *et al.*, 1989) though the patients in the present research were not followed up beyond six months after treatment. The overall conclusion is that CBT treatment appears to be as effective for CDH patients as it is for chronic headache sufferers with less frequent headaches.

Whilst no previous work has been done to test the efficacy of CBT with CDH patients, the current findings contrast with the conclusions from the small body of empirical evidence pertaining to psychological treatment of 'refractory' headache patients (Blanchard *et al.*, 1989; Michultka, *et al.*, 1989). This body of research has found that patients with chronic, frequently recurring headache tend not to respond to psychological treatment. A number of explanations can be put forward for this apparent discrepancy:

Firstly, the treatments described by Blanchard *et al.* (1989) and Michultka *et al.* (1989) did not appear to be as structured as the treatment programme used in the present research and they did not include a substantive cognitive therapy component. In fact in their conclusions, Blanchard *et al.* speculated that CDH patients might respond better to a treatment programme that includes cognitive therapy. Another possibility for the discrepancy is that the headache sufferers in the present research differed considerably from those in previous research. However, in terms of pre-treatment headache index scores, the CDH patients in the present research are comparable to those who participated in the studies reported by Blanchard *et al.* and Michultka *et al.* (headache index scores around 10). In addition to this, no major differences in psychiatric symptoms are apparent as both the current research and previous studies reported moderate anxiety levels and slightly elevated depression in the studied groups.

A more plausible explanation for the comparatively good response to CBT treatment reported here is that most patients were not on high doses of analgesic medication. Previous research has shown that high medication consumption is associated with much poorer response to psychological treatment (Michultka *et al.*, 1989). The skew towards low or no medication use reported by patients in the present group of studies did not allow for statistical analysis of high versus low medication users. However, the indications were that patients who struggled to reduce medication intake tended not to respond as well to the CBT treatment programme.

As expected, treatment effects for peak headache intensity ratings were less marked than those for headache index scores but the efficacy of CBT treatment compared to no-treatment was still found to be statistically significant on this measure. Although specific headache symptoms were not monitored on the headache diary, it is possible that for some patients the peak intensity represented migrainous symptoms which have usually been found to be more intractable to cognitive-based treatments (Blanchard *et al.*, 1989; Laher, 1994). Headache-free days were found not to change significantly as a result of treatment. The suggestion, therefore, is that CBT treatment is more effective in reducing intensity rather than frequency of headaches. An implication of this with regard to the treatment mechanism is that CBT works not through controlling presumed precipitants of CDH (e.g. stress) but through strategies to lessen ongoing pain. This possibility is examined in more detail in the section looking at the role of stress.

7.2.2 Clinically Significant Change

The findings indicated that when clinically significant improvement on the headache index is considered as an outcome measure the efficacy of CBT is less pronounced but still better than no-treatment. Whilst the 50% cut-off, to define clinically significant improvement within each patient, is a meaningful clinical indicator (Blanchard & Schwarz, 1988), it is, nonetheless, arbitrary. An obvious problem with percentage improvement as an indicator of change is that, for a given level of change, this would be influenced by the absolute level of pre-treatment headache index scores. Thus there would tend to be over-estimation of the level of change in those who start with a low headache index whilst there would be an under-estimation in those who

start with a much higher headache index. For example: a patient who improves by 4 points from a headache index of 5 at pre-treatment to 1 at post-treatment has achieved 80% improvement, whereas a patient whose headache index improves from 11 to 7 has achieved just 36% improvement and would not be classified as clinically improved. Yet for the latter patient the improvement may be far more beneficial in terms of daily functioning (as was the case with patient C in Study 3).

The above point is particularly relevant in the context of CDH where patients have much higher headache index scores than those reported for chronic headache groups generally. It is therefore suggested that if a cut-off for clinically significant improvement is to be maintained in clinical research then this might be more useful if it was set according to a band of high, mid and low headache index scores. Whilst there are no norms for headache index scores, a review of the literature suggests that a headache index of 8 or above can be considered to be in the high range and a score of 3 or below may be considered in the low range. A less stringent but no less meaningful cut-off of at least 30% improvement for those whose headache index is in the high range (e.g. CDH sufferers) may be a standard well worth establishing.

7.2.3 Wider Outcome Measures

The finding that CBT treatment was found to be significantly effective, compared to no-treatment, in reducing anxiety and depression whilst improving physical, role and social functioning supports the multidimensional efficacy of such treatment. Despite the wide acceptance of the Gate Control Theory of Pain (Melzack & Wall, 1965, 1996) a criticism of much past outcome research in the headache field is that there has been an over-reliance, through use of the headache index, on the sensory component of pain as an outcome measure. The findings from the present research suggest that outcome from cognitive based treatments for chronic headache may have been underestimated in the past as positive changes on affective, behavioural and cognitive variables were not routinely measured or reported. Where these wider treatment effects have been reported, the tendency has been to view them as separate effects to pain or headache reduction rather than as integral aspects of the pain experience itself (Blanchard, Steffek, *et al.*, 1991).

Treatment effects were more marked for anxiety than for depression. The main explanation for this is that the pre-treatment clinical status of the studied patients was such that anxiety levels were much higher (and in the clinical range) compared to low, non-clinical levels of depression. The finding that depression levels were low to begin with, in a group of patients with chronic frequently recurring pain, seems surprising if one assumes that the level of pain bears a close relationship to mood. However, whilst some research has shown there to be an association between pain and mood (Blanchard, Kirsch, Appelbaum, & Jaccard, 1989; Cox & Thomas, 1981) a more likely hypothesis in the context of the CBT model and Gate Control Theory is that other variables such as behavioural impact, social support and cognitive coping have a moderating effect on this relationship (Philips & Jahanshahi, 1985).

A further explanation for low levels of depression is that the patients who participated in the research may have been a self-selecting sample in that they were already motivated to actively participate in a psychological treatment programme that was advertised as a 'self-management approach'; those with high levels of clinical depression may have been desperate for medical/pharmacological treatment rather than psychological treatment in which they would be required to actively engage. This assertion is contrary to the conclusions of Rokicki & Holroyd (1994) who studied factors influencing treatment-seeking behaviour and found that levels of depression and neuroticism were not predictive of whether or not chronic headache sufferers sought treatment. However, a major omission in Rokicki and Holroyd's paper is that they neglect to specify whether they refer to psychological or medical treatment or both. It is possible that high levels of depression predict poor take-up of psychological treatment only.

Significant improvements in quality of life measures following treatment lend further support to the wider efficacy of a multicomponent CBT treatment package. As expected, the CDH patients in the present research had more adverse scores on quality of life measures than those reported for chronic headache sufferers generally (Solomon *et al.*, 1993). This supports the view that CDH is far more debilitating than other headache disorders (Blanchard *et al.*, 1989; Solomon *et al.*, 1992).

The finding that physical functioning (a measure of such things as mobility and ability to carry out usual daily activities) improved most as result of treatment compared to social functioning and role functioning (ability to perform work) can be explained in a number of ways. Firstly, the post-treatment review session indicated that many patients felt more able and motivated to change aspects of their daily physical functioning whereas they did not see social engagements as their top priority, and it was easier to avoid social activities. Ability to perform work was felt to be less in their control and the perceived demands of role fulfilment may have been seen as too great to effect meaningful improvement. Secondly, it is possible that patients were much clearer about their goals for change regarding physical functioning compared to other aspects of quality of life and therefore improvement in physical functioning was more easily discernible.

Whilst the tendency was for medication-free days to increase following treatment, firm conclusions cannot be drawn about the efficacy of CBT with regard to this measure as most patients, particularly in Study 2, were not taking any medication from the outset. The fact that many patients still presented with CDH despite them not taking any headache medication suggests that the analgesic rebound hypothesis about the aetiology and maintenance of CDH is not as robust as claimed by some researchers (e.g. Kudrow, 1982; Rapoport, 1987). The indication is that other factors are also important.

7.3 Treatment Mechanisms: Interpretation

The findings from the present research suggest that there is no significant difference in the treatment mechanism between MC-CBT and I-CBT and that treatment mechanisms within CBT can be considered at two levels: (a) At a macro level, the treatment component which is mainly responsible for long-term change seems to be the component which teaches explicit cognitive coping strategies; (b) at a micro level, change in perceived self-efficacy and catastrophising self-statements appear to be important mediators of treatment change. The interpretation of these findings is discussed in more detail below.

7.3.1 The Contribution of the Cognitive Component

The support for the importance of the cognitive component within the overall treatment package in terms of longer-term outcome and multidimensional outcome helps to clarify mixed findings from previous research. Attanasio *et al.*, 1987 concluded that whilst minimal contact treatment was cost-effective the inclusion of a cognitive component did not make a significant difference to treatment outcome. However, the researchers did not conduct a follow-up on their treatment groups and hence the crucial test of maintenance of treatment gains was omitted. The current findings clearly suggest that the inclusion of the cognitive component is vital if treatment gains are to be maintained. This supports the assumption within the CBT model that deeper cognitive change rather than simple expectancy effects play a major role in the treatment mechanism.

The current findings also prompt a reappraisal of the conclusions from two studies by Blanchard's research group pertaining to the incremental efficacy of the cognitive therapy component in the treatment of tension headache (Appelbaum *et al.*, 1990) and migraine (Blanchard, Appelbaum, Nicholson, Radnitz, Morill, *et al.*, 1990). In both studies it was concluded that, statistically, the cognitive component did not significantly add to treatment gains even at four-month follow-up. In terms of clinical significance however, there was an advantage in including the cognitive component in tension headache but not migraine. A criticism is that these conclusions were based solely on headache index and medication index change scores whereas the current findings suggest that the cognitive component is crucial when wider outcome measures are considered.

7.3.2 The Mediating Role of Cognitive Changes

Overall, the current findings lend some support to previous work which has shown that changes relating to cognitive variables such as perceived self-efficacy, locus of control and appraisal self-statements play an important role in the treatment process (Holroyd, *et al.*, 1984; Mizener *et al.*, 1988; Newton & Barbaree, 1987; ter Kuile *et al.*, 1995). The current findings suggest that three important considerations need to be kept in mind with respect to the role of cognitive changes. Firstly, the specificity of cognitive variables needs to be considered. The current findings showed that different

cognitive processes exert differing degrees of influence on the treatment process. Secondly, one needs to consider the extent to which the cognitive changes are due to treatment factors other than the specific teaching of cognitive coping strategies. The current findings indicate that whilst non-specific expectancy effects, and cognitive changes arising from other treatment components occur, more enduring cognitive change is brought about by the explicit teaching of cognitive coping strategies (e.g. cognitive restructuring). Thirdly, and most crucially in terms of the cognitive change hypothesis, outcome change needs to be causally linked to cognitive change. An obvious limitation of the current findings is that causality cannot be inferred from the correlational approach used to look at the relationship between cognitive changes and outcome changes.

7.3.2.1 The Role of Self-Efficacy

The finding that self-efficacy increases significantly at post-treatment regardless of whether explicit cognitive treatment strategies are taught partly supports Bandura's assertion that self-efficacy plays an important role in any treatment intervention (Bandura, 1977, 1997) This finding also appears to be compatible with Bandura's view that mastery of performance is a more potent determinant of self-efficacy than (in order of potency), vicarious learning, verbal persuasion, and emotional or physiological arousal. It is possible that patients' self-efficacy increased as they gained more practice in, and became more skilled at applying practical headache management strategies such as relaxation training. However changes in emotional and physiological arousal may have also been important in changing self-efficacy beliefs and this has been demonstrated in experimental studies reviewed earlier (e.g. Holroyd *et al.*, 1984).

One of the main findings from the current research is that, despite the conclusions drawn from post-treatment evaluations of self-efficacy changes, the key question of whether explicit cognitive strategies add significantly to such changes cannot be addressed unless longer-term effects are measured. The clear indication from the current research is that, over the longer-term, cognitive strategies are very important if the initial increases in self-efficacy are to be maintained. This suggests that mastery of practical or behavioural management strategies is not enough to sustain positive

change in self-efficacy and that mastery of cognitive restructuring strategies is vital in this respect. This is consistent with the finding that increases in self-efficacy can arise from non-specific treatment factors (e.g. expectation of improvement). However, the current findings suggest that any opportunistic improvements in self-efficacy still need to be harnessed, through explicit cognitive strategies, if the gains are to be maintained once the treatment programme has ended.

Establishing that cognitive therapy strategies are more effective (than other planned and non-specific treatment variables) in inducing increases in self-efficacy only partly supports the postulates of a CBT model. The real test is whether changes in self-efficacy are causally associated with treatment outcome. Whilst causality cannot be inferred from correlational analyses, the current findings suggest that there is an association between increases in self-efficacy and positive outcome changes (e.g. lower headache index, lower anxiety levels, better physical functioning). With respect to these findings, three observations merit further discussion.

Firstly, the moderate size of the correlations between self-efficacy changes and outcome changes suggest that other variables are also important in determining outcome. Multiple regression analyses indicated that, even after other cognitive variables were considered, a large part of the variance in outcome still remained unexplained. It is also possible that the true strength of the correlation between self-efficacy and outcome is much greater and that this could have been demonstrated if a much larger sample was used. However, using tables provided by Cohen (1988) and Aron and Aron (1999) it appears that lack of power was not a significant factor. Most of the bivariate correlations were around .5, which is deemed to be a large effect size. To achieve 80% power with alpha set at $p < .05$ and a large effect size requires a sample size of 28 (two-tailed) or 22 (one-tailed) – the correlations reported here are based on a sample size of 37.

Secondly, self-efficacy changes were found to be more strongly associated with outcome on the headache index and physical functioning whereas correlations with anxiety and depression changes were relatively weak. This suggests that self-efficacy for headache management bears a closer relationship to the sensory and behavioural component of pain than to the affective component. The floor effect in depression

scores precludes any meaningful conclusions with regard to the relationship between headaches, affect, and self-efficacy. However, the finding that anxiety was significantly reduced as a result of treatment whilst there was only a weak correlation between increase in self-efficacy and decrease in anxiety is interesting. A possible explanation, based on the predictions of a multicomponent CBT model, is that cognitive variables other than self-efficacy beliefs mediate in the CBT treatment of chronic headache. The much stronger correlation between decrease in catastrophising and decrease in anxiety partly supports this assertion. This specific finding is discussed later.

Thirdly, the correlation between a daily measure of self-efficacy and daily changes in the headache index (Study 3) was found to be much stronger than the correlations based on mean pre-post changes between self-efficacy and the headache index (Studies 1 & 2). One reason for this may pertain to the way in which data was collected and analysed. The single case studies allowed a more detailed (daily) and prospective monitoring of how self-efficacy and headache index ratings covaried whereas this level of detail was lost when the data consisted of four-week mean scores for the headache index and a retrospective questionnaire based score of headache self-efficacy. This suggests that whilst the Headache Self-Efficacy Scale is sensitive to global changes in self-efficacy, it may overlook meaningful fluctuations that occur on a daily basis. The implication from a theoretical point of view is that self-efficacy beliefs may consist of two interacting components that are separated according to their temporal characteristics: (1) a relatively stable component that is amenable to change over the course of a twelve-week treatment programme, and (2) a fluid component that may exert a more immediate impact on daily functioning and pain.

The theory that self-efficacy plays a mediating role in treatment outcome has gained much support amongst researchers. However, the key premise of this theory, that self-efficacy change precedes outcome change, remains contentious. It can be argued that self-efficacy itself may be an outcome variable that changes concurrently with other outcome variables (Borkovec, 1978). If this is the case, then the question remains as to what the main mechanism of treatment is within CBT treatment.

A further area of contention relates to the status that Bandura (1977) accorded to self-efficacy expectations as opposed to outcome expectations, which pertain to the expected impact of the course of action. That is, individuals make two types of efficacy appraisals: to what extent they can initiate and maintain a strategy to change toward a desired goal (e.g. being able to learn/practise relaxation strategies to manage headaches); and to what extent the perceived goal might be realised (e.g. being headache-free and being able to enjoy social activities again). Bandura has consistently maintained that self-efficacy rather than outcome-efficacy is the more powerful predictor of behaviour change but the literature is inconclusive on this. A problem is that Bandura seemed to have used the concept of outcome quite narrowly to refer to some sort of global treatment outcome. However, in the context of CBT treatment, small targets for change are usually planned throughout treatment. It may be that achievement of these 'mini-outcomes' is associated with significant increases in outcome efficacy and it is this rather than self-efficacy expectations which lies at the heart of the treatment mechanism.

7.3.2.2 The Role of Locus of Control Beliefs

The clinical status of patients at pre-treatment showed that the strength of locus of control beliefs was similar for internal locus of control (I-LOC) and chance locus of control (C-LOC) whereas locus of control beliefs in health professionals (HP-LOC) were somewhat weaker. This seems surprising in that the literature on refractory chronic pain and headache populations predicts that sufferers would have a little belief that they can control the headaches or pain and strong beliefs that control lies mainly with powerful others such as health professionals (Harkapaa, *et al.*, 1996; Scharff *et al.*, 1995). It is possible that this discrepancy might be due to an 'anti-doctor' phenomenon whereby patients who have had their symptoms for a long time and who may have unsuccessfully tried numerous medical interventions and gone through several investigations lose faith in doctors and other health professionals. Whilst this phenomenon can be commonly observed in clinical practice it remains to be tested empirically.

The finding that internal locus of control beliefs for headache management strengthened as a result of psychological treatment is consistent with previous

empirical work (Fisher & Johnston, 1998; N.J. Martin *et al.*, 1990; Scharff *et al.*, 1995). However, the present research also showed that a cognitive based treatment is more effective in inducing an increase in internal locus of control and that MC-CBT is just as effective in this respect as I-CBT. The results from Study 2 indicate that the inclusion of the cognitive component in the treatment package is important if the increase in internal locus of control is to be maintained over the longer term. This lends some support to previous empirical work, relating to chronic pain and headaches, which has suggested that strength of internal locus of control beliefs mediates treatment outcome and predicts engagement in self-management strategies. However, two observations tend to mitigate these conclusions.

Firstly, the treatment effects for internal locus of control beliefs, whilst being statistically significant, were of a much lower magnitude than those for self-efficacy or catastrophising self-statements. This suggests that locus of control is not the most important cognitive variable in the treatment process. Such a conclusion seems incompatible with previous findings that those with a high internal locus of control are more likely to take up psychological treatment, more likely to benefit, and less likely to drop out. A possible explanation for this is that previous findings related to the predictive capacity of *initial* status regarding internal locus of control beliefs whereas the current research was concerned with *change* in locus of control beliefs of patients who stayed for the whole duration of the treatment programme.

Secondly, increase in the strength of internal locus of control beliefs was found to correlate only weakly with change on outcome measures. This suggests that whilst internal locus of control beliefs may predict take-up of psychological treatment in the first place, they do not play a major part in the treatment mechanism once the patient decides to persevere with treatment.

With regard to external locus of control beliefs, health professional locus of control which was weak to begin with was found to be largely unchanged in all patients regardless of the type of treatment offered or whether or not treatment was offered. In contrast, attribution of locus of control to chance factors decreased as a result of psychological treatment and this decrease was much better maintained if the treatment included a cognitive component. In parallel with most of the results, MC-CBT was

just as effective as I-CBT in inducing a reduction in the strength of belief that locus of control for headaches lay in chance or fate.

Disillusionment with health professionals has already been discussed above as a possible reason why locus of control beliefs in health professionals remained weak. However, interestingly, at post-treatment review, many patients discriminated between the role of the psychologist whom they perceived as helpful and enabling, and the role of doctors with whom they had lost faith regarding management of headaches. Both these points suggest that the health professional component of external locus of control may not always be an accurate indicator of beliefs in powerful others' ability to control headaches and that strong or weak external beliefs may not be linearly related to adaptive and non-adaptive coping respectively, as has traditionally been assumed. In other words, the current findings suggest that it is possible to attribute strong control of headaches to one type of health professional and weak control to another type of health professional while, at the same time, feeling that headaches are largely within one's personal control. Such a scenario would be consistent with the enabling role of the therapist in CBT treatment. As the Headache-Specific Locus of Control Scale does not distinguish between different types of health professional, it was not possible to test this assertion.

Overall, the findings suggest a number of things with regard to the locus of control construct. Firstly, the findings tend to support the presumed independence of the separate loci. This means that internal and external loci are not necessarily inversely related. Secondly, the finding that the internal locus of control measure is particularly sensitive to treatment change supports much previous research that has suggested that strength of internal control beliefs is particularly important predictor of the degree of coping and level of engagement in treatment. Thirdly, it seems that locus of control beliefs may be important in determining treatment participation but are not, in themselves, a major part of the mechanism of treatment change.

7.3.2.3 The Role of Appraisal Self-Statements

The finding that both I-CBT and MC-CBT were able to significantly reduce headache-related catastrophising self-statements and increase coping self-statements

tends to support the postulates of the CBT model and is consistent with previous empirical findings (Flor *et al.*, 1993; Gil *et al.*, 1990; Newton & Barbaree, 1987). This support is further strengthened by the finding that the cognitive component of treatment is crucial with regard to the longer-term maintenance of reduced catastrophising and improved coping.

The magnitude of the treatment changes in respect of catastrophising self-statements was generally greater than that for coping self-statements. This lends some support to previous research that has suggested that CBT works mainly through controlling catastrophising thoughts rather than through enhancing coping thoughts (Rosenteil & Keefe, 1983; Turk & Rudy, 1992). However, if statistical significance of treatment changes on these cognitive measures is taken into account then the conclusions from the current findings are that CBT has a significant effect on both catastrophising self-statements and coping self-statements. It is possible that a part of this discrepancy is due to differences in the way in which the CBT programme was defined and applied as well as differences in the way in which appraisal self-statements were elicited and measured. For example: the present research used the Pain-Related Self-Statements Scale which automatically elicits information on both catastrophising and coping cognitions; other studies using open-ended assessment of cognition may have been biased towards eliciting catastrophising statements as people in distress find it easier to identify negative thoughts (Beck *et al.*, 1979; Turk & Rudy, 1992).

The finding that reduction in catastrophising and increased coping thoughts were significantly associated with better outcome, particularly in terms of the headache index and physical functioning, is consistent with the view that cognition mediates treatment outcome. However, the caveats to this tentative conclusion are same as those that have been discussed with regard to perceived self-efficacy changes. These are (a) causality cannot be inferred from correlations (b) the size of the correlation coefficients were around .5 with none being higher than .56, suggesting that a substantial part of the variance in outcome change is not associated with change in appraisals (c) appraisal self-statements may also be part of treatment outcome and change concurrently to other outcome variables rather than being part of the treatment process.

7.4 Theoretical Implications

It is widely accepted that the CBT model is based on the interaction of cognitive, emotional, behavioural and physiological variables with cognitive processes playing some sort of central role. In addition, the application of the CBT model to the understanding of chronic headaches has usually also involved assumptions about the role of stress. Whilst emerging evidence concerning treatment mechanisms has given tentative support to this broad framework, the current research suggests that a reappraisal of the CBT model as applied to chronic headaches may be worthwhile.

It is asserted that the current findings help to clarify some of the specific postulates of the CBT model, particularly with regard to the role of cognitive factors. Therefore, the present discussion focuses on the role of cognitive factors but also goes on to discuss emotional factors, behavioural factors, and the role of stress. Physiological processes were discussed at length in the Introduction and are not discussed here as no specific new implications emerged in the light of the current findings. Physiological processes are, however, incorporated in a revised CBT framework for chronic headache, which is suggested at the end of this section.

7.4.1 Cognitive Factors

Although cognitive factors are widely assumed to be at the heart of the CBT model, clear information about the nature of such factors and empirical research that has convincingly demonstrated purported mechanisms of CBT (e.g. reducing catastrophic thinking) is still relatively scarce. Furthermore, it seems that debate has been hampered by the fact that numerous cognitive constructs have been invoked with the result that a meaningful integrated framework is lacking. However, despite the apparent lack of unifying framework and drawing on the present findings together with emerging developments in the literature it is possible to speculate on the key cognitive processes and variables that are important in CBT.

The present findings suggest that whilst there are likely to be a number of cognitive and non-cognitive variables that form part of the treatment mechanism in CBT, two sets of cognitive processes seem particularly important. These pertain to (1) general

appraisal style (e.g. tendency to catastrophise) and (2) control beliefs and expectations (e.g. perceived self-efficacy for applying treatment strategies). Given that these processes alone were found to be insufficient in terms of explaining the mediating role of cognition prompts speculation about the role played by other cognitive processes. A prime contender as a third cognitive process that might be important relates to all those phenomena that can be subsumed under information processing.

Information processing variables refer to cognitive processes and presumed structures pertaining to attention, memory, and perception. This whole area which has traditionally been seen as the preserve of experimental cognitive psychology and neuropsychology is now drawing increasing interest from researchers in the fields of mental health and pain. This interest has been fuelled by findings, which have shown that information-processing variables interact with subjective appraisal, emotion, behaviour, and physiology. For example, studies have shown that pain patients selectively attend to pain stimuli (Eccleston, 1995; Pearce & Morley, 1989; McCracken, 1997) and that patients' self-report of pain may be influenced by an interaction between memory variables, current state, and whether it is the sensory, affective, behavioural, or cognitive dimension of pain that is recalled (Bryant, 1993; Feine, Lavigne, Thuan Dao, Morin, & Lund, 1998; Morley, 1993). These findings are of relevance to the assessment and treatment of chronic headache and have provided empirical credence to well established clinical strategies such as attention-diversion training.

Therefore, the forgoing discussion suggests that three main types of cognitive processes are important in the treatment mechanism for CBT, and by implication, in the coping mechanism for chronic headaches. These are: (1) information processing, (2) appraisal style, and (3) control beliefs and expectations. Whilst the current findings tend to support this assumption, further empirical research that identifies the purported cognitive processes, is clearly required. In addition to identifying the cognitive processes that operate, a robust CBT model requires clear information about the nature of the relationships between different cognitive processes and how these, in turn, interact with behavioural, emotional and physiological variables. A preliminary hypothesis is that the three putative cognitive processes interact in reciprocal fashion such that none is dominant or super-ordinate. Based, on the prevalent view about the

mechanism within the CBT model, it is asserted that this dynamic interplay between different cognitive processes determines the content of cognition and reciprocally interacts with other psychosocial and physiological variables. For example: a negative appraisal style may encourage selective recall of episodes when the individual failed to control headaches which in turn strengthens negative beliefs and maintains a perception of poor self-efficacy. This may lead to cognitions such as "I just will not be able to cope if I go to that wedding..." and encourage avoidance thus maintaining poor self-efficacy and so on.

A further level of cognitive processing that has usually been invoked in the context of a CBT model concerns the presumed activation of a more stable knowledge structure about the world, the self, and pain. This is largely based on Beck et al's (1979) concept of a *schema* in which "Relatively stable cognitive patterns form the basis for the regularity of interpretations of a particular set of situations." (p. 12). The idea of a fairly stable underlying cognitive structure that determines and is maintained by ongoing cognition and behaviour whilst at the same time being amenable to modification on the basis of experience, is well established in CBT theory. Converging lines of evidence relating to CBT theory and social cognition theories suggest that global schemas and specific representations of illness are important in driving the cognitive processes described above (Williams, 1997).

In summary, the interpretation of the current findings regarding the role of cognition in a CBT model is consistent with the established view that cognitive processes can be separated from a deeper cognitive structure with which they interact. The important conclusion from the current research is that cognitive processes themselves subdivide into the categories of appraisal style, control beliefs and expectations, and information processing. It is hypothesised that the balance between these cognitive processes determines the content of cognition. Such content is likely to be a combination between pain- or headache-specific cognition and cognition about wider psychosocial factors. The hypothesised relationship between different levels of cognition in a CBT model are depicted in Figure 7.1.

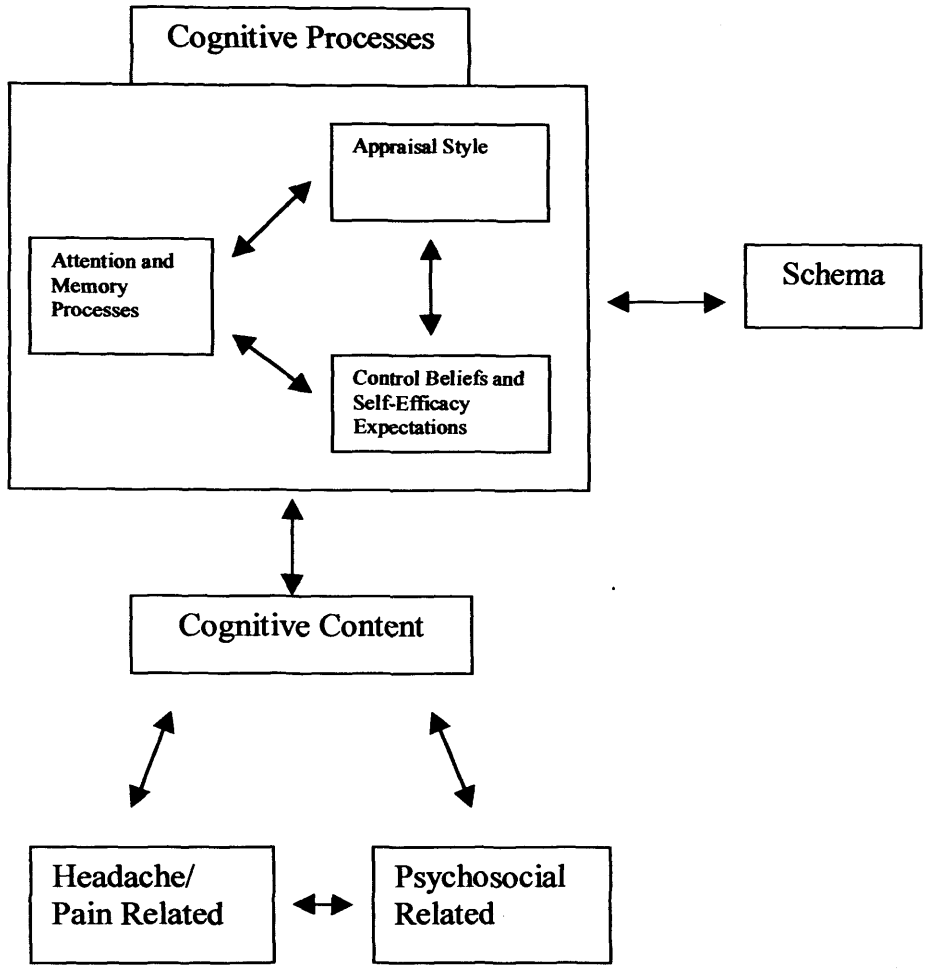


Figure 7.1 Chronic headache and cognitive factors: A working model. The interactional relationships between the different classes of cognition and between the content of such cognition are shown by the several feedback loops.

7.4.2 The Role of Emotional Factors

Although the relevance of psychological input for the emotional component to chronic headache seems self-evident, its position in a CBT framework needs to be clarified. Firstly, from the outset, CBT approaches in psychiatric settings acknowledged a link between cognition and emotion (Beck, 1976). This was initially, presumed to be a simple and direct causal link such that negative cognition led to emotional distress. Later, as with the rest of the CBT model, while cognition remained central, reciprocal interaction was acknowledged. Secondly, emotion itself is known to interact with behavioural and physiological factors. For example, there appears to

be an association between depressive symptoms and avoidance of activity. This is discussed within the next section.

7.4.3 Behavioural Factors

The current findings are consistent with the view that pain behaviour such as taking analgesics and avoiding activities play some part in how headache sufferers cope and how they respond to treatment. However, more relevant to the CBT approach is the consistent indication that behavioural factors also interact with cognitive and emotional variables. Philips (1987) proposed that while avoidance behaviour was a prominent feature of pain maintenance, cognitive factors such as feelings of self-efficacy and memories of past exposures to pain reciprocally interacted with avoidance behaviour. For example, avoidance could lead to decreased self-efficacy, which in turn encouraged further avoidance of anticipated or perceived pain-inducing situations. A similar view emerges from the functional model of chronic headache proposed by Martin and colleagues (Martin, 1993; Martin, Milech & Nathan, 1993). This model is not too divergent from a CBT framework but the emphasis is on a range of antecedents and consequences of headaches rather than specifically on cognitive appraisal.

7.4.4 The Role of Stress

Although stress was not directly quantified in the current research, information from clinical assessment of patients and from post-treatment review prompts a reappraisal of the role of stress in headache coping and in the treatment mechanism. CBT treatment packages for chronic headache have tended to include specific stress-management components or have assumed, in the overall approach, that 'stress' needs to be controlled. As has already been discussed, such interventions for chronic headache have been based on either the stress-coping approach or the disorder-related distress approach. In the stress-coping approach headaches (especially tension-type headaches) are presumed to be triggered or aggravated by stress (stress-coping hypothesis) whereas in the disorder-related distress approach it is posited that by the very fact of its chronicity, the headache disorder itself has become the main stress (cognitive shift hypothesis).

In examining the stress-headache relationship, the finding that headache-free days did not increase significantly despite significant reduction in headache index scores and peak headache intensity ratings merits further discussion. This finding suggests that treatment strategies were more effective in terms of helping patients to manage and reduce the intensity of existing headaches rather than preventing headaches from occurring. Such an interpretation would tend to support the view that most stress relates to the headache disorder itself rather than being attributable to external sources. This is consistent with the cognitive-shift hypothesis.

In contrast to the current findings, Murphy *et al.*'s (1990) findings tend to support the stress-coping hypothesis. Having found that the superiority of cognitive therapy over relaxation treatment was particularly marked in terms of headache-free days, Murphy *et al.* speculated that this might be explained by participants' greater ability to prevent headaches from occurring, through controlling stressors that might provoke them. Once a headache did take hold, the stress-headache link was weaker and, therefore, stopping an existing headache through managing stress was less successful.

One explanation for the discrepancy between the current conclusions and those presented by Murphy *et al.* (1990) relates to the assumptions implicit in both views that headache-free days relate directly to the control of presumed stress triggers and that stress can only be considered as an initial trigger. In frequently recurrent headache such as CDH, it is also possible that ongoing, daily stress aggravates existing headaches and it is the control of such ongoing stress through which treatment works. Whilst this is compatible with the stress-coping hypothesis it suggests that the main source of stress is in daily events. Such a conclusion is consistent with empirical findings which have shown that headache sufferers do not experience significantly more major life events than matched headache-free controls (Andrasik, Blanchard, Arena, Teders, Teeevan *et al.*, 1982; Fernandez & Sheffield, 1996; Martin & Theunissen, 1992) but they may be prone to greater suffering in terms of so-called daily hassles (Fernandez & Sheffield, 1996; Holm, Holroyd, Hursey, & Penzien, 1986).

The above discussion suggests that both the stress-coping hypothesis and the cognitive shift hypothesis are plausible and not necessarily incompatible as has been assumed previously. In other words, from the perspective of a multifactorial CBT framework for chronic headache, a general cognitive shift to disorder-related distress does not rule out the possibility that other sources of psychosocial stress (especially daily hassles) can continue to play a role in aggravating headaches. This combined approach which incorporates both the stress-coping hypothesis and the cognitive shift hypothesis has generally been rejected in the clinical research literature or it has not been made explicit as researchers have felt inclined to support one approach or the other thus dichotomising the debate. The widely assumed dichotomy, unlike the proposed combined theory, is at odds with the consensus that both general stress-management and specific cognitive strategies for pain coping seem very relevant and effective in CBT treatment for chronic headache.

The debate on whether stress arises from psychosocial factors or whether it is a reaction to the chronic headache disorder itself has also tended to overshadow a potentially more fruitful avenue for investigation: the role of appraisal in mediating the stress response in headache sufferers. The observation that the impact or source of stress was variable within individuals over time as well as between individuals is consistent with the theory that stress is a process that is mediated by appraisal of threats as well as coping resources (Lazarus & Folkman, 1984; Marlowe, 1998b).

Although the role of appraisal in the stress-headache link has been widely accepted by researchers the focus of research has tended to be based on the stress-coping hypothesis. Again, a more integrated approach would require consideration of appraisal processes relating not just to psychosocial stress but also to headache-related distress. This might explain some of the inter- and intra- individual variability in the impact of stress with respect to CDH: e.g. the observation in the current research that some people reported having no life-stress yet significant headache distress whereas others were able to clearly identify sources of psychosocial stress that aggravated their headaches.

7.4.5 An Integrated CBT Model of Chronic Headache

Figure 7.2 shows a modified CBT model of chronic headache that attempts to integrate the various findings discussed above with existing theory. The right hand side of the diagram is exactly the same as Figure 7.1 (which was discussed above) and represents presumed cognitive processes. The left-hand side of the diagram shows how headaches might develop and how they might be maintained. The main tenet of the model is that cognitive processes are active at all times and interact with headache development and maintenance at all stages. Whilst the idea of reciprocal causation is not new, the model proposed here attempts to clarify the role of cognition in CBT without getting entangled in the tricky question of temporal precedence of cognition. In fact, it could be argued that temporal precedence is irrelevant and it is the strength and balance of different processes that are important. This view is consistent with a biopsychosocial framework.

A further important suggestion that is apparent from the model is that cognitive processes, as well as being central to the overall relationships between different variables, can also be part of headache symptomatology. This is similar to the notion of meta-cognition that has been proposed in anxiety models. For example, a thought such as: " I don't think I can cope with this headache." may be a seen as cognitive symptom but also feeds into the overall cognitive process.

Predisposing and vulnerability factors refer to the role of such things as genetics, hormones and personality that might play an important part in the onset of headache and that might continue to make the sufferer vulnerable. For example, a high level of trait anxiety might lead to heightened daily sensitivity to many situations which may then manifest itself in the form of daily tension headaches. Lifestyle, precipitants and specific triggers are presumed to be interrelated. For example, a lifestyle that involves considerable VDU work or significant marital tension may precipitate headaches and specific headaches are then set-off in the presence of triggers which themselves might be related or unrelated to the precipitants (e.g. children fighting, missed sleep, dietary triggers). Headache symptoms and processes refer to the actual headache. These consist of a mixture of phenomena that can be described according to their physical (e.g. sensory pain, visual disturbances etc.), affective (e.g. irritability), behavioural

(e.g. screaming in agony), and cognitive (e.g. catastrophising and increased attention to pain site) characteristics. Maintaining factors for the immediate headache might be the same as the phenomena just described, therefore fuelling a vicious circle (e.g. catastrophising might lead to greater tension and increased pain sensitivity). Maintaining factors over the longer term might include persistent depression and avoidance of activity, which then feed into a lifestyle that maintains vulnerability to further headaches.

The model suggested here is intended to be a working model that draws together established ideas from CBT theory and a number of specific theories concerning cognitive processes that are presumed to operate in the context of chronic headaches and pain. Whilst the current findings give some basis to the model, further empirical investigation of the putative constructs and processes is required. However, it is asserted that this model can help researchers and clinicians to be more explicit about the rationale for using CBT treatment approaches with chronic headache and pain sufferers – a rationale which has often been poorly articulated or has been omitted altogether.

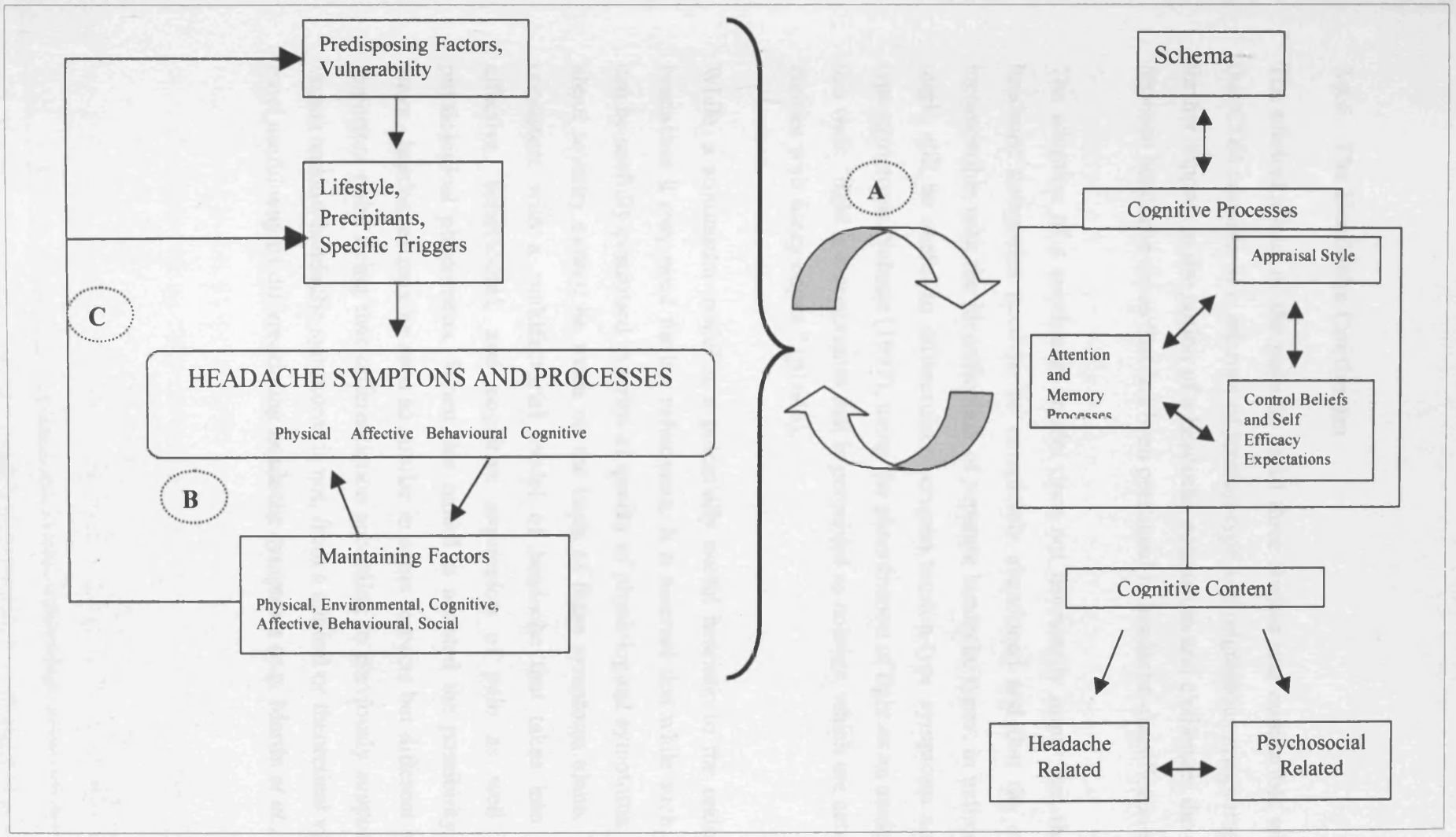


Figure 7.2 **A CBT model of chronic headache.** Process A represents the role of and interaction with cognitive processes at all levels. Process B represents the maintenance of an existing headache. Process C represents maintenance of headaches over the longer term.

7.4.6 The Headache Continuum

The clinical status of the patients in all three studies was compatible with the view that CDH consists of a mixture of tension-type and migrainous symptoms. This lends further support to the notion of a headache continuum and challenges the demarcation between headache types that has been presumed in headache classification systems.

The adoption of a continuum model does not necessarily imply that the traditional headache categories need to be completely abandoned and that the continuum is incompatible with the identification of separate headache types; in individual cases it might still be useful to differentiate between tension-type symptoms and migraine-type symptoms. Schade (1997), using the phenomenon of light as an analogy, explains this well: "light is a continuum that is perceived as colours, which are actually distinct entities with fuzzy edges." (p. 648).

While, a continuum model is a potentially useful heuristic to the understanding of headaches it may need further refinement. It is asserted that while such a continuum can be usefully construed in terms of quality of physiological symptoms, assumptions about severity cannot be made on the basis of these symptoms alone. This view is consistent with a multifactorial model of headache that takes into account the affective, behavioural, and cognitive expression of pain as well as sensory-physiological phenomena. When this model is adopted the possibility works both ways - headaches may be seen as similar in some respects but different in others; the important point being that differentiation according to previously supposed migraine versus tension-headache symptoms is not, from a clinical or theoretical viewpoint, the most useful way of differentiating headache symptoms (e.g. Martin *et al.*, 1992).

7.5 Clinical Implications

7.5.1 Assessment Considerations

Whilst there might be a temptation to rely solely on the headache diary, as has tended to be the case in much clinical research, the current findings underline the need to take a more multidimensional approach in two respects. Firstly, clinical assessment should at least attempt to cover the broad areas of cognition, affect, behaviour and functional aspects of chronic headaches rather than focusing on just those variables relating to the sensory component of pain (e.g. intensity, frequency, duration). Secondly, a more sophisticated assessment should consider sub-constructs within these major variables. For example, assessment of cognition might be split into information about self-efficacy and information about appraisal style. Such detailed information may allow therapists to identify the key aetiological and maintaining factors more clearly and therefore enable better treatment tailoring.

A further consideration that is relevant to the use of assessment scales relates to the dimensions on which patients are asked to provide ratings. A measure such as intensity of pain might appear to be an easily interpretable unidimensional measure but patients may interpret intensity in different ways. This problem may be more marked in scaling affective, cognitive and behavioural measures, each of which might be interpreted along a number of dimensions simultaneously (e.g. the level of distress and attentional focus). This possibility of multidimensional scaling, whilst confounding the information from existing scales, could be harnessed to allow more sensitive assessment (Morley, 1989; Morley & Pallin, 1995). However, until standardised scales with greater sensitivity are developed, and from an immediate practical point of view, the recommendation is that patients' ratings on existing self-report scales (e.g. for affect) should be more carefully evaluated in individual cases: a number of dimensions rather than just the one assumed might be evident.

Apart from the general implications discussed above, the current research also leads to a number of specific recommendations regarding assessment: (a) Whilst information concerning different headache types should continue to be assessed, such

information should be seen as adding to the overall patient profile rather than as a central component of the assessment. This is based on support for a biopsychosocial and continuum model of chronic headache in which it is the interplay of different psychosocial and physiological variables and not headache categories per se, which determines headache symptoms and coping; (b) The utility of the daily headache diary was supported. Studies 1 and 2, whilst employing the diary for four weeks per treatment phase supported previous empirical research that has shown that even a two-week period yields reliable information. Study 3, on the other hand employed the headache diary continuously throughout the treatment programme and it is possible that for some patients, continuous monitoring acts as an incentive which strengthens their engagement in self-management treatment; (c) The inclusion of a measure for medication-free days seems to be useful for those who take frequent analgesics but does not add anything in the assessment of other patients; (d) The Headache-Specific Self-Efficacy Scale and the Pain-Related Self-Statements Scale were found to be reasonably sensitive to changes on the type of cognitive targets that are part of CBT interventions. Their use might be enhanced if clinical norms are developed. In addition, it seems well worth measuring self-efficacy on a daily basis as this yields useful information about the stability of self-efficacy beliefs and their association with ongoing treatment goals in different patients. Therefore, treatment could be modified accordingly; (e) The current research did not employ detailed measures of pain behaviour or used other information sources (e.g. spouse reports) but it is possible that these could enhance assessment of headaches.

7.5.2 Treatment and Service Implications

The major implication of the finding that minimal-contact CBT is just as effective as therapy-intensive CBT concerns the potential to enhance cost-effectiveness. As MC-CBT requires less professional time, this has potential benefits for the therapist as well as the service. For the therapist, advantages include having more time for other professional duties, feeling less 'burnt-out' by intensive clinical work, and being able to deliver the components of treatment in a consistent and clear manner through a detailed treatment manual. For the service, the benefits include the ability to treat more patients concurrently thus reducing waiting lists, potential cost-savings if additional therapists are considered to be unnecessary, and the opportunity to devote

more resources to other aspects of service development and provision. It is also possible that MC-CBT treatment could be administered by support clinical staff (e.g. Assistant Psychologists in the UK) under the close supervision of clinical psychologists, thus leading to further resource benefits.

MC-CBT also has potential benefits for the patient. The combination of sessions with the therapist at key stages together with the use of a step-wise manual that patients can keep, means that the whole treatment process is less of a mystery and patients have ready access to detailed written instructions for managing headaches. This can enhance self-efficacy and reduce feelings of hopelessness and powerlessness. Whilst the ethos of CBT treatments generally has been to cultivate an atmosphere of openness and collaborative empiricism (Beck *et al.*, 1979), MC-CBT takes this a step further by encouraging even greater participation from the patient. This could also help to break down any barriers that patients perceive between themselves and therapists. Additionally, a rather ironic possibility is that, as therapist contact is minimal in MC-CBT, more attention might be paid to all therapy processes by both the therapist and the patient and less is taken for granted. This minimises the 'that can wait till next week' factor and ensures that both therapist and patient work hard to make the most of treatment.

Whilst a lot of the content and several treatment strategies relate to chronic headache and pain, the MC-CBT treatment developed here could be fairly easily adapted to other clinical applications within clinical health psychology. The relevance of the biopsychosocial framework and the importance of cognitive strategies have already been demonstrated in clinical research pertaining to a number of other disorders (Nicassio & Smith, 1995; Sarafino, 1998).

Although, the overall conclusion is that MC-CBT has many advantages, there are also some potential pitfalls. Firstly, the largely self-management format, guided by a treatment manual, is likely to require patients who are both well motivated to engage and also intelligent enough to make sense of the detailed written instructions. Patients who do not meet these criteria are unlikely to adhere to treatment and may be more suitable for conventional CBT treatment with regular sessions.

Secondly, a manualised treatment format might be too rigidly applied and individuals' unique requirements may be neglected. However, mitigating against this possibility, the MC-CBT treatment developed here includes plenty of flexibility, within the overall CBT framework, to allow tailoring of treatment to individual requirements. A related concern is that headache patients for whom there are considerable unresolved issues (e.g. sexual abuse or prolonged grief) and which impact on their overall health status would clearly require a conventional regular therapy approach with treatment being initially focused on the problems that are seen to be priority by patient and therapist. An alternative solution would be to improve patient screening so that patients who present with more complex psychological problems are referred to the appropriate psychiatric service. However, the researcher's clinical experience is that patients who have primarily presented within a medical psychology service tend to resist referral to a psychiatric service (possibly due to a combination of perceived stigma and a practical fear that their medical consultant may not want to see them again).

Thirdly, a criticism of a minimal-contact approach is that it does not make use of the therapist's full repertoire of skills thus eroding the richness of the therapist's training and it neglects important process issues that might develop in the course of conventional therapy. This criticism can be answered by considering two issues: (a) Minimal-contact treatments (including the one suggested here) are not intended to completely replace conventional therapies – rather, they are intended to offer a further cost-effective treatment option which might be suitable for some patients and certain conditions only; (b) The more fundamental issue, that might seem rather threatening to a profession such as clinical psychology, is raised by the following question: does the presumed richness of conventional therapy matter if the treatment outcome is the same from minimal-contact treatment? The assumption underlying a critique of MC-CBT treatment is that such treatment is mechanistic and devoid of the multitude of processes which are presumed to occur in the course of regular therapy. However, it can be asserted that MC-CBT involves the same amount of importance being placed on such things as the therapeutic relationship, and that it merely distils out non-essential contact in a select group of patients.

7.6 Methodological Limitations

Whilst it is asserted that the current findings add considerably to the understanding of the efficacy and mechanisms of minimal-contact CBT treatment of chronic headaches some caution is warranted in the light of a number of methodological criticisms. These criticisms pertain to epistemology, sampling, research design, measures, and analyses.

7.6.1 Epistemology

A possible criticism of this research, which has also been levelled at process type research generally, pertains to the reliance on quantitative methodology when perhaps a qualitative approach might yield richer information. However, a quantitative approach to the research programme was adopted for a number of compelling reasons. Firstly, the primary focus of this research programme was a number of *a-priori* predictions concerning treatment outcome, treatment change, and headache coping. These predictions, while being informed by clinical practice, were derived from previous empirical research and theory. Secondly, in order to test the predictions, controlled experimental manipulation together with some correlational analysis was strongly felt to be necessary. Thirdly, the existence of a number of validated measurement tools meant that the predictions could be operationally defined and the effects could be quantified while maintaining ecological validity.

7.6.2 Sampling

An ostensibly obvious limitation in both Studies 1 and 2 is the relatively small sample size, which meant that there were a modest number of participants per treatment condition. This raises questions about the generalisability of the results and the power of the experimental designs in both studies (i.e. the probability of finding a difference between treatment groups when such a difference does exist in reality). However, consideration of a number of relevant observations and arguments suggests that the modest sample sizes reported here do not limit the findings in a major way.

Firstly, relative to conventions suggested in the literature (e.g. Cohen, 1988), the effect sizes found here were mostly quite large (upwards of $\delta = 1$). Sample sizes required for between-group designs with these effect sizes and with the requirement that power is maintained at a level of at least 80%, are below 40 (Howell, 1997; Aron & Aron, 1999). In fact post-hoc power calculations showed that well over 80% power was achieved on the majority of statistical tests. These observations indicate that the possibility of Type II error was minimised despite the modest number of participants.

Secondly, the strength of the findings are also enhanced when one considers that participants came from a reasonably homogeneous population (CDH sufferers). Statistically, this has the advantage of reducing the standard deviation thus increasing power. However, the disadvantage is that the results may not generalise to a wider headache population. In the literature on chronic headache treatment, effect sizes are generally large ($\delta > 0.5$) in terms of Cohen's (1988) conventions but smaller than those reported here. This discrepancy adds support to the presumption that the CDH population studied here is less diverse (on headache and psychosocial parameters) than other chronic headache populations.

Thirdly, the use of standardised measures meant that pre-intervention variability was kept to a minimum and that treatment effects could be more clearly discerned. This again is likely to have contributed to increasing the power of the studies.

7.6.3 Design

A major criticism of the design of Study 2 is the lack of a no-treatment control group. This was mainly due to a combination of referrals slowing down and a third of referred patients declining to participate in the study. Therefore, in order to retain reasonable numbers per treatment condition it was decided that the study would consist of two experimental groups only. In some respects, however, the no-treatment control condition was not felt to be vital in Study 2 as the main focus was to compare two minimal-contact treatments of equal length which differed only in the presence or absence of the cognitive component.

Although a no-treatment control group was included in Study 1 this group was not followed-up at six months as it was considered that a further wait for treatment was unethical. The main limitation of this is that a longer-term comparison with the treatment groups was not possible. Showing that a no-treatment group does not tend to improve over the longer-term would have strengthened the findings regarding treatment efficacy.

7.6.4 Measures

Although a possible strength of the series of studies reported here is that instruments were carefully chosen, with reliability, validity, and practicality in mind, a number of limitations are worth discussing, particularly with regard to the headache diary.

It has often been cited in the literature that a strength of the headache diary is that it can yield information about frequency, intensity and duration of headaches in one instrument. Most researchers do not dispute the utility of the headache diary in providing reasonably reliable information about these headache parameters (Blanchard, 1992; Martin, 1993). However, a number of limitations need to be mentioned.

Firstly, compliance in completing the diary prospectively may be poor and patients may rate several of the scores retrospectively at the end of the day or even after several days. The requirement to rate headaches just four times per day at roughly equal intervals - waking, lunchtime, teatime, and bedtime - is assumed to be an easy task for the rater but Collins and Thompson (1979) found that 40% of a student sample were non-compliant. On the other hand, it is possible that a clinical sample would be more motivated to rate accurately.

Secondly, the six-point rating scale is anchored by behavioural referents, e.g. a rating of 4 is anchored by: 'severe headache...can do undemanding tasks'. There are two problems with this: (a) patients could be inconsistent across time in how they interpret the verbal descriptors and such interpretation may itself be pain-dependent; and (b) it cannot be assumed that the perceived sensory intensity of pain has a simple linear relationship to pain behaviour (Morley, 1986; Philips & Jahanshahi, 1985).

Thirdly, the headache diary is, strictly, a time-sampling procedure, therefore, assumptions about the frequency and duration of headaches may not always hold. Very brief headaches that last a couple of hours may be missed in the six-hourly intervals, or continuous ratings may suggest several headaches rather than the duration of one headache. On the other hand, clinical and empirically supported knowledge about the phenomenology of chronic headaches supports the assumption that consecutive ratings greater than zero are likely to signify headache duration and consecutive periods with a zero rating are likely to signify headache-free intervals (Blanchard & Andrasik, 1985).

In terms of other measures, an obvious limitation of the current research is that pain behaviour measures besides medication intake were not used. Physiological measurements were also omitted as it was considered that such information was not essential to answering the key questions posed by this research programme. However, if a multidimensional model of chronic headache is assumed then proper evaluation of treatment effects and mechanisms would require a thorough assessments on all dimensions. The problem with such a thorough approach is two-fold. First, the full range of assessment procedures and instruments may not be readily available to the researcher. Second, and more important, bombarding the research participants with numerous assessment instruments could be unethical as well as being impractical. Such an approach is also likely to discourage participation and increase the level of incomplete or spoilt data. In fact, in the light of the problems just discussed, the current research programme could be criticised for using too many rather than too few measures.

7.6.5 Analyses

The separation of dependent measures meant that several statistical tests were conducted for a given hypothesis. It is possible that this increased the likelihood of Type I error as some tests may have shown a significant result by chance. One solution to this might have been to collate the effects on some of the dependent measures through a multivariate analysis of variance (MANOVA). The researcher opted not to undertake MANOVAs as a central aspect of the research was to show the

separate effects of treatment on a wider range of measures than have been used in previous empirical research.

However, when considering planned comparisons between pairs of treatment effects Type I error was minimised by dividing the conventional alpha level of $p < .05$ by the number of comparisons. Although, a more stringent alpha level increases the likelihood of Type II error, it was found that, in the vast majority of computations, the conclusion would not have altered had alpha been set at $p < .05$ (the SPSS statistical software package can compute exact probability levels).

The limitation of correlational analyses with regard to establishing causality has already been discussed.

7.7 Future Directions

Although, current findings support the efficacy of MC-CBT treatment for CDH and highlight the importance of the mediating role of cognitive change a number of important areas merit further empirical research.

With regard to identifying the mechanism of treatment change, future research could build on the tentative suggestions emerging from correlational analyses by paying closer attention to causal relationships. Given the difficulties in manipulating purported causal mechanisms (such as self-efficacy) in a clinical setting, it may not be possible to set up true experimental designs to test these. An alternative would be to address causality through structural equation modelling (SEM, Tabachanick & Fidell, 1996). This requires one or more clear theoretical models of the processes involved in therapeutic change, as well as regular measurement of each conceptual variable. SEM then allows one to infer causality by examining sets of relationships between pre-treatment variables, hypothesised cognitive changes, and outcome measures. However, it should be noted that no matter how elegant the mathematical model is, attributing causality is still ultimately based on an interpretation of scientific theory.

Whilst the investigation of causality in treatment has become a sort of holy grail, it is worth considering a further point if future clinical research is to contribute usefully to

an understanding of treatment mechanisms and coping processes. This is that, given evidence supporting a biopsychosocial framework in which several variables are presumed to interact reciprocally, is it really feasible or useful to establish linear causality? This is akin to asking as where the circle starts. A more fruitful paradigm for future research might be to investigate key processes within this complex circular framework and how the influence of these might vary across patients and over time. This should add to an understanding of multicomponent CBT treatment, for example, by providing information about how different strategies work for different people.

In terms of specific cognitive variables, there remain a number of questions. Firstly, the relationship between hypothesised cognitive variables such as perceived self-efficacy and catastrophising needs to be clarified. For example: is one of these variables more influential in the treatment mechanism? Future research might also benefit from drawing on theoretical developments in related areas of psychology. An exciting development in recent years has been the attempts that have been made to synthesise ideas from different fields within psychology to form more integrated explanatory models (e.g. the role of attention and memory processes in pain and emotion: Eccleston, 1994, 1995; Morley, 1993; Williams, Watts, Macleod and Mathews, 1997). While this is a welcome trend, there is still much work to be done, and the literature on chronic headache and cognition still appears disordered and confusing.

Secondly, with regard to the self-efficacy construct, it seems that a closer empirical evaluation is warranted to test Bandura's (1977, 1997) consistent assertion that self-efficacy rather than outcome-efficacy is the main determinant of behaviour. A further avenue of investigation relates to the subject of self-efficacy appraisals and how global these are. The current research focused on the possibility that treatment works through change in perceived self-efficacy for headache management strategies but did not consider the extent to which other specific or global self-efficacy beliefs might change and the extent to which these are also important in the treatment mechanism. For example, high perceived self-efficacy for headache management might really be based on high self-efficacy for managing stress when headache-free, whereas self-efficacy for specific pain control strategies during a headache may still be very low. In addition, the current research indicates that it may be worth measuring and

investigating the impact of the stability of self-efficacy beliefs. Daily fluctuations may be superimposed on a more stable pattern. Apart from theoretical implications with regard to the self-efficacy construct, the issue of stability is important from a clinical perspective as it might relate to the consistency with which specific treatment strategies are practised and the amount of effort expended in these.

Thirdly, future research should investigate whether meaningful treatment change in CBT always requires explicit strategies to induce deeper cognitive change at schema level or whether it is sufficient to induce changes in cognitive processes at a surface level. In other words, should CBT treatment take a 'top-down' approach (i.e. which assumes that schema change should gradually occur automatically if changes in cognitive processes are sustained) or a 'bottom-up' approach (i.e. which assumes that cognitive change is dependent on a-priori changes at the schema level)? This has implications with regard to the sequencing of treatment strategies and whether explicit strategies to induce schema change (e.g. challenging dysfunctional assumptions) should be included in the treatment package.

Based on emerging empirical findings, cognitive processes have been assumed to be at the heart of the treatment mechanism in CBT and other treatments. However, given the relative newness of this area of investigation, further empirical research and replication is clearly needed, especially concerning the influence of non-cognitive factors (e.g. amount of practise with treatment strategies).

In terms of the relative efficacy of MC-CBT and I-CBT, an important clinical consideration that needs to be addressed in future research relates to the matching of treatment to patients. The suggestion is that MC-CBT will be more effective for patients with a reasonable level of intelligence, who are not severely depressed, are well-motivated to change and have a propensity towards self-management whereas I-CBT might be more effective for patients who do not meet these criteria. It is also possible that other variables such as level of family support, type of headache symptoms and experience of the therapist, might be important in determining who benefits from MC-CBT and who benefits from I-CBT. These considerations are also relevant to the question of treatment mechanisms. Finally, it seems that MC-CBT treatment in a group format would offer further opportunities to increase cost-

effectiveness. However, further empirical research would be required to establish the efficacy of such treatment and investigate how the treatment processes compare to those that have been invoked in the context of individual MC-CBT.

7.8 General Conclusions

This is the first study to evaluate a minimal-contact CBT approach for patients who present with chronic daily headache. The overriding conclusion is that while CDH is a challenging headache disorder that transcends the traditional boundaries of migraine and tension headache, MC-CBT treatment is as effective as therapy-intensive CBT in bringing about meaningful improvement for sufferers. This finding is in contrast to the tone of resignation in previous empirical research and in received clinical wisdom, which has assumed that CDH patients respond very poorly to self-management psychological treatment.

MC-CBT holds a lot of promise, not just in headache treatment but in other chronic pain and chronic illness conditions. In an age when the provision and cost of health services are ever greater concerns amongst clinicians and policy-makers, treatments such as MC-CBT are likely to receive increased attention. However, the assertion made here is that, such developments need not be governed solely by issues pertaining to cost-effectiveness. From both a therapeutic and theoretical viewpoint, the development of MC-CBT offers potentially fruitful avenues for further investigation. For example, in some patients, MC-CBT may discourage dependency on the therapist, and the CBT goal of inducing positive cognitive change might not always require intensive therapy as previously assumed. The challenge for future researchers is to determine the client, therapist and process variables that predict good response to MC-CBT.

The current research addressed the important area of therapeutic process through investigating and attempting to make explicit, the role that cognition plays in CBT treatment of chronic headache. It appears that, as assumed by the general CBT model, appraisal style (particularly degree of catastrophising) may have some role to play in the treatment mechanism. However, other cognitive variables such as self-efficacy

beliefs and health locus of control appear to exert their own influence on headache coping and use of treatment strategies. While this latter assertion has been the subject of much empirical research, little attempt has been made to integrate the ideas and findings with the CBT model. Based on the current findings as well as the emerging empirical work looking at the role of information processing theory in health disorders, this thesis has attempted to present an integrated CBT model. This model retains the notion that cognition is central to the explanation of emotional, behavioural and physiological changes and that such cognition occurs at a number of levels. However, it is posited that cognitive processing primarily involves an interaction between the hypothesised schema and the following three grouped components: information processing, appraisal style, and self-efficacy expectancies and control beliefs. It is suggested that this dynamic interplay between the different components of cognition might be a more fruitful subject of investigation, from the point of view of understanding the CBT model, than merely taking a unidimensional approach to cognition.

These conclusions must be seen as tentative due to a number of reasons. Firstly, in view of several methodological limitations (e.g. modest sample sizes), further replication is clearly required if firmer conclusions are to be drawn about treatment efficacy and mechanisms. Secondly, it seems important to investigate the efficacy and mechanisms of MC-CBT in other clinical applications. Thirdly, while the primacy of cognition in the CBT model seems to be easily construed at a conceptual level, its importance is less clear from an empirical perspective. The current findings support a more multifactorial model in which, besides cognition, many physiological, behavioural, affective, and environmental variables also play a role: sometimes the role of cognition may not be crucial. Finally, in the context of CBT and adjustment to health dysfunction, the whole study of cognition is peppered with constructs that seem to defy logical ordering and precise definition, let alone empirical validation. Until there emerges a clearer scientific map and a more consistent language, then any cognitive model remains open to the accusation that it is merely another addition to a conceptual free-for-all.

Appendices

Appendix A

Ethical Approval

(Presented on next page)

Melanie Sursham
Direct Dial 0116 2588610
07 November 1995

Health for Leicestershire
LEICESTERSHIRE HEALTH
Gwendolen Road, Leicester LE5 4QF
Tel: (0116) 273 1173 Fax: (0116) 258 8577

Mr A Laher
Department of Medical Psychology
Hadley House
Leicester General Hospital

Dear Mr Laher

A cognitive functional approach to the treatment of chronic frequently-recurrent headaches and the role of cognitive changes: A controlled evaluation - our ref. no.4000

Further to your application dated 25 October, you will be pleased to know that the Leicestershire Ethics Committee at its meeting held on the 3 November, 1995 approved your request to undertake the above-mentioned research conditional upon it being noted that patients should have received further details of the research and you should have checked that they understand what the research is about and answer any questions **before** they sign the consent form.

I would remind you, however, that your research project has been given approval only in relation to its acceptability from an ethical point of view. If, subsequently, departure from the methodology outlined in your protocol is contemplated, the Ethics Committee must be advised in order that the proposed changes may be approved. Also a report should be made to the Ethics Committee if any significant adverse reactions are noted during the course of the study. In addition, any NHS resource implications of your project must be discussed with the appropriate Trust Chief Executive. Similarly, it may be that the research project has implications for other disciplines and, if so, you are advised to discuss them with the appropriate departmental manager. Researchers should also be able to assure the Ethics Committee that satisfactory arrangements have been made for the labelling, safe storage and dispensation of drugs and pharmaceutical staff are always willing to provide advice on this.

Researchers' attention is also drawn to correspondence from the Regional Director of Public Health dated 28 January, 1991 relating to Clinical Trials which sets out revision of the procedures to be followed, and the Clinical Trials Indemnity Letter and Deed of Guarantee. Researchers should ensure that these indemnity arrangements have been complied with.

Researchers intending to study selective groups of patients in the community are reminded that their first approach should be to the individual patient's general practitioner to ascertain whether the particular patient was suitable for inclusion in the study. Equally, when the researcher contacts the patient it should be emphasised that the approach is made with the knowledge of the General Practitioner, with whom the patient may discuss this research, if the patient so wished.

Yours sincerely

M. Sursham

for **Director of Public Health**

Appendix B

Headache Classifications of the International Headache Society's Headache Classification Committee

Migraine and tension-type headache

1 Migraine
- 1.1 Migraine without aura
- 1.2 Migraine with aura
  - 1.2.1 Migraine with typical aura
  - 1.2.2 Migraine with prolonged aura
  - 1.2.3 Familial hemiplegic migraine
  - 1.2.4 Basilar migraine
  - 1.2.5 Migraine aura without headache
  - 1.2.6 Migraine with acute onset aura
- 1.3 Opthalmoplegic migraine
- 1.4 Retinal migraine
- 1.5 Childhood periodic syndromes that may be precursor to or associated with migraine
  - 1.5.1 Benign paroxysmal vertigo of childhood
  - 1.5.2 Alternating hemiplegia of childhood
- 1.6 Complications of migraine
  - 1.6.1 Status migrainosus
  - 1.6.2 Migrainous infarction
- 1.7 Migrainous disorder not fulfilling above criteria

2 Tension-type headache
- 2.1 Episodic tension-type headache
  - 2.1.1 Episodic tension-type headache associated with disorder of pericranial muscles
  - 2.1.2 Episodic tension-type headache unassociated with disorder of pericranial muscles
- 2.2 Chronic tension-type headache
  - 2.2.1 Chronic tension-type headache associated with disorder of pericranial muscles
  - 2.2.2 Chronic tension-type headache unassociated with disorder of pericranial muscles
- 2.3 Headache of the tension-type not fulfilling above criteria

Additional Classifications

3 Cluster headache and chronic paroxysmal hemicrania
4 Miscellaneous headaches unassociated with structural lesion
5 Headache associated with head trauma
6 Headache associated with vascular disorders
7 Headache associated with nonvascular intracranial disorders
8 Headache associated with substances or their withdrawal
9 Headache associated with noncephalic infection
10 Headache associated with metabolic disorder
11 Headache or facial pain associated with disorder of the cranium, neck, eyes, ears, sinuses, teeth, mouth, or other facial or cranial structures
12 Cranial neuralgias, nerve trunk pain and deafferentiation pain

Source: International Headache Society (1988)

Appendix C-1

Information Leaflet

(Presented from next page onwards)

(Note. This is the version used for Study 1. It was amended slightly for both Studies 2 and 3 so that it appropriately reflected the procedures and therapists involved in those studies)

INFORMATION LEAFLET

A RESEARCH STUDY LOOKING AT THE CONTRIBUTION OF PSYCHOLOGICAL TREATMENT FOR RECURRENT HEADACHES

What is the research about?

The research looks at how, and to what extent, psychological treatment might help to lessen the overall distress and pain which you experience through living with your headaches.

Why is psychological treatment relevant?

Previous research and treatment experience has shown that psychological treatment can be effective in helping some headache sufferers to manage their difficulties more effectively and to gain confidence in controlling their symptoms. Looking at psychological factors does not mean that your headaches are seen to be 'all in the mind'. Psychological treatment is simply aimed at helping you to manage your overall headache experience through addressing the complex relationship between physical symptoms, feelings, thoughts and behaviour.

Am I suitable for inclusion?

If you suffer from headaches (however mild) for three or more days per week on average, this research study is likely to be of interest to you. Your headaches may be migraine or tension-type or a mixture of these. If you are unsure of your suitability to join the study but are interested please get in touch with us. Your headaches do not have to be obviously related to "stress" for you to join this research trial.

What will the treatment involve?

The treatment involves a combination of established talking therapy and home-based guided practice of headache management strategies. These strategies will include the broad areas of pain control, stress management, relaxation training, activity planning, diet and exercise. Most of the treatment procedures that are used in this study are based on coping skills which you might already be using to some extent, in your everyday life. Treatment will be aimed at helping you to build on these together with providing you with the opportunity to safely explore any difficulties and to learn further things that might be helpful to you. You will be offered the same high standard of professional care that is given to on-going patients.

You will be asked to monitor your headaches through a simple headache diary before, during and after treatment (for a short time). In addition you will also be asked to complete some simple questionnaires about your headaches.

You may be put on a short waiting list before treatment begins. if this happens you will be requested to continue filling your headache diary as this forms an important aspect of your overall participation in the research.

How long will treatment last and where will it take place?

Treatment will involve no more than 8 sessions of therapy over a 12-week period. Each session will be about 1 hour long. You will be asked to complete some simple 'follow-up' information about your headaches six months after your treatment (this will be done through the mail).

Sessions will usually be held at either the Department of Medical Psychology, Leicester General Hospital, or at Knighton Street Outpatients Department, Leicester Royal Infirmary depending on whichever is convenient for you. In some cases we may be able to visit you at home.

Will I have to give-up my medication?

Psychological treatment generally compliments medical treatment. Therefore, you do not have to stop any existing medication which you are taking for your headaches. However, we will be unable to include you in the study if you have just started a new medication. If you are able to postpone the start of any new medication until you have completed your participation in the study this will be very helpful.

Who will conduct the research?

The research will be undertaken by Mr Aftab Laher, Chartered Clinical Psychologist and Camilla Watters, Assistant Psychologist, both of whom work in the Department of Medical Psychology, Leicester General Hospital.

Dr Alan Sunderland, Lecturer in Clinical Psychology, University of Leicester will supervise and monitor the overall research. Dr Chris Cordle, Head of Department of Medical Psychology, Leicester General Hospital, will supervise clinical work. The research also has the support of Dr Richard Abbott, Consultant Neurologist, Leicester Royal Infirmary and Dr Brian Kendall, General Practitioner and Clinical Assistant in Neurology, Leicester Royal Infirmary.

How do I join the headache study?

You will normally be offered a referral to us by a doctor at the Neurology or Migraine Clinic. The doctor will send a written referral directly to us. We will then write to invite you to a preliminary assessment (if you do not hear anything within three weeks then please contact us). Please bring the Consent Form along with you to your preliminary assessment.

If you have seen this Information Leaflet first or if you have heard about this Study from elsewhere, and you are interested in joining, please get in touch with us. If appropriate, we will arrange for you to be formally referred to the Study, through the Consultant who has been responsible for your treatment.

If you require further information about this research study then please contact AFTAB LAHER or CAMILLA WATTERS at The Department of Medical Psychology, Hadley House, Leicester General Hospital, Gwendolen Road, Leicester, LE5 4PW. Telephone (0116) 2584958 (direct line). Any queries will be happily received.

CONFIDENTIALITY AND YOUR RIGHTS

(1) You will be treated with the utmost respect and dignity and will be offered the same high standard of professional care that is given to on-going patients.

(2) You are completely free to opt out of the research at any point should you change your mind.

(3) Your GP will need to be informed that you are taking part in this research. Apart from this requirement, information which you give will be kept strictly confidential and will never be divulged to other professionals without your prior consent. At the end of the information gathering part of the research and prior to writing up the findings all information which identifies actual individuals (eg names and addresses) will be destroyed.

(4) You will never have to do anything against your will during the research study.

(5) The treatment involves talking therapy and relaxation. Throughout the treatment you will be in control and will be fully aware of the rationale for strategies used. The treatment does not involve medication, intravenous substances or anything that would put you in physical danger.

To take part in the research you need to:

a) Have given your written consent and have attended a preliminary session.

b) Have recurrent headache which has been Neurologist .

c) Be 18 - 65 years of age inclusive.

d) Have at least three days with headache (however mild) per week, on average.

e) Have had your headaches for at least 6 months.

Appendix C-2

PRIVATE AND CONFIDENTIAL

CONSENT FORM

A Study Looking at the Contribution
of Psychological Treatment for Recurrent Headaches

Please complete either Section 1 or Section 2 as appropriate. If you are definitely not interested in taking part please destroy this Form.

SECTION 1

I have read the Information Leaflet carefully and am satisfied that I understand what the research is about. I wish to take part in the research. I understand that I can withdraw my consent at any time during the whole of the research study.

NAME: ..... SIGNATURE: .....DATE:.....

ADDRESS: .....

.....

.....

TELEPHONE NUMBER: .....DATE OF BIRTH: ..... SEX: ...

Number of days with headache per week (on average) .....

SECTION 2

I would like further information before I decide whether I want to take part in the research.

NAME: .....

ADDRESS: .....

.....

.....

TELEPHONE NUMBER: .....

Please return to: Mr Aftab Laher
Chartered Clinical Psychologist
Department of Medical Psychology
Hadley House
Leicester General Hospital
Gwendolen Road
LEICESTER LE5 4PW (0116) 2584958

Appendix D-1

REFERRAL FORM

CLINICAL PSYCHOLOGY HEADACHE RESEARCH

CONFIDENTIAL

DATE: .....

To: Aftab Laher, Clinical Psychologist, Department of Medical Psychology, Hadley House, Leicester General Hospital. (ext 4958).

INCLUSION CRITERIA

(ALL criteria must be met)	**PLEASE TICK**
Patient has tension headaches or migraine or mixed headaches	....
At least three days with headache per week	....
Headache symptoms for at least last 6 months, continuously	....
Age within 18 - 65 years	....
No new medication initiated in last 8 weeks	....
No other medical investigations/treatment pending for headaches	....
Has been informed (verbally and through Information Leaflet)	....
Patient is agreeable to preliminary assessment by psychologist	....

PATIENT DETAILS

PATIENT ..... **DOB** .....

ADDRESS ..... **SEX** .....

..... **U.N.** .....

.....

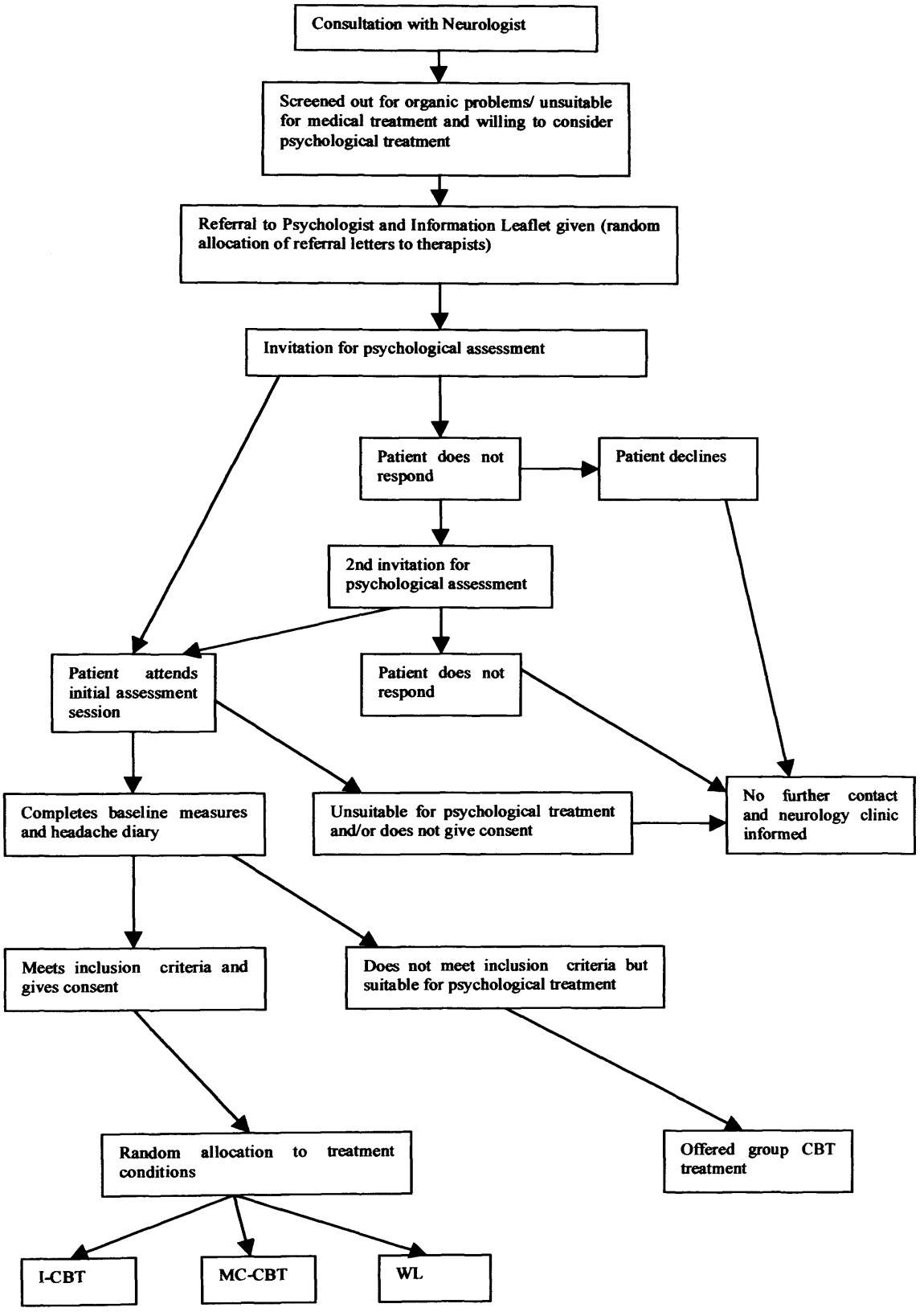
.....

REFERRING AGENT ..... **CONSULTANT** .....

OTHER INFORMATION:

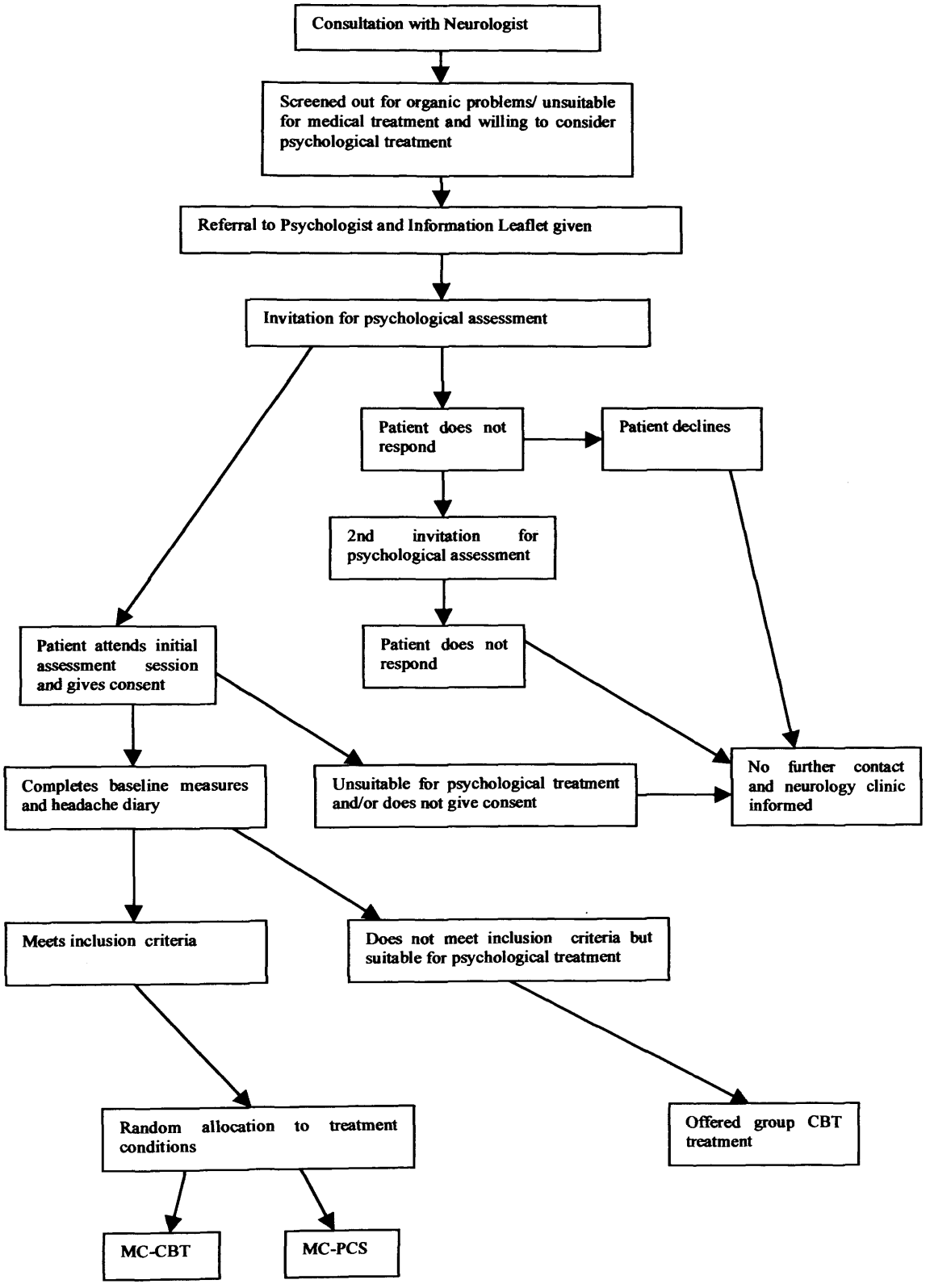
Appendix D-2

Patient Flow in Study 1



Appendix D-3

Patient Flow in Study 2



Appendix E-1

HEADACHE ASSESSMENT

Date	.....
Name	.....
Code	.....
Therapist	.....

Clinical Presentation (mood, appearance, expressiveness etc.)

Brief Description of Symptoms

Headaches

Diagnosis if any (by whom diagnosed and when)

General description of pain (intensity, quality)

Location

Duration

Frequency

Any particular time pattern

Variability of headaches in terms of above, over the last six months?

Detailed Description of Symptoms

	BEFORE	DURING	AFTER
PHYSICAL			
COGNITIVE			
AFFECTIVE			
BEHAVIOURAL			
SITUATIONAL			
ENVIRONMENTAL			

What makes headaches better? (and how?)

What makes headaches worse? (and how?)

Clinical History

Onset (When did the headaches first begin?)

Precipitants (What initially led to headaches developing?)

Time Course (e.g. Continuous since onset? Intermittent? Longest period without and when/why?)

Have headaches remained the same or have they changed in terms of:

frequency?

duration?

intensity?

type of symptoms?

Reasons for any major changes in the last six months

Treatment

Current (What, when started and how useful?)

Past (What, when and how useful?)

Headache Impact (personal feelings; relationships; activity level; work; social/leisure; sleep; diet; other))

Patient's Beliefs about their Headaches (Causes? Prognosis? etc.)

Psychosocial Background and History

Marital Status (and patient's evaluation of relationship)

Family (children, parents, siblings etc.)

Childhood/upbringing (infancy, school, adolescence, adulthood)

Occupation (career history)

Health (general health/ other illnesses etc.)

Leisure (How is this spent?)

Other Stresses (past or current)

General Expectations of Treatment (and why motivated to join study)

Formulation

Assessment Outcome

Overall Impression/Summary

Suitability (Is treatment likely to be of benefit?)

Motivation , Commitment and Enthusiasm

Appendix E-2

Headache Diary

(see next page)

HEADACHE DIARY

Week Commencing..... Patient Code.....

	DAY													
	1		2		3		4		5		6		7	
	H	M	H	M	H	M	H	M	H	M	H	M	H	M
Waking														
Midday														
Evening														
Bedtime														

Week Commencing.....

	DAY													
	1		2		3		4		5		6		7	
	H	M	H	M	H	M	H	M	H	M	H	M	H	M
Waking														
Midday														
Evening														
Bedtime														

KEY

Please enter a headache intensity rating of 0-5, four times a day in the column marked ***H***, using the scale shown below:

0	no headache
1	very low level pain; aware of it only if focusing
2	headache can be ignored at times
3	painful headache, but can continue work etc.
4	severe headache, makes concentration difficult
5	intense, incapacitating headache

Please enter below, any medication you take for your headaches:

A_____

B_____

C_____

D_____

Use the appropriate letter (e.g. B) to identify the medication you have taken each time and enter this in the column marked ***M***. Please, also, enter the dose each time, (e.g. B, 50 mg).

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