A study into the effectiveness of cognitive behavioural therapy for sufferers of chronic tension type headache.

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Declaration

The research described in this thesis is the unaided work of the author, except where acknowledgement is made by reference. No part of this work has previously been accepted for any other degree, nor is any part of it being concurrently submitted in candidature for another degree.

Simon Pestell

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1 Introduction

Overview

Definitions of pain have varied considerably over time and despite advances in medical technology, notably the advent of sophisticated scanning techniques, general agreement on what pain is and what it means continues to evade clinicians and researchers (Merskey & Bogduk, 1994).

One of the central difficulties in defining pain is that the mechanisms are poorly understood. Although popular and medical opinion attributes a direct lineage between pain and tissue damage, the evidence supporting this association is questionable (Sharp, 1997). A number of recent studies cast doubt on theories that attempt to understand pain as a purely physical phenomenon. Jensen, Brant-Zawadzki, Obuchowski, Modic, Malkasian & Ross (1994) reported that many people with lumbar abnormalities (e.g. disk bulges) did not report back pain. By contrast, Spitzer (1987) reports that many people complaining of back pain do not have any identifiable pathology. Therefore, although no doubt related, the relationship between physical impairment and reports of pain is only moderate (Waddell & Main, 1984).

Overall the evidence in the literature suggests that level of physical pathology is not a clear predictor of the level of pain experience. The literature also describes a high degree of variability in the way that individuals respond to chronic pain (Turk, 1996a). Turner & Romano (1984) noted that not all pain patients suffering from similar level of pain are depressed and disabled to the same degree. Several studies have demonstrated that the association between pain, depression and disability appears to be mediated by other variables; in particular psychosocial variables (Fordyce, 1995; Lackner, Carosella & Feuerstein, 1996; Rudy, Kerns & Turk, 1988; Waddell, Newton, Henderson, Somerville & Main, 1993)

These two strands of evidence taken together suggests that pathological changes alone cannot explain reports of pain, pain-related disability or depression. As a consequence, biomedical interventions targeting pathology alone may only be of limited success in these selective areas and in the overall reduction of the pain experience.

A number of multi-dimensional models have been advanced in an attempt to better account for the perception of pain and its clinical phenomenology. In addition, advances in the understanding of the mechanisms behind pain perception have aided the development of effective treatment protocols, including those relating to psychological treatment. The next section provides a general overview of three of the more well established multi-dimensional models of pain perception.

1.1 Pain Perception

1.1.1 The Gate Control Theory (Melzack & Wall, 1968)

This theory attempts to integrate the role of both physiological and psychological factors in chronic pain. Specifically, this theory advocates that incoming pain messages encounter "nerve gates" in the spinal cord that when open allow the pain messages to access cerebral structures and when closed prevent access.

Several factors may determine the response of the "nerve gates" to pain messages and these include: the intensity of the pain message, competition from other incoming nerve signals and signals from cortical and sub cortical structures which increase or decrease the priority of the pain message. Further to these modulating factors, recent research suggests that the message may be inhibited by endorphin production from the hypothalamus at the sub cortical level. At the cortical level a number of discrete anatomical structures are involved in attaching meaning to the message based on the personal and social context in which the pain is experienced.

The model also advocates the role of descending messages from cortical and sub-cortical structures, which modulate the status of the "nerve gates". In times of emotional dysregulation, descending messages from the brain may amplify the pain signal at the "nerve gate" or alternatively descending messages may "close" the nerve gate, preventing access.

There has been significant debate over the actual physiological details of the model (Nathan, 1976; Price, 1987). However, the Gate Control Theory has led to advances in treatments, including neurophysiologically based treatments (North, 1989), pharmacological advances (Fordyce, Roberts & Sternbach, 1985), and interventions targeting modification of attentional and perceptual processing in the pain experience (Turk, Meichenbaum & Genest, 1983).

1.1.2 Neuromatrix Theory (Melzack, 1999)

Central to the neuromatrix theory is the assumption that the pain experience is manufactured/derived by characteristic patterns of nerve activation across a distributed neural network, termed the "body-self neuromatrix". This neuromatrix has a genetic underpinning but is subject to modification by sensory experience and learning. A further important feature of this model is that patterns of activation are initially based on peripheral sensory input but once established can be activated centrally to the exclusion of peripheral stimulation e.g. phantom limb.

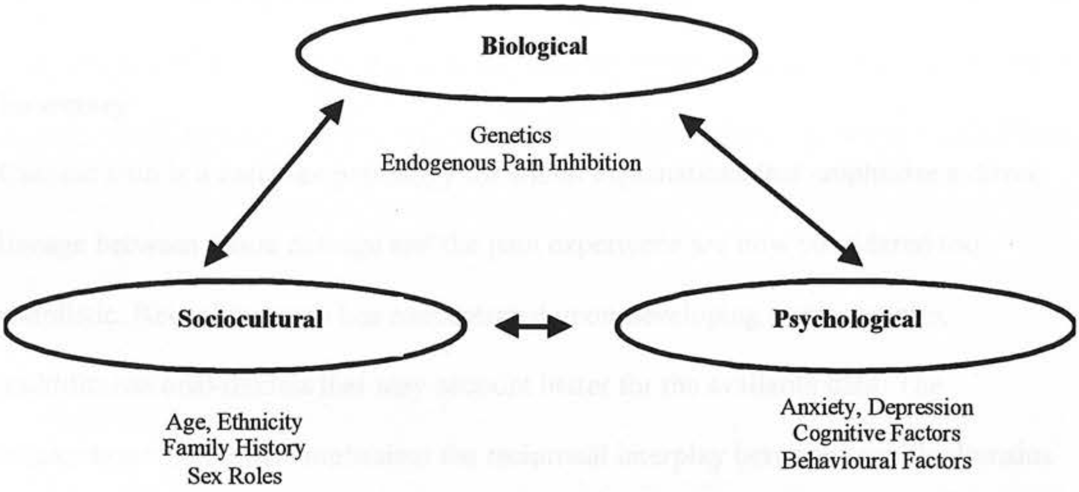
According to Melzack (1999), a person's unique body-self neuromatrix is the primary determinant of whether the organism experiences pain and is the basis for individual differences observed. Building on the Gate Control Theory, pain experience can be exacerbated/ suppressed by sensory and evaluative processes as well as activation/deactivation of the endogenous opioid system (endorphins).

The theory emphasises prior learning as an active agent in shaping the neuromatrix by influencing interpretative processes and individual physiological and behavioural response patterns. The neuromatrix theory also proposes that there is an interaction between predisposing factors and acute stressors (Turk, 2002). This operates within a diathesis-stress model framework and serves to explain the initial development of pain. The pain then evolves into a stressor in and of itself and exacerbates the pain experience. Evidence from animal research suggests that ongoing/ repetitive experience of pain can lead to structural and functional changes that may cause altered perceptual processing and contribute to pain chronicity (Woolf & Mannion, 1999). Once these changes have occurred they may contribute to the experience of pain even after the initial cause has resolved. The permanence of these changes explain the reports of pain in many chronic pain syndromes (FMS, whiplash associated disorders and back pain) even when no physical pathology is identified. This theory has provoked a degree of research interest but still requires systematic investigation.

1.1.3 The Biopsychosocial Model

The biopsychosocial model views pain as a complex interaction between biological, psychological and social variables. It is assumed that these factors interact in a way to shape a person's perception and response to illness. The biopsychosocial model is based on an understanding of the dynamic interactions between these factors. The relative influences of these biological, psychological and social factors may vary during the evolution of a disease or impairment. During the acute phase for example biological factors may prevail but over time psychological and social factors may assume a disproportionate role in accounting for symptoms. Crook, Weir & Tunks (1989) suggest that there is considerable variability in behavioural and psychological manifestations of dysfunction across individuals but also within the same individual over time.

Figure 1 Graphic Illustration of the Reciprocal relationships Involved In the Biopsychosocial Model


Biological
Genetics
Endogenous Pain Inhibition
Sociocultural
Psychological
Age, Ethnicity
Family History
Sex Roles
Anxiety, Depression
Cognitive Factors
Behavioural Factors

In the biopsychosocial model (Figure 1), biological factors may initiate, maintain and modulate physical manifestations, whereas psychological variables influence appraisals and perception of internal physiological signs and social factors shape patients behavioural responses to the perception of their physical symptoms.

Operating in a dynamic and often reciprocal interplay, psychological factors may influence biology by affecting hormone production (Bandura, O'Leary, Taylor, Gauthier & Gossard, 1987), brain structure and processes (Knost, Flor, Braun & Birbaumer, 1997) and the autonomic nervous system (Flor, Turk & Birbaumer, 1985).

Additionally, behavioural responses may also affect biological status. A person may avoid engaging in certain activities in order to reduce his or her experience of pain. This avoidance has the effect of reducing the symptoms in the short term but repeated avoidance leads to physical deconditioning which can exacerbate pain experience longer term.

Summary

Chronic pain is a complex pathology for which explanations that emphasise a direct lineage between tissue damage and the pain experience are now considered too simplistic. Recent research has concentrated upon developing more complex multidimensional models that may account better for the available data. The biopsychosocial model emphasises the reciprocal interplay between specific domains and permits a more dynamic understanding of pain. The next sections consider the psychological and social aspects of this model.

1.2 Psychological Aspects of the Biopsychosocial Model of Pain

1.2.1 Behavioural Components

Fordyce, Fowler, Lehmann & DeLateur (1968) and Fordyce (1976) identified a distinction between the original cause of pain and displays of pain, known as pain behaviours. Pain behaviours are subject to conditioning such that pain behaviours may persist for longer than the expected healing time (Sanders, 1996, 2002). Pain behaviours may be positively reinforced by attention from a spouse or a healthcare provider. They may also be maintained by negative reinforcement by escape from pain itself, by use of drugs, rest or avoidance of undesirable activities such as work.

Fordyce, Shelton & Dundore (1982) suggested that anticipation of pain alone might be sufficient to maintain avoidance behaviour through a process of respondent learning. To clarify, once an acute pain problem becomes established fear of activities associated with pain develop and avoidance ensues (Vlaeyen, Kole-Snijders, Boeren & van Eek, 1995). Non-occurrence of pain following avoidance is a powerful reinforcement for future reduction in activity. Following this, by a process of operant learning, the conditioned response (avoidance of activity) now occurs in the absence of an unconditioned stimulus (pain). Over time, avoidance leads to anticipatory anxiety relating to activity. This anxiety is a conditioned stimulus for sympathetic activation (the conditioned response). Sympathetic activation is maintained after the original unconditioned stimulus (injury) and unconditioned response (pain) have subsided (Philips, 1987a).

Further to these processes, other studies have examined "stimulus generalisation" where previously neutral activities elicit anxiety and anticipatory pain and are avoided. Persistent avoidance prevents disconfirmation and no adjustment is made to expectations of the outcome of engaging in these behaviours (Rachman & Arntz, 1991).

Behavioural learning theories usefully explain the development and maintenance of pain behaviours but are limited in their applicability to treatment situations. Fordyce (1976) noted that therapists needed to reach "a shared conceptualisation" with patients. Even the founders of behavioural therapy had noted the need to "remove patients mistaken beliefs" (Wolpe, 1958).

1.2.2 Cognitive Components

Individuals are not passive responders and actively seek to derive some meaning from their experience. Experiences are appraised at cognitive and metacognitive levels. Cognitions affect reports of pain, level of disability and response to treatment (Flor & Turk, 1988; Jensen, Turner, Romano & Lawler, 1994; De- Good & Tait, 2001; Jensen, Turner, Romano & Karoly, 1991; Turk & Rudy, 1992). Cognitive components that have received attention in the research literature relate to beliefs and attributions in respect to the pain experience. Particular types of beliefs and attribution are detailed in the section below.

1.2.2.1 Beliefs and Attribution

Chronic pain patients differ greatly in their beliefs about pain. Beliefs that the pain is an indication of continuing damage rather than a stable problem that may improve are likely to increase suffering and behavioural dysfunction even though actual pain experience is objectively the same (Flor & Turk, 1988).

Beliefs that pain is likely to persist can lead to a passive coping approach. Williams & Keefe (1991), reported that beliefs that pain was an inexplicable mystery led to minimal use of cognitive behavioural strategies to decrease pain.

Attributions regarding the significance of pain can effect perception of the symptoms. Spiegel & Bloom (1983) reported, in cancer patients, that pain severity could be predicted by interpretation of the pain. Specifically, patients who perceived their pain as relating to a worsening of their underlying condition experienced more pain that did those who held a more benign interpretation of their pain in spite of comparable levels of disease progression.

1.2.2.2 Self-Efficacy

Self-efficacy is defined as a personal conviction that one can successfully execute a course of action to produce a desired outcome in a given situation (Bandura, 1997). This construct is considered to be a major mediator of therapeutic change.

Efficacy judgements are based on prior performance, performance of others considered to be similar to oneself on the task or similar tasks, verbal persuasion by others that emphasise capabilities and perception of one's own state of arousal, which is in part based on prior efficacy judgements. Converging evidence suggests that perceived self-efficacy operates as a powerful cognitive factor in pain control (Lorig, Chastain, Ung, Shoor & Holman, 1989), adaptive psychological functioning (Lorig, Chastain, Ung, Shoor & Holman, 1989; Spinhoven, Ter Kuile, Linssen & Gazendam, 1989), disability (Dolce, Crocker & Doleys, 1986), impairment (Lorig, Chastain, Ung, Shoor & Holman, 1989) and treatment outcome (Philips, 1987b).

Cioffi (1991) has suggested four psychological mechanisms for the association between self-efficacy and behavioural outcome. Firstly, high self-efficacy decreases anxiety and as a consequence there is reduction in physiological activity, which may mean that an individual approaches a task with less potentially distressing physical information to begin with. Secondly, an efficacious individual will have the ability to distract attention away from potentially distressing physiological sensations. Thirdly, the efficacious individual may perceive distressing physical sensation but perseveres in the face of them. Finally, physical sensations are neither ignored nor distressing but are free to take on board a broad distribution of meanings.

1.2.2.3 Locus of Control

Research documents a positive relationship between a person's perceived sense of control and that person's health (Tait De Good & Carron, 1982; Wells 1994). The two main types of locus of control are internal, which reflects a belief in personal control over behaviour/health, and external, characterised as a belief that chance or powerful others have control over behaviour/health. Patients with chronic pain who have an internal locus of control are more likely to describe their pain as being less frequent and severe (Lipchik, Milles & Covington, 1993). They also have more effective pain control strategies than patients with an external locus of control (Hadjistavropoulos & Craig, 1994). Similarly, Scharff, Turk & Marcus. (2000) have suggested that patients' perceptions of the impact chronic headache has on their lives, as well as perceived control of their headaches, may be associated with the intensity, duration, and exacerbation of pain they experience.

Increased controllability of aversive stimuli reduces its impact (Jensen & Karoly 1991, Wells, 1994). while increased expectation of uncontrollability of an aversive stimuli leads to more intense reports of pain perception upon stimulation (Leventhal & Everhart, 1979).

Chronic pain sufferers typically perceive a lack of personal control and appear to believe that they have limited ability to exert control over their pain (Turk & Rudy, 1988). The relationship between perceived controllability and pain has been demonstrated in a variety of chronic pain syndromes. Increases in perceived controllability over physiological activity have been associated with decreases in migraine activity (Mizener, Thomas & Billings, 1988). Further, for patients with either low back pain or rheumatoid arthritis, both general and situation specific thoughts, relating to uncontrollability and helplessness, are stronger predictors of pain and disability than are other disease variables (Flor & Turk, 1988). Perceptions of controllability have important implications for medication usage, activity levels and psychological functioning (Jensen & Karoly, 1991).

Non-pharmacological therapy can have a beneficial impact upon perception of control in patients with chronic pain. Coughlan, Ridout, Williams & Richardson (1995) reported that patients increased their perception of personal control over their pain following participation in a multi-disciplinary program. In addition, their perception of the role of external influences affecting the experience of pain, such as fate or powerful others, abated by the end of treatment. The authors of this study concluded that a multidisciplinary approach to the management of chronic pain could alter patients' beliefs about pain and change their locus of control. Helping patients adopt an internal locus of control over pain is essential for successful treatment.

It is important to note that although the above study reported beneficial effects, generally once cognitive belief structures are formed they can be resistant to change and will remain stable over time. This is primarily due to the fact that individuals with pain will actively avoid experiences that could potentially invalidate their beliefs. In this way individuals do not receive corrective feedback and maladaptive beliefs become strengthened under a mechanism of self reinforcement.

1.2.2.4 Cognitive Errors

A number of studies have explored the issue of somatic hypersensitivity in chronic pain sufferers. Arntz & Schmidt (1989) report that pain sufferers experience maladaptive changes in pain related information processing. Thus, there develops an internal focus of threat in which benign physical symptoms are interpreted as painful/harmful sensations. These misinterpretations are also termed cognitive errors. A cognitive error is a negatively distorted belief about oneself or one's situation.

Several studies have suggested a common set of cognitive errors that influence perceptions of pain, affective distress and disability (Smith, Aberger, Follick & Ahern, 1986; Smith, Follick, Ahern & Adams, 1986; Smith, Peck, Milan & Ward, 1990). Typical types of cognitive error include catastrophizing, overgeneralization, personalisation and selective abstraction. Out of these, catastrophizing appears to be a particularly potent cognitive error that influences pain and disability. A number of studies have demonstrated that catastrophizing is important in determining the reaction to pain. People who spontaneously used fewer catastrophizing self statements rated experimentally induced pain as lower and tolerated painful stimuli longer than those that reported using more catastrophizing self-statements (Heyeneman, Fremouw, Gano, Kirkland & Heiden, 1990; Spanos, Horton & Chaves, 1975). Other studies have shown that catastrophizing leads to greater dependence on medication usage in post-surgical patients (Butler, Damarin, Beaulieu, Schwebel & Thorn, 1989) and that reduction in catastrophic thinking are related to increases in pain tolerance and reductions in physical and psychosocial impairment (Turner & Clancy, 1986).

Summary

Psychological factors in chronic pain are best represented as an interplay between behavioural and cognitive variables. Recent research has placed a greater emphasis on the role of cognitive variables in the experience of pain. In particular cognitive variables such as locus of control, self-efficacy and catastrophic thinking have received the most attention.

1.3 Social Aspects of the Biopsychosocial Model

In addition to cognitive-behavioural factors, social resources are assumed to have an impact on long-term chronic pain outcomes. Such resources include social networks and perceived support from close others. The literature relating to social factors in the experience of pain is divided between studies which assert that support from significant others is detrimental to rehabilitation and those which emphasise the beneficial effect of support on pain patients (Paulsen & Altmaier, 1995)

1.3.1 Detrimental Effects of Social Support

In terms of pain behaviours, Fordyce et al. (1973) proposed that patients displayed such behaviour in an effort to communicate to others that they are in pain and suffering. According to Fordyce (1976), pain behaviours are subject to operant reinforcement and increase in frequency when the patient receives desirable consequences or is able to avoid undesirable activities. The operant model suggests that pain behaviours, while initially related to actual tissue damage, are later maintained by the environmental contingencies through a process of operant conditioning.

Block, Kremer & Gaylor (1980) suggested that the spouse is a particularly salient discriminative cue for patients pain behaviours. They cite evidence that pain patients reported different levels of pain in an experimental situation, depending on whether they knew that they were being observed by their spouses or ward clerks. Block et al.

(1990) stated that reports of pain severity were dependent on perceptions of spouse support and whether the spouse was observing the interview. Patients who reported that their spouses were inattentive in response to pain behaviours rated their pain as lower when observed by their spouses and higher when observed by the ward clerk. Conversely, pain patients who rated their spouses as attentive, in response to pain behaviours, reported high levels of pain irrespective of who was observing. Other studies have corroborated the importance of spouse reinforcement and social support in-patient reported pain intensity (Flor, Turk & Scholz, 1987) and observed pain behaviours (Gil, Keefe, Crisson & VanDalfsen, 1987; Romano, Turner, Friedman, Bulcroft, Jensen & Hops, 1992).

1.3.2 Facilitative Effects of Social Support

In contrast, to the studies cited above a large body of literature exists suggesting that social support is beneficial in the moderation of life stress, resulting in lower levels of distress and less illness (Wallston et al. 1983; Cohen and Syme; 1985; Cohen and Wills, 1985). In addition, social support may inhibit avoidance of physical and social activities and have a beneficial impact on functional disability and pain (Cohen and Wills, 1985; Keefe, Smith, Buffington, Gibson, Studts and Caldwell, 2002 and Uchino, Cacioppo and Kiecolt-Glaser, 1996). Kerns and Turk (1984) demonstrated, under experimental conditions, that patient perception of spouse support was associated with lower levels of reported pain intensity and improved mood.

Available evidence thus far suggests that, overall, social support affects rehabilitation outcomes beneficially via a social support model and deleteriously through an operant model. Competition between these models leads to the paradox that supportive responses from spouses may have the potential to buffer against harmful effects associated with pain (under the social support model) while the same responses may reinforce maladaptive pain behaviours (under the operant model). These discrepant findings may reflect the inconsistent definition and effects of support on pain perception. Whereas some studies have considered the quantitative or enacted aspects of support, other studies have considered the qualitative, or perceived aspects of support.

Interestingly, while a number of studies have examined the impact of social support on chronic pain very few studies have examined the impact of chronic pain on social support and significant others. A study by Block & Boyer (1984) showed that spouse's cognitive interpretation of the patient's chronic pain syndrome is closely associated with the spouse's emotional adjustment and marital satisfaction. In particular, perceptions that the patient has a positive attitude along with few psychological problems all were associated with more positive emotional status in spouses. Rowat & Knafl (1985) reported that the factors of uncertainty and helplessness with regard to a partner with chronic pain were found to be central to the distress experienced by the spouses. Flor, Turk & Sholtz (1987) reported that not only is chronic pain associated with problems in the marital relationship but heightened distress and physical symptoms in spouses as well. The existence of this data suggests that in chronic pain detrimental effects on social support relationships is related less to the existence of a chronic pain condition per se but rather to patients' and spouses' manner of coping with the situation.

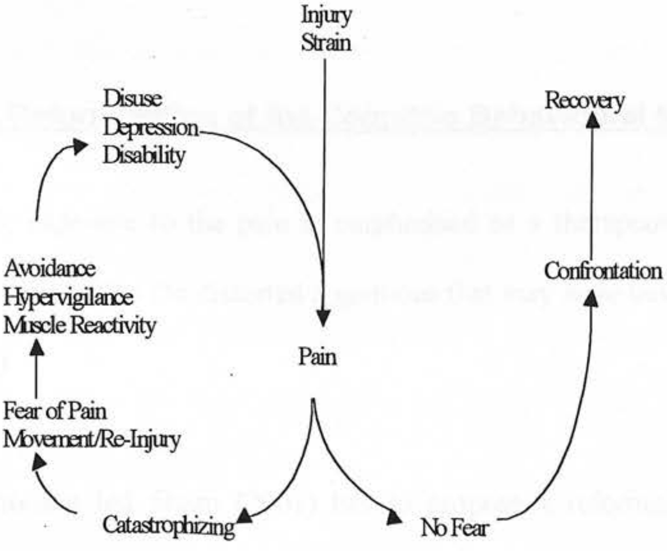
Summary

The role of social support has been investigated in a number of chronic pain populations including patients with cancer pain (Zaza & Baine, 2002), mixed conditions (Gil, Keefe, Crisson &Van Dalfsen, 1986), rheumatoid arthritis (Evers et al. 2003) and chronic lower back pain (Keefe & Block, 1982). Fewer studies have examined the impact of chronic pain on spouses and relationships. Examination of the literature reveals that no research, as yet, has been carried out looking at the function of social support in headache or how therapy interacts with social support.

1.4 Cognitive Behavioural Perspective on Pain

Considering intervention in chronic pain, the cognitive behavioural model has become the most commonly accepted psychological conceptualisation of pain, as it appears to have heuristic value in explaining the experience and response to chronic pain. Early conceptualisation of a cognitive behavioural theory of pain by Turk, Meichenbaum & Genest (1983) drew on the work of behavioural learning theorists and of Beck (1976),Beck, Rush, Shaw & Emery (1979) and Meichenbaum (1977) .

Figure 2 Cognitive Behavioural Model of Pain Related Fear (Vlaeyen &Linton, 2000)


Injury
Strain
Disuse
Depression
Disability
Recovery
Avoidance
Hypervigilance
Muscle Reactivity
Confrontation
Pain
Fear of Pain
Movement/Re-Injury
Catastrophizing
No Fear

The cognitive behavioural model draws heavily upon anxiety research. Salkovskis (1991) describes the relationship between "threat cognitions" and "safety–seeking behaviours", arguing that certain behaviours are understandable given a belief that danger is imminent. In the model above (Fig 2) when pain is experienced there is an immediate catastrophic interpretation regarding the significance of the pain. This then incurs a fear of movement which may lead to further injury. Consequently, there is an avoidance of the situation of action that resulted in the pain. While avoidance reduces anxiety in the short term, the individual suffering from pain becomes hypervigilant to somatic changes that might signify further suffering. Avoidance leads to a decrease in self-efficacy beliefs regarding the pain and this can result in feelings of learned helplessness, which may develop into depression. Reduction in coping beliefs mean further pain is perceived as more threatening and interpreted catastrophically as a vicious circle is created.

1.4.1 A Reformulation of the Cognitive Behavioural Model

In Fig 2, exposure to the pain is emphasised as a therapeutic technique with little emphasis placed on the distorted cognitions that may have developed (see to the right of Fig 2).

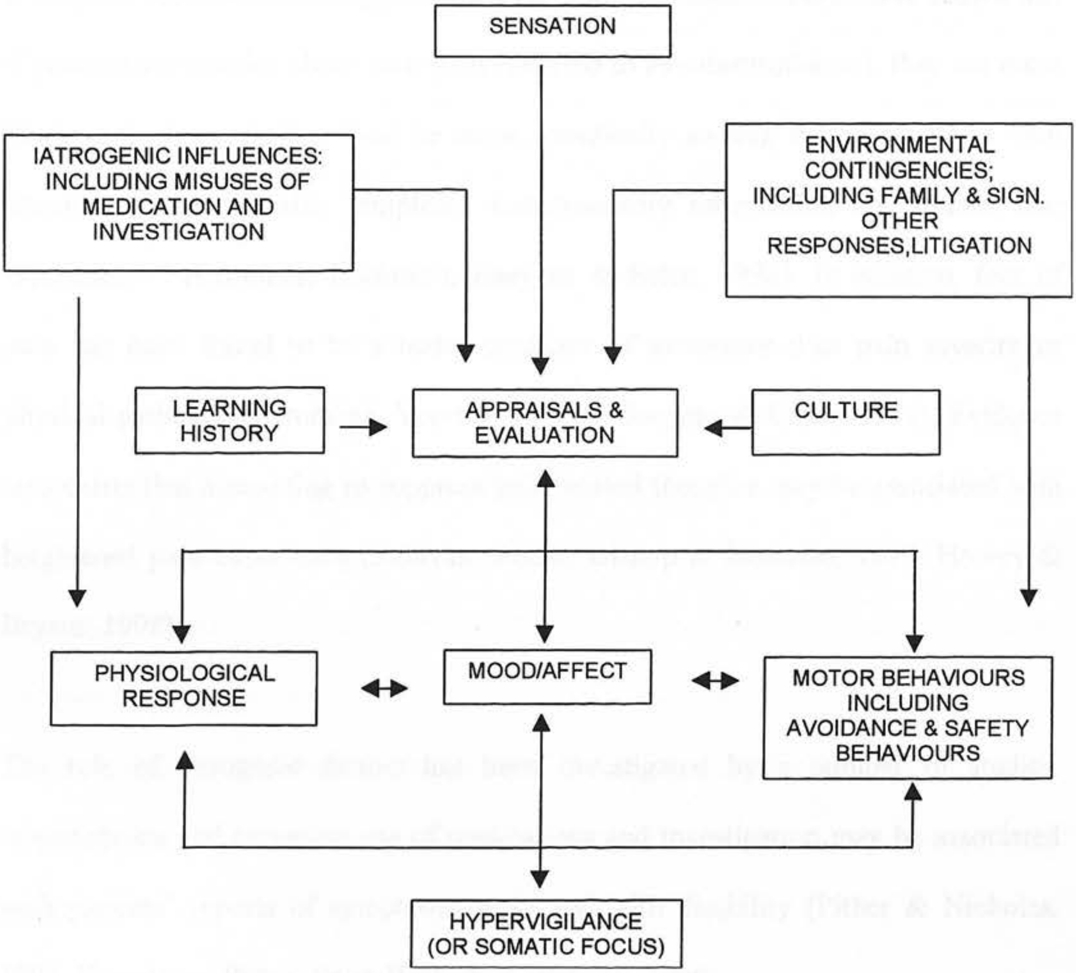
This omission led Sharp (2001) has to propose a reformulation of the cognitive behavioural model (Fig 3). In this model, patients appraisals of the pain are given a primary role in determining the level of disability encountered. Avoidance and/or safety seeking behaviours prevent the disconfirmation of negative appraisals and cognitions. At the same time anxiety and distress may maintain cognitive arousal and may reinforce beliefs that some underlying pathology exists. Anxiety and depression enhance the probability that patients make cognitive errors and also maintains avoidance which perpetuates the cycle.

The model also takes into account the likelihood that stress and iatrogenic factors may exacerbate anxiety, arousal or maintain certain behaviours. Borrowing heavily from the hypochondria literature (Salkovskis & Bass, 1997), Sharp (2001) draws additional attention to the ways that doctors respond to a patient with chronic pain which may inadvertently reinforce patient's anxieties and may reinforce excessive disability and a passive approach (Kouyanou, Pither, Rabe-Hesketh & Wessley, 1998).

Sharp's (2001) model also implicates the role of meta-cognitions in the experience of chronic pain. It is assumed that patients not only make negative appraisals about their conditions but also interpret their pain-related thoughts as indicating something negative about their condition. If these thoughts are anxiety provoking then the patients may attempt to suppress them. This model predicts that attempts to suppress or neutralise pain related thoughts might lead to an increase in their frequency and in their perceived aversiveness (Salkovskis & Campbell, 1994; Salkovsiks, Westbrook, Davis Jeavons & Gledhill, 1997).

Fig 3: The reformulated cognitive-behavioural model of chronic pain.

(Sharp, 2000)


SENSATION
IATROGENIC INFLUENCES: INCLUDING MISUSES OF MEDICATION AND INVESTIGATION
ENVIRONMENTAL CONTINGENCIES; INCLUDING FAMILY & SIGN. OTHER RESPONSES,LITIGATION
LEARNING HISTORY
APPRAISALS & EVALUATION
CULTURE
PHYSIOLOGICAL RESPONSE
MOOD/AFFECT
MOTOR BEHAVIOURS INCLUDING AVOIDANCE & SAFETY BEHAVIOURS
HYPERVIGILANCE (OR SOMATIC FOCUS)

1.4.2 Evidence Supporting Reformulated Model

A range of recent research supports this reformulated model. Studies have shown that if patients are worried about their pain (referred to as catastrophisers), they are more likely to be hypervigilant (and be more somatically aware), which interferes with attention but which also "amplifies somatosensory information and primes fear mechanisms" (Crombez, Eccleston, Baeyens & Eelen, 1998). In addition, fear of pain has been found to be a better predictor of avoidance than pain severity or physical pathology (Crombez, Vervaet, Lysens, Baeyens & Eelen, 1998). Evidence also exists that attempting to suppress pain related thoughts may be associated with heightened pain experience (Sullivan, Rouse, Bishop & Johnston, 1997; Harvey & Bryant, 1998).

The role of iatrogenic factors has been investigated by a number of studies. Inappropriate and excessive use of medications and investigation may be associated with patients' reports of symptomatology and with disability (Pither & Nicholas, 1991, Kouyanou, Pither, Rabe-Hesketh & Wessley, 1998).

Kouyanou et al. (1998) compared chronic pain patients whose symptoms were medically explained with those whose symptoms could not be medically explained. Findings suggested that patients whose symptoms could not be medically explained showed a higher incidence of over-investigation and over prescribing. It was hypothesised that the management of these patients was often unhelpful and, that by frequently "disconfirming" patients pain and by suggesting that it was "all in their mind", they were maintaining the patient in the sick role. The authors suggested that this maintenance could be attributed to the patients' determination to legitimise their pain by seeking more investigations and more treatment. Thus the outcome Kouyanou et al.'s(1998) study was to suggest that health care staff can be instrumental in influencing patients beliefs about their pain and, as a consequence, influence their pain related treatment-seeking behaviours.

Summary

Early conceptualisations of the cognitive behavioural model of pain placed a heavy emphasis on the role of behavioural factors, such as avoidance, in the maintenance of pain. Recent models acknowledge the role of behavioural factors, and have expanded the range of behavioural factors involved, however, they place a greater emphasis on the role of cognitive attributions and beliefs in the maintenance of chronic pain. In so doing these models emphasise the utility of cognitive therapy in the treatment of chronic pain.

1.5 Chronic Pain and Psychopathology

1.5.1 Depression

Romano and Turner (1985) reported that 40-50 per cent of chronic pain patients suffer from depression. In the majority of cases, depression occurs as a reaction to the development of the chronic pain condition. Other studies have suggested that chronic pain may be a masked form of depression and that emotional dysregulation is expressed in somatic terms (Turk & Salovery, 1984). Several studies have reported that depression is an important predictor of disability in chronic pain patients (Haley, 1985; Doan & Wadden, 1989) as well as a predictor of motivation for treatment (Kerns & Haythornthwaite, 1988)

Depression does not appear to be automatically associated with the development of chronic pain. It is not the case that all individuals with chronic pain are depressed. A number of studies (Okifuji, Turk & Sherman, 2000, Rudy, Kerns & Turk, 1988) suggest that pain alone is not a sufficient condition for the development of depression. Such evidence has been used to support a cognitive behavioural mediation model whereby perceptions and appraisals of the impact of the pain, the degree to which the individual is able to exert control over their pain and the impact it has upon their life, influence the development of depression. Rudy et al. (1988) found that although depression and chronic pain were modestly correlated, this relationship was virtually zero when perceptions of impact and life control were controlled for.

In longitudinal studies (Okifuji, et al. 2000), it has been reported that those individuals who believed that they were able to continue to function, despite the pain and that they could maintain some control over their pain and life, did not become depressed.

1.5.2 Anxiety Disorders

Several studies have reported high rates of anxiety disorders among chronic pain patients (Burton et al., 1997; Polatin et al, 1993). Asmudson, Jacobson, Allerdings & Norton, (1996) have reported prevalence of anxiety disorders in chronic pain range from 16.5 per cent to 28.8 per cent with panic disorder and generalised anxiety disorder being the most commonly diagnosed. Burton et al. (1997) have reported that current rates of anxiety disorder in chronic pain are higher than in the general population.

Various studies view anxiety as a premorbid characteristic of some individuals with chronic pain. Polatin et al. (1993) found that 95 per cent of those diagnosed with anxiety disorders in their sample of patients with chronic lower back pain had experienced anxiety disorders before the onset of pain. An emergent theory, based on this evidence, is that pain is a somatic expression of anxiety.

Other studies such as that of Gatchel, Garoalo Ellis & Holt (1996) suggest that anxiety develops as a reaction to pain and increases in severity as the pain becomes chronic. They found that although chronic pain patients had much higher rates of overall psychopathology than did acute pain patients, anxiety disorders were diagnosed frequently in both groups. These data support Gatchel's model of the evolution from acute to chronic pain disability in which anxiety is considered to be a common reaction to acute pain with more disabling and varied psychopathology associated with chronic pain (Gatchel, 1991; Gatchel, 1996)

Once an anxiety response is in place, chronic pain may be maintained or exacerbated through direct physiological mechanisms (Flor & Turk, 1989). Fear of pain and fear of movement lead to further physical deconditioning through avoidance of physical activities which contribute to the maintenance of the pain experience (Asmundson, Norton & Norton, 1999). Anxiety is maintained through both avoidance and continued operant learning mechanisms. Cognitive factors also become involved, with avoidance leading to reduction in self efficacy beliefs and an increase in expectancy that stimulation will increase pain (catastrophic thinking), which in turn leads to further avoidance.

1.5.3 Somatoform Disorders

One of the most common diagnoses that a chronic patient pain will receive is that of one of the somatoform disorders listed in the DSM IV (American Psychiatric Association, 1994).

"The common feature of the Somatoform Disorders is the presence of physical symptoms that suggest a general medical condition and are not fully explained by a general medical condition, by the direct effects of a substance or by another mental disorder" (American Psychiatric Association, 1994).

Research that has attempted to identify a typical personality substrate to the development of chronic pain, has consistently failed to demonstrate this to be the case (Gatchel, 1996; Mayer & Gatchel, 1988). The literature shows that significant pathology only develops after months following the onset of the pain condition. However, there appears to be a subset of patients that demonstrate tendency to express or communicate emotional distress as somatic symptoms (Fishbairn, 1999). This syndrome has been labelled as somatisation. Main, Wood, Hollis, Spanswick & Waddell (1992) use the term somatic distress to bring attention to the function of somatisation as an alternate means of communicating emotional distress.

Although somatisation is a widespread phenomena in the chronic pain population, there appears to be a subset of individuals for whom this has become amplified (Fishbairn, 1999). For these patients, somatisation is conceptualized as a stable trait (diathesis) that becomes activated in response to stressful situations and events that the individual finds stressful, such as painful injury. Although this does not meet the criteria for a somatoform pain disorder, somatisation has been found to be associated with increased risk for developing chronic pain and greater health utilization in acute pain patients (Dworkin, 1995) and poorer treatment outcomes in chronic pain patients (Vassend, Krogstad & Dahl, 1995).

A typical somatoform diagnosis is that of pain disorder. This is diagnosed when the pain is the predominant focus of the clinical presentation, when the pain causes significant distress or functional impairment and when psychological factors are considered to have a large part to play in the onset, severity, exacerbation, or maintenance of the pain (American Psychiatric Association, 1994).

Controversy over the current diagnostic definitions, the change in diagnostic criteria and the subjectivity needed to make these diagnoses has made reliable and valid prevalence estimates difficult to establish. Specific studies examining chronic lower back pain have estimated prevalence to range from 73 per cent to 97 per cent (Polatin et al. 1993; Fishbairn, 1986). These data were derived from diagnostic classification prior to DSM IV. It is suggested that, given the inclusiveness of the current diagnostic classification, the prevalence rates are likely to be very high, perhaps above the estimates derived from the studies cited above.

1.5.4 Personality Disorders

A number of studies have reported high rates of personality disorder among chronic pain patients. Prevalence rates range from 31 per cent to 81 per cent which is well above that exhibited in the general population (Burton et al. 1997; Gatchel et al. 1996; Weisberg, Gallagher, & Gorin, 1996). Little consistency has been found over the relationship between chronic pain and specific types of personality disorder. Several studies have reported that among the personality disorders, histrionic (Reich et al. 1983), dependent, (Fishbain et al. 1986), paranoid (Polatin et al. 1993) and borderline (Weisberg et al. 1996) are most commonly diagnosed alongside chronic pain. Inconsistency in the findings reflect discrepancies in patient samples, diagnostic methods, and the overlap between the various DSM IV personality disorder categories (Widiger, Trull, Hurt, Clarkin & Frances, 1987).

Again, as with other disorders associated with pain, there is a great deal of evidence supporting the diathesis stress model between chronic pain and personality disorder. According to Weisberg & Keefe (1997,1999), personality patterns that are associated with marginally adaptive coping styles usually decompensate under the stress of injury, disability and pain resulting in the expression of the personality disorder.

Summary

Chronic pain is co-morbid with a number of psychopathologies including depression, anxiety, somatoform disorders and personality disorders. Given this co-morbidity, questions arise as to what should be the primary focus of any therapy i.e. either psychopathology or chronic pain or a combination of both. This decision rests on the overall question of whether chronic pain is a somatic expression of emotional dysfunction or whether emotional dysfunction is a reaction to the experience of pain. No clear conclusion exists regarding this question.

1.6 Mechanisms of Change in Pain

1.6.1 Transition from acute to chronic pain

Gatchel (1991) and Gatchel (1996) developed a broad conceptual model to explain the transition from acute to chronic pain. This model defines three hypothesised stages involved in this transition. Stage 1 is associated with fear, anxiety and worry resulting from the perception of pain during the acute phase. This tends to be viewed as a normal emotional reaction. If the pain persists for longer (e.g.2-4 months) then there is a progression into stage 2. During stage 2, a wider array of behavioural-psychological reactions and problems such as, learned helplessness, distress-anger and somatisation develop. This theory is based on a diathesis stress model which implies that problems in Stage 2 depend on the *premorbid* characteristics of the individual. In those individuals with premorbid depression, stage 2 will lead to an exacerbation of their problems.

Persistence of behavioural and psychological problems leads to stage 3, in which the sick role is adopted. During this stage patients become excused from social obligations and normal responsibilities. This can serve as a potent reinforcer for maintaining the sick role. An additional maintaining process known as the physical deconditioning syndrome (Mayer & Gatchel, 1988), which refers to a decrease in physical capacity (strength, flexibility and endurance) can have a feedback effect on levels of emotional well being and self-esteem. Such deleterious feedback increases this deconditioning as levels of motivation deteriorate (Gatchel, Baum & Krantz, 1989).

Gatchel & Epker (1999) delineated a number of risk factors or barriers that are associated with the transition from acute to chronic pain (Table 1). These are potential "flags" to clinicians in order to anticipate possible barriers to recovery.

Table 1 Summary of Psychosocioeconomic Risk Factors That May Predict the Development of Chronic Pain Disability.

- High self reported pain and disability
- Elevation of MMPI Scale 3 (Hysteria)
- Depression
- Somatisation
- Poor Coping Skills
- Poor quality of social support
- Unresolved workers' compensation/personal injury cases
- Gender
- Reinforcement of pain behaviours
- Job dissatisfaction
- Maladaptive attitudes and beliefs about pain
- History of childhood sexual abuse

Note: From Gatchel and Epker (1999).

1.6.2 Change In Treatment

The Transtheoretical Model (Prochaska & DiClemente, 1983; Prochaska, DiClemente, & Norcross, 1992; Prochaska & Velicer, 1997) is an integrative model and an attempt to define the underlying structure of change. The transtheoretical approach uses the structure of intentional change as the foundation of its integrative efforts (Prochaska, 1984). It is based on an analysis of the most popular theories of psychotherapy, including affective, behavioural, cognitive, dynamic, experiential, relationship, and systems approaches to therapy (Prochaska, 1984), and subsequent integration of the key concepts from these theories. The model describes how people modify problem behaviour or acquire a positive behaviour. The central organizing construct of the model is the Stages of Change. The model also includes a series of independent variables that facilitate change called the Processes of Change.

The Transtheoretical Model involves four primary constructs in explaining health behaviour change: stages of change, processes of change, decisional balance (pros and cons), and self-efficacy (temptations). The latter two are classed as intervening variables.

1.6.2.1 Stages of Change

The Transtheoretical Model construes change as a process involving progress through a series of five stages: Precontemplation, Contemplation, Preparation, Action and Maintenance (see table 2)

Table 2: Stages of Change in the Transtheoretical Model

Stages of Change	*Definition*
1. Precontemplation	Individuals are entering a change situation, but they do not believe they have a problem or know they do not want to change. Has no intention to take action within the next 6 months
2. Contemplation	Individuals in this stage are beginning to be aware that the problem exists or that they are bothered by something about themselves. They are struggling to understand the problem (i.e., cause, solution) and are seeking more information. They are considering change but have not made a commitment to change. Intends to take action within the next 6 months
3. Preparation	Individuals in this stage intend to take action. They have made some reductions in their problem behaviours, but they have not yet reached a criterion for effective action. Intends to take action within the next 30 days.
4. Action	In the action stage, people have decided to change and have actively started to work on changing, often seeking help to do so more effectively. Has changed overt behaviour for less than 6 months
5. Maintenance	Individuals work to prevent relapse and consolidate gains accomplished during the action stage. Has changed overt behaviour for more than 6 months.

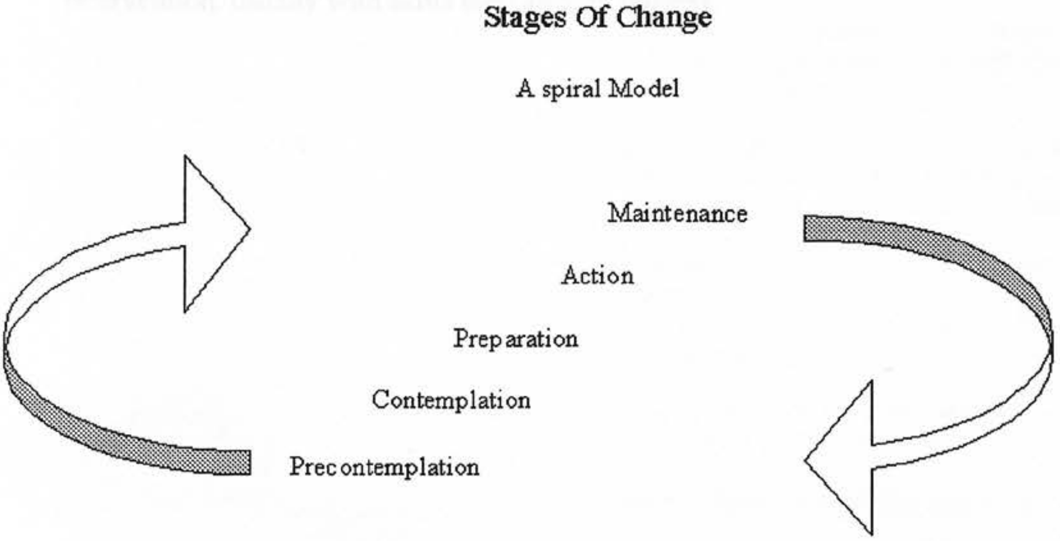


Figure 4 Stages of Change: A Spiral Model

The Stages of Change model implies that, for most persons, a change in behaviour occurs gradually, with the patient moving from being uninterested, unaware or unwilling to make a change (precontemplation), to considering a change (contemplation), to deciding and preparing to make a change. Determined action is then taken and, over time, attempts to maintain the new behaviour occur. Relapses are almost inevitable and become part of the process of working toward life-long change. Relapse is an opportunity to learn, gain missing information, redefine plans and take action again. Prochaska (1994) found that people who take action and fail in the next month, are twice as likely to succeed over the next six months as those who don't take any action at all.

The Stages of Change model is useful for selecting appropriate interventions. By identifying a patient's position in the change process, physicians can tailor the intervention, usually with skills they already possess.

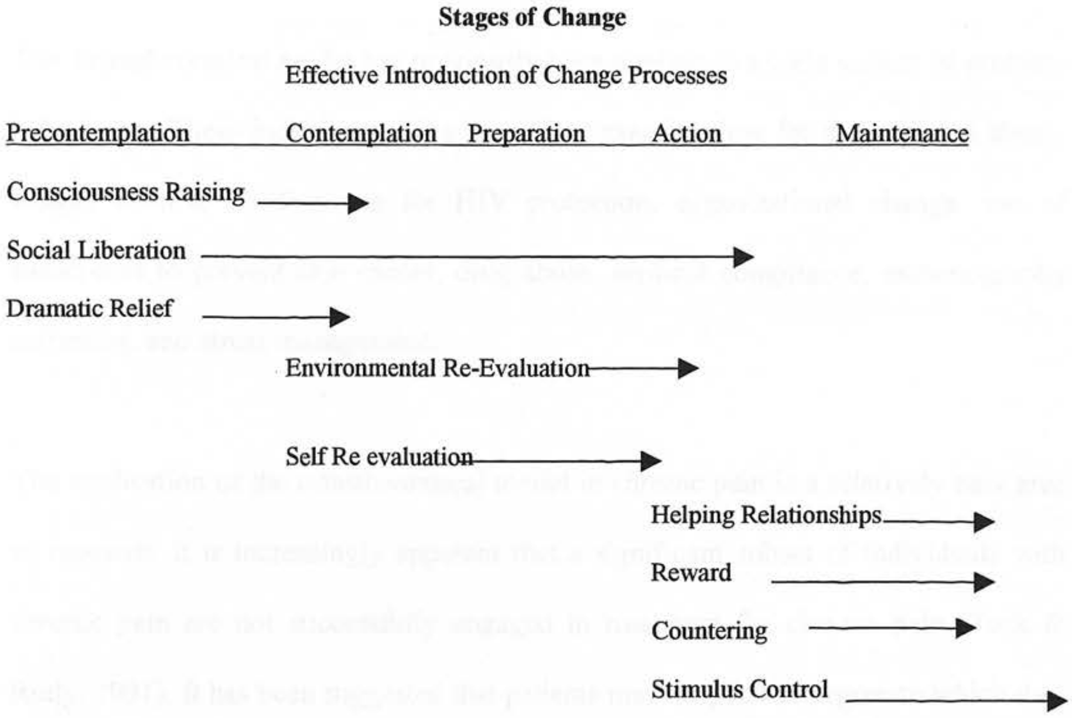
Table 3 Processes of Change in the Transtheoretical Model

Process	Definition
I Experiential	
1. Consciousness Raising	Involves providing information regarding the nature and risk of unsafe behaviours and the value and drawbacks of the safer behavioural alternatives.
2. Dramatic Relief	Experiencing and expressing feelings about the problem behavior and potential solutions/ psychodrama, grieving losses, role playing.
3. Environmental Re-evaluation	Allows the individual to reflect on the consequences of his or her behaviour for other people. It can include reconsideration of perceptions of social norms and the opinions of people important to him or her.
4. Self Re-evaluation	Entails the reappraisal of one's problem
5. Social Liberation	Seeking to help others with similar situations.
II Behavioural	
6. Self Liberation	Encourages the person to consider their confidence in their ability to change and their commitment to doing so.
7. Helping Relationships	Assists the person in a variety of ways, including providing emotional support, modelling a set of moral beliefs, and serving as a sounding board.
8. Reward	Developing internal and external rewards and making them readily but contingently available to improve the probability of the new behaviour occurring or continuing.
9. Countering	Learning of healthier behaviours that can be substituted for problem behaviours.
10. Stimulus Control	Removal of cues for unhealthy behaviour.

1.6.2.2 Processes Of Change

In explaining transition from stage to stage a set of cognitive and behavioural processes are hypothesised that engender change. It is further hypothesised that individuals in the earlier stages (e.g., precontemplation, contemplation, preparation) are more likely to use cognitive-experiential processes (Table 3, Figure 5), such as dramatic relief and self-re-evaluation. By contrast, individuals in the action and maintenance stages are more likely to use behavioural processes, such as counter-conditioning and stimulus control (Prochaska, 1984; Rossi and Wilcox). Furthermore, individuals in the precontemplation stage use processes the least. Each of the processes of change includes hundreds of techniques of change. For example, an overeater may drink water when feeling tempted to snack, while someone in an Alcoholics Anonymous program might pray when tempted to drink. Although using different techniques, both are practising the process of counterconditioning: substituting healthy responses for unhealthy ones. Successful changers use the processes of change that are most appropriate to each stage of change.

Figure 5: Illustration of the Processes Involved at Each Stage of Change


Stages of Change
Effective Introduction of Change Processes
Precontemplation
Contemplation
Preparation
Action
Maintenance
Consciousness Raising
Social Liberation
Dramatic Relief
Environmental Re-Evaluation
Self Re evaluation
Helping Relationships
Reward
Countering
Stimulus Control

1.6.2.3 Intervening Variables

In addition to the stages and processes of change, the model also includes two intervening variables: decisional balance (the pros and cons of behaviour change) and self-efficacy (situational confidence or temptations regarding the behaviour). These intervening variables are strongly related to the individual's stage of change, may be used to monitor intervention effectiveness, assist in assessing individual progress toward behaviour change, and indicate potentially troublesome situations that need to be targeted in order to make change and prevent relapse.

1.6.3 Applicability of the Transtheoretical Model to Chronic Pain

The Transtheoretical model has previously been applied to a wide variety of problem behaviours. These include smoking cessation, exercise, low fat diet, alcohol abuse, weight control, condom use for HIV protection, organizational change, use of sunscreens to prevent skin cancer, drug abuse, medical compliance, mammography screening, and stress management.

The application of the transtheoretical model in chronic pain is a relatively new area of research. It is increasingly apparent that a significant subset of individuals with chronic pain are not successfully engaged in treatment for chronic pain (Turk & Rudy, 1991). It has been suggested that patients may vary in the degree to which they are ready to adopt a self management approach to pain and that this variable may influence the engagement process as well as drop out and relapse rates (Kerns, Rosenberg, Jamison, Caudill & Haythornwaite, 1997).

Unlike many traditional biomedical treatments, psychologically based pain treatments require patients to make substantial changes in the way that they view and cope with pain. It is hypothesised that only those individuals ready to consider making these changes would be expected to benefit from psychological pain treatments (Jensen, 1996).

The Transtheoretical model predicts that individuals at different stages of change should evidence different treatment success rates. Individuals in the action stage should show greater success than individuals in the contemplation and preparation stages. Treatment outcome research has supported this hypothesis for smoking cessation (Ockene, Ockene, & Kristeller, 1988) and weight control (Prochaska, DiClemente & Norcross, 1992). The model also predicts that a person's behaviour should reflect their stage of change. Individuals in the precontemplative stage may be expected to disagree with suggestions that a change in behaviour is needed and would not be expected to exert effort towards making changes (Jensen, Neilson, Romano, Hill & Turner 2000).

The stages of change model therefore has the potential to shed light on the reasons for differences in patient outcome and help to identify those individuals who are most likely to benefit from treatment. In addition, the theory has the potential to help identify a persons particular stage of change and therein tailor intervention to maximise therapeutic yield (Miller & Rollnick, 1991, Jensen, 1996, Kerns et al. 1997).

Summary

Studies are beginning to examine the applicability of the transtheoretical model, to explain variable treatment outcomes. Studies have begun to examine the process of health behaviour change in heterogeneous chronic pain samples (Kerns et al., 1997; Kerns & Rosenberg, 2000; Jensen et al. 2000) including; fibromyalgic patients (Jensen et al. 2000) and community based samples (Habib, Morrissey & Helmes, 2003). As yet no studies have looked at the transtheoretical model in relation to health behaviour change in a chronic tension headache sample.

1.7 Treatment in Chronic Pain

1.7.1 A Cognitive Behavioural Perspective

The chronic pain literature is replete with research relating to cognitive behavioural intervention. Research relating to other psychotherapeutic intervention is noted only by its general absence. Behavioural and cognitive treatments for chronic pain have become established over the 30 years since their initial usage (Fordyce et al., 1968, 1973; Turk et al. 1983). While there are many published open trials of treatment, few use control groups in which patients are randomised to treatments.

Reviews, however, conclude that there is strong, if not overwhelming evidence for the efficacy of cognitive behavioural therapy (CBT) in restoring function and mood and in reducing pain and disability-related behaviour (see e.g. McCracken, 1991; Hawley, 1995; Morley, Eccleston & Williams, 1999; van Tulder et al., 2000).

Recently Morley, Eccleston & Williams (1999) conducted a systematic review and meta-analysis of published Randomised Control Trials (RCTs) of CBT for chronic pain, excluding headache. They concluded that cognitive-behavioural therapy (including behaviour therapy and biofeedback) is effective relative to waiting list control conditions. CBT produced significant changes in measures of pain experience, mood/affect, cognitive coping and appraisal (reduction of negative coping and increase in positive coping), pain behaviour and activity level, and social role function. When compared across the same range of outcomes with other treatments or control conditions, the efficacy of CBT showed a smaller effect size and was limited to the outcomes of pain experience, positive coping and social role function. The overall effect sizes, in the order of 0.5, is less than those from larger meta-analyses which consider psychological treatments for a variety of disorders (Shadish et al., 1997).

Evidence in the literature point to four components of cognitive behavioural pain interventions: education, skill acquisition, behavioural rehearsal and generalisation/maintenance (Bradley, 1996). The skills acquisition phase for chronic pain patients often includes graded activity scheduling, exercise, relaxation, medication reduction and cognitive techniques (Linton, 1993; Philips & Rachman, 1996). Cognitive-behavioural models posit that improvement is due, in part, to changes in patient coping strategies and beliefs (appraisal). The cognitive behavioural method therefore attempts to change patterns of negative thoughts and dysfunctional attitudes to foster more healthy and adaptive thoughts, emotions, and actions in the patient.

Other methods of behavioural therapy are also integrated into the cognitive behavioural approach. Relaxation techniques comprise a group of therapeutic approaches that allow the patient to achieve non-directed relaxation, and are effective in the treatment of chronic pain (Morley, Eccleston & Williams, 1999). Although there are several ways to achieve relaxation, one method may be more effective than another for an individual patient. Biofeedback techniques provide the patient with information on physiological functions to help in the relaxation process. Feedback information that is provided to the patient can include electromyography, electro-encephalography, galvanometry, and temperature. Hypnotic techniques can help induce states of directed relaxation.

Moore, Von Korff, Cherkin, Saunders & Lorig (2000) evaluated a brief cognitive behavioural intervention for primary care back pain patients. Treatment effects were not observed across all outcome domains. Participants assigned to the CBT intervention showed significantly greater reductions in back-related worry and fear-avoidance beliefs than the control group. Modest, but statistically significant, effects on pain ratings and interference with activities were also observed.

Summary

In spite of the encouraging effects of psychosocial interventions in chronic pain disorders, particularly CBT, the variability in outcomes between patients and the magnitude and maintenance of effects in the long run is a point of continuing discussion (e.g. Turk, 1990; McCracken, 1991; DeVellis and Blalock, 1993; Keefe and van Horn, 1993; Hawley, 1995; Turk and Okifuji, 1998; Gatchel, 2001).

1.8 Headache

1.8.1 Tension Type Headache

Definition

According to the Headache Classification Committee of the International Headache Society (Headache Classification Committee of the International Headache Society (IHS) (1988) headache disorders are classified into two categories: primary and secondary. Primary headaches are defined as having no apparent underlying organic disease process. Secondary headaches on the other hand are defined as symptomatic of an underlying organic disease.

Tension-type headache is classified as a primary headache and is symptomatically defined as pain that radiates in a band-like fashion bilaterally from the forehead to the occiput. Pain often radiates to the neck muscles and is described as tightness, pressure, or dull ache. Migraine-type features (unilateral, throbbing pain, nausea, photophobia) are not present although migraine may be a co-morbid disorder (Jensen, 1999). Tension-type headaches can last from 30 minutes to several days and can be continuous in severe cases.

Tension-type headache is classified into two types: episodic and chronic. Generally, episodic headaches occur randomly and are often the result of temporary stress, anxiety, fatigue or anger. A tension-type headache that occurs just about every day, and may have been going on for months, is referred to as chronic.

1.8.1.1 Episodic tension-type headache

This is described as recurrent episodes of headache lasting minutes to days. The pain is typically pressing or tightening in quality, of mild to moderate intensity, bilateral in location and does not worsen with routine physical activity. Nausea is absent, but photophobia or phonophobia may occur.

Diagnostic criteria for Episodic Tension-Type Headache

A. At least 10 previous headache episodes fulfilling criteria B-D listed below.
Number of days with such headache <180/year.

B. Headache lasting from 30 minutes to 7 days

C. At least two of the following pain characteristics:

1. Pressing/tightening (non-pulsating) quality
2. Mild or moderate intensity
3. Bilateral location
4. No aggravation by walking stairs or similar routine physical activity

D. Both of the following:

1. No nausea or vomiting (anorexia may occur)
2. Photophobia and phonophobia are absent, or one but not the other is present

Source: Headache Classification Committee 1988.

Fig 6: Definition of Episodic Tension Type Headache According to the Headache Classification Committee, 1988

1.8.1.2 Chronic tension-type headache

Headache is present for at least 15 days per month for at least 6 months. The pain is typically pressing or tightening in quality, of mild to moderate intensity, bilateral in location, and does not worsen with routine physical activity. Nausea, photophobia, or phonophobia may occur.

Diagnostic criteria (IHS) Chronic Tension-Type Headache

A. Average headache frequency of more than 15 days per month for over 6 months.

B. At least 2 of the following pain characteristics:

   1. Pressing (non-pulsating) quality
   2. Mild or moderate intensity (may inhibit, but not prohibit activities
   3. Bilateral location
   4. No aggravation by walking stairs or similar routine physical activity

C. Both of the following:

   1. No vomiting
   2. No more than one of the following: nausea, photophobia, phonophobia

D. Secondary headache types not suggested or confirmed

Comment: Although this headache is generally continuous, it is seldom disabling. It fluctuates in intensity. During moderate or severe exacerbations, it often has mild migrainous features, such as throbbing, nausea, and mild hypersensitivity to light. Its persistence makes it hard to endure. It commonly persists for many years, although preventive medications often provide considerable relief

Fig 7: Definition of Chronic Tension Type Headache According to Headache Classification Committee, 1988.

1.8.2 Epidemiology

In surveys of the general population in the industrialized part of the world, reported prevalence of tension-type headache ranges from about 30 per cent to about 80 per cent (Rasmussen, Jensen, Schroll, Olesen, 1991; Edmeads, Findlay, Tugwell, 1993: Ramussen, 2001). Differences in definitions, methodology, and study population may be largely responsible for this variation (Ramussen, 2001).

Tension-type headache varies widely in both frequency and severity, from rare short-lasting episodes of discomfort (episodic type) to frequent, long-lasting, or even disabling headaches (chronic type) (Jensen, 2001). A criticism of many epidemiological studies is that pooling these extremes in an overall prevalence may be misleading. Prevalence data should consider level of disability, severity, duration and frequency of the disorder (Ramussen, 2001; Rasmussen, Jensen, Schroll, Olesen, 1991)

In a recent population-based study of a random general Danish population (Rasmussen, Jensen, Schroll, Olesen, 1991), 41 per cent of subjects with tension-type headaches did not have their daily activities inhibited because of the headache, whereas 59 per cent had moderate or severe impairment of their daily activities. Of subjects experiencing tension-type headache in the previous year, 59 per cent had it one day a month or less, and 37 per cent several times a month. In the total population, 3 per cent had chronic tension-type headache (i.e., headache 180 or more days a year; Rasmussen et al. 1991), which is in agreement with other reports which examined epidemiology in the Canadian population (Pryse-Phillips, Findlay, Tugwell, Edmeads, Murray & Nelson, 1992) and German population (Göbel, Petersen-Braun, Soyka, 1994).

Population-based studies including data on the frequency of tension-type headache have agreed with the Danish study (Rasmussen et al., 1991) in finding a rather large proportion of subjects with mild and infrequent (once a month or less) tension-type headache(Abramson, Hopp, Epstien, 1980; Hollnagel & Nørrelund 1980; Nikiforow 1981; Rasmussen et al. 1991; Edmeads et al. 1993). In addition, these same studies reach agreement on the one-year prevalence of frequent tension-type headache (more that once a month) as being in the region of 20–30 per cent.

In terms of gender differences, tension-type headache is more prevalent in females than in males (male/female ratio about 1:1.5) (Ramussen, 2001), and in both sexes prevalence declines with age (Waters 1972, 1974, 1975; Philips 1977; Abramson et al. 1980; Rasmussen et al. 1991; Pryse-Phillips et al. 1992). 1993; Rasmussen 1993). The most common age at onset of tension-type headache is in the second decade, somewhat younger than that of migraine (Rasmussen 1993).

1.8.3 Psychophysiology of Headache

For decades it has been a matter of debate whether the pain in tension-type headache originates from myofascial tissues or from central mechanisms in the brain (Jensen, 1999). Frequent tension-type headaches are now thought to be maintained primarily by a central nervous system (CNS) dysfunction, not solely by input from peripheral nerves in contracted facial, neck, and shoulder muscles (Olesen & Schoenen, 2000). This CNS dysfunction may involve the sensitisation of pain transmission circuits in the trigeminal nucleus, where input from nerves in the face and head is first integrated and relayed toward the brain (Bendtsen & Ashina, 2000; Bendtsen, Jensen, & Olesen, 1996; Olesen, 1991). Such sensitization would lower the threshold of these circuits for the transmission of pain signals, so that little or no input from peripheral nerves (noiciceptors) is required for the transmission of pain signals to the brain. A dysfunction in supraspinal (limbic) pain modulation circuits may also maintain pain by permitting, or even facilitating, the transmission of pain signals in the brain (Schoenen, 1993; Schoenen, Jamart, Gerard, Lenar-duzzi, & Delwaide, 1987; Schoenen & Wang, 1997). This shift from peripheral to central mechanisms has stimulated a resurgence of interest in the psychophysiology of tension-type headache (Jensen, Ramussen, Pedersen, Olesen, 1993; Bendtsen, Jensen, Olesen, 1996; Langemark, Jensen, Jensen, Olesen, 1989).

The role of psychopathology as a mechanism in the maintenance of tension type headache has received considerable attention. Headaches are generally reported to occur in relation to emotional conflict and psychosocial stress, but the cause-effect relationship is not clear. Stress and mental tension were the most frequently reported precipitating factors but they occurred with similar frequency in tension-type headache and migraine (Rasmussen 1993, Ulrich, Russel, Jensen, Olesen, 1996). These results correspond with the findings of widely normal personality profiles in individuals with episodic tension-type headache, whereas studies of subjects with the chronic form often reveal a higher frequency of depression and anxiety (Holroyd, France, Nash, Hursey, 1993; Rasmussen,1992; Mitsikostas &Thomas, 1999). As in other chronic pain disorders, psychological abnormalities in tension-type headache are viewed as secondary rather than primary, and anxiety and depression are probably co-morbid with chronic tension-type headache (Jensen, 1999). However this view is not consistent with a diathesis stress model in which a common predisposing factor may account for both the presence of headache and psychopathology. The attraction of the diathesis stress model necessitates further work to be carried out in this area.

The progression of episodic tension-type headache into chronic tension-type headache can occur spontaneously but often occurs in relation to frequent use of analgesic medication. Repeated use of analgesics, especially ones containing caffeine or butalbital, can lead to "rebound" headaches as each dose wears off and patients then take another round of medication. Common features of chronic daily headache associated with frequent analgesic use are early morning awakening with headache, poor appetite, nausea, restlessness, irritability, memory or concentration problems, and depression (Rapoport, Stang, Gutterman, Cady, Markley, Weeks et al. 1996). Additionally, this view is to the neglect of important psychological processes, which may also explain the transformation form acute pain to chronic pain. Again further work needs to be conducted to understand these processes.

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1.8.4 Headache Disorders and Co-morbid Psychopathology

Epidemiological data suggest the prevalence of anxiety and mood disorders are not elevated in Episodic Tension Type Headache "ETTH" (Merikangas, 1994; Merikangas, Stevens, & Angst, 1993). However, it is doubtful this finding can be generalised to Chronic Tension Type Headache "CTTH".

In clinical samples there is a high prevalence of both anxiety and mood disorders in CTTH. Over 40 per cent of CTTH sufferers in primary care settings receive either an anxiety or mood disorder diagnosis by standardized diagnostic assessments (Goncalves & Monteiro, 1993; Guidetti, Galli, Fabrizi, Giannantoni, Napoli, Bruni & Trillo, 1998; Holroyd, Stensland, Lipchik, Hill, O' Donnell, Cordingley, 2000; Puca, Genco, & Prudenzano, 1999). This figure is purportedly higher for CTTH sufferers seen in speciality settings e.g. 45 per cent (Holroyd, Stensland, Lipchik, Hill, O' Donnell, Cordingley, 2000). Moreover, this becomes an important therapeutic issue as co-morbid anxiety or mood disorders appear to increase the disability associated with CTTH's. Consequently, the identification and effective management of co-morbid psychiatric disorders may play an important role in the management of CTTH's (Holroyd et al., 2000).

1.9 Psychological Management of Chronic Tension Type Headache

1.9.1 Overview

Although medication is the most commonly used treatment for chronic tension-type headache, a number of other methods have begun to amass evidence relating to efficacy and effectiveness.

Holroyd (2002) reviewed three forms of psychological treatment which were judged to have received empirical support for the management of tension-type headache: (a) relaxation training (RLX), typically in the form of progressive muscle-relaxation training; (b) electromyographic biofeedback training (EMG-BF), typically to reduce muscle activity in forehead and often neck and shoulder muscles; and (c) specific forms of cognitive–behavioral therapy (CBT) (i.e stress management, pain management, minimal contact). Similar effect size and percentage improvement data were found for the three behavioural treatments with a reported 40 per cent to 50 per cent reduction in tension-type headache activity when results are averaged across trials. This is consistent with results from earlier, more inclusive meta-analyses (Blanchard, Andrasik, Ahles, Teders, & O'Keefe, 1980; Bogaards & ter Kuile, 1994; Holroyd & Penzien, 1986) that used different statistical techniques. This evidence has been cited by other authors who report "Behavioral treatments for tension-type headache have a consistent body of research indicating efficacy" (McCrory, 2001).

Blanchard, Appelbaum, Radnitz, Morrill, Michultka, Kirsch (1990) reported reductions in headache frequency and medication usage with an overall effect size of 0.55 for the former and 0.42 for the latter using cognitive behavioural therapy. Mosley, Grotheus, and Meeks (1995) extended this finding to an older (over 65 years of age) patient population and found that CBT is more effective than RLX alone.

In a study examining the effectiveness of CBT on CTTH, Holroyd, O'Donnell, Stensland, Lipchick, Cordingley & Carlson (2001) found that CBT was more effective than pill placebo and comparable in effectiveness to tricyclic antidepressant medication in reducing headache activity, analgesic medication use, and headache-related disability.

1.9.2 Alternative Therapies

A recent systematic review of acupuncture treatment for headache (Melchart, Linde, Fischer, White, Allais, Vickers et al. 1999) found 40 randomized controlled studies, but only one study was categorized as "rigorous." In all of the trials of tension-type headache that were examined, patients receiving acupuncture had superior outcomes, compared with patients in the control groups. The authors of the review concluded that, "overall, the existing evidence suggests that acupuncture has a role in the treatment of recurrent headaches" (Melchart, Linde, Fischer, White, Allais, Vickers et al. 1999).

Studies have also been conducted investigating the role of spinal manipulation for headache relief. (Boline, Kassak, Bronfort, Nelson & Anderson, 1995). In a trial comparing manipulation with the use of amitriptyline, both modalities showed improvement in headache intensity, frequency, and medication usage. However, headache intensity was significantly less in the amitriptyline group. Four weeks after cessation of therapy, patients in the spinal manipulation group continued to experience benefits from the intervention.

1.9. 3 Therapeutic Mechanisms

The belief that EMG-BF training reduces tension headache activity by enabling individuals to control sustained contractions in pericranial muscles is now being challenged (Andrasik & Holroyd, 1980; Holroyd, Penzien, Holm, & Hursey, 1984). A competing cognitive–attributional model that emphasises cognitive change as the key therapeutic mechanism in EMG-BF and CBT has received initial support (Holroyd & Penzien, 1983; Holroyd et al., 1984).

More recent studies provide additional support for this cognitive– attributional model. In the first study, false feedback was used to manipulate patients' perceptions of their performance during RLX (Blanchard, Kim, Hermann, & Steffek, 1993). High-success feedback yielded larger improvements (54 per cent reduction) in tension-type headache activity than moderate success feedback (21 per cent reduction), even though behavioral ratings of actual relaxation behaviors during training did not differ in the high-success and moderate success feedback groups (Poppen, 1988).

In a second study, combined EMG-BF and RLX produced larger reductions (51 per cent of patients clinically improved) in tension-type headache activity than were observed in untreated controls (15 per cent of patients clinically improved) (Rokicki, Holroyd, France, Lipchik, France & Kvaal, 1997). A regression analysis revealed that changes in self-efficacy scores over time explained more of the variance (43 per cent) in improvement scores following treatment than changes in EMG activity during biofeedback training. This suggested that changes in self-efficacy ratings were a greater predictor of improvements following treatment.

1.9.4 Integrating Drug and Psychological Therapies

Reich & Gottesman (1993) examined the benefits of adding amitriptyline (up to 75 mg/day) to EMG-BF. The combination of amitriptyline HCl and EMG-BF yielded more rapid improvement in chronic tension-type headache activity than EMG-BF alone; however, longitudinal data over a period of 14 months showed this combined treatment showed no advantage over EMG-BF alone.

Holroyd et al. (2001) examined the separate and combined effects of CBT and tricyclic antidepressant medication for CTTHs. Patients received one of four treatments: tricyclic antidepressant medication (amitriptyline HCl to 100 mg/day or nortriptyline HCl to 75 mg/day), medication placebo, limited-contact CBT (three clinic sessions) plus antidepressant medication, or CBT plus placebo. Antidepressant medication and CBT yielded similar reductions in CTTHs, analgesic medication use, and headache-related disability at a 6-month evaluation, but again improvements tended to be more rapid in the two antidepressant medication conditions than with CBT. However, the combined treatment was more likely to produce clinically significant reductions in CTTHs than either antidepressant medication alone or CBT alone.

The combination of antidepressant medication and CBT appears to be a promising treatment for chronic tension-type headache, particularly for patients who do not respond to one of the individual treatments. Nonetheless, methods of enhancing the effectiveness of this treatment are needed, because many patients continue to experience frequent headaches even following the combined treatment. Information about the long-term treatment outcomes with CTTH are also needed, because CTTH may be more prone to relapse than ETTH, particularly following withdrawal of antidepressant medication. The possibility that CBT or other psychological interventions can help CTTH sufferers successfully withdraw from antidepressant medication also deserves evaluation.

In spite of recent advances in medicine, it has been noted that most individuals with a recurrent headache disorder do not have the means to effectively manage their headaches (Adelman, 2000; Dowson & Jagger, 1999; Lipton, Stewart & Simon, 1998). In addition, a third of patients who receive medical treatment for headache problems discontinue treatment because they are dissatisfied with the care they receive (Edmeads, Findlay, Tugwell, Pryse-Phillips, Nelson & Murray, 1993). Advances in drug therapy alone are unlikely to remedy these difficulties. Rather, effective headache management may require that individuals be empowered to manage their own headache problems. Psychological treatment strategies can play a central role in empowering patients, but to do this effectively psychological treatment strategies must adapt to developments in the medical and public health sciences and rigorously assess the benefits and limitations of psychological interventions in the clinical settings where headache problems are treated.

Summary

Already, there is good empirical evidence supporting the use of cognitive behavioural therapy alone or in conjunction with other theapies, in headache (Holyroyd, 2002). However, Holyroyd, O'Donnell, Stensland, Lipchik, Cordingley & Carlson (2001) have reported that while the effectiveness of CBT is relatively well established for chronic tension type headache, studies are needed to evaluate the feasibility of integrating CBT into primary practice and speciality medical settings.

1.10 Conclusion

Evidence from the literature reviewed thus far suggests that chronic pain can best be understood as a multidimensional concept, which involves complex relationships between biological, psychological and social factors. It follows that treatment of chronic pain can only be partly successful if only one of these factors is addressed. Treatment success requires a holistic approach that encompasses those factors deemed to be important in the maintenance of chronic pain.

Recent research has focused heavily on the role of psychological factors in the development and maintenance of chronic pain conditions. Psychological models of pain have been developed which incorporate predisposing, precipitating and maintaining factors in the development of chronic pain conditions. Further research has been devoted to identifying specific common factors involved in the maintenance of chronic pain. Identified factors include the presence of psychopathology, catastrophic thinking, external locus of control/ poor self efficacy beliefs, current stage in the stages of change model, level of social support, medication usage and type of pain. Evidence suggests that each individual will vary in terms of the relative contributions that each of these specific maintaining factors plays in the perpetuation of their particular chronic pain condition.

In accepting the presence of differences in the relative contributions of each of these factors, an obvious question arises about how effective generic treatment packages are for individuals with chronic pain. A review of the literature indicates some degree of variability in terms of the effectiveness of generic CBT packages in chronic pain. In studies specifically related to episodic and chronic forms of tension-type headache, however, effectiveness of CBT is relatively well established. The majority of studies which support CBT, have involved patients from primary care. Studies are now needed to evaluate the effectiveness of on patients from speciality medical settings.

1.11 Aims of Study and Hypotheses

The aim of this research was to examine the effectiveness of Cognitive Behavioural Therapy on patients with Chronic Tension Type Headache referred from an acute medical setting. This study also aimed to the examine the impact of therapy on other areas known to be implicated in the maintenance of chronic pain conditions. From the review of the literature outlined above, it was hypothesised that:

1. Treatment as Usual plus Cognitive Behavioural Therapy participants will show a significant reduction in Headache Frequency, Headache Duration, Headache Pain Severity and Medication Usage compared to Treatment as Usual Participants over time

2. Treatment as Usual plus Cognitive Behavioural Therapy participants will show a significant shift towards the latter stages of the pain stages of change model (as measured by the PSOCQ) compared to Treatment As Usual Participants over time.

3. Treatment as Usual plus Cognitive Behavioural Therapy participants will show significantly stronger beliefs concerning controllability of headaches (as measured by the Multidimensional Health Locus of Control Scale) compared to Treatment As Usual Participants over time.

4. Treatment as Usual plus Cognitive Behavioural Therapy participants will show a significant improvement in levels of emotional well being (as measured by Beck Depression Inventory II and the Beck Anxiety Inventory) compared to Treatment As Usual Participants over time.

5. Treatment as Usual plus Cognitive Behavioural Therapy participants will show a significant improvement in levels of satisfaction with both emotional and practical social support (as measured by the Significant Others Scale) compared to Treatment As Usual Participants over time.

2. Method

Design

The study followed a 2 (Group)*2(Assessment) repeated measures design, which assessed the effect of a cognitive behavioural intervention upon headache frequency, severity and medication usage in chronic tension type headache sufferers. The design also assessed what effect therapy had on other variables known to be involved in the maintenance of chronic pain.

2.1 Participants

A total of 24 participants were recruited from an outpatient Headache Clinic run at the Western General Hospital. Participants were included in the current study if they were diagnosed by the clinic specialist (R.C.) as having chronic tension type headache according to criteria devised by the Headache Classification Committee of the International Headache Society (1988).

Exclusion criteria included, experience of migraine, evidence of tumour, recent substance abuse, systemic disease, a history of head trauma and involvement in other forms of current psychotherapy.

A total of 81 patients were randomly assigned to “treatment as usual” (TAU) or “a treatment as usual plus cognitive behavioural therapy group” (TAU+CBT). Of these patients 44 participants were allocated to the TAU+CBT group and 37 participants were allocated to the TAU groups. Following dropouts a total of 12 participants completed therapy in the TAU+CBT group and 23 participants remained in the TAU group at week 10.

Informed consent to be involved in the research project was obtained from each participant. The clinic specialist was responsible for approaching the patients regarding the study and supplying both the patient information sheet and consent form (appendix A) prior to contact by the principal researcher.

Following their agreement to take part an initial interview was arranged during which all participants were asked to complete a series of questionnaires:

2.2 Measures

2.2.1 Initial Assessment Data

Demographic information regarding age, gender, education and number of months since chronic headache onset were recorded. Using a diary format (appendix) information was also collected over 10 weeks on number of headaches per day, severity of the headaches rated on a likert scale of 0 (no pain) to 10 (maximum pain), duration of the headache in hours, type and number of tablets taken of over the counter medication.

2.2.2 Questionnaires

2.2.2.1 Psychological Health

A number of measures were used to assess psychological health.

- The Beck Anxiety Inventory (Beck, Epstein, Brown & Steer, 1988) is a 21 item self report measure designed to assess the severity of anxiety symptoms. Each BAI item is rated on a 4 point scale from 0 (not at all) to 3 (severely, I could barely stand it). The summed score on all items of the BAI (range 0-63) taps the severity of anxiety symptoms. Scores of <7 indicate minimal levels of anxiety, scores of 8-15 indicate mild levels of anxiety, scores of 16-25 indicate moderate levels of anxiety and scores of 26-63 indicate severe levels of anxiety. The Beck

Anxiety Inventory shows good internal consistency, convergent validity and reliability (Beck, Epstein, Brown & Steer, 1988; Steer, Beck, Brown & Beck, 1993)

- The Beck Depression Inventory II (Beck & Steer, 1987) is a 21 item self report measure designed to assess severity of depression in adults and adolescents aged 13 years and older. Each BDI II item comprises 4 representative statements that are related to depressive symptoms and attitudes. These statements are arranged based on the severity of their content and assigned an ascending score from 0-3. The measure requires individuals to select the statement that is most relevant to the way they have been feeling over the last week. A total score is derived by summing up all item scores. This summed score (range 0-63) taps the severity of depressive symptoms. Scores of <13 indicate minimal levels of depression, scores of 14-19 indicate mild levels of depression, scores of 20-28 indicate moderate levels of depression and scores of 29-63 indicate severe levels of Depression. The Beck Depression Inventory II shows good internal consistency, convergent validity and test-retest reliability (Beck, Steer and Brown, 1996).

- The Pain Stages of Change Questionnaire (Kerns et al. 1997) is a thirty item self report questionnaire designed to assess readiness to adopt a self management approach in relation to pain. Individuals are asked to rate a series of statements across four scales representing four stages of change, precontemplation, contemplation, action and maintenance, on a scale of 1 (strongly disagree) to 5 (strongly agree). Scores are summed across each scale and the scale with the highest score determines the stage of classification for that individual. The individual scales show good internal consistency and test-re-test reliability (Kerns et al. 1997).

- The Significant Others (Scale B) *(Power, Champion & Aris, 1988)* is a self report questionnaire designed to assess four different social support functions (2 emotional and 2 practical) in key individuals selected by the respondent. For each of the four social support functions, each chosen individual is rated in terms of the level of support received and the ideal level of support. Ratings are made using seven point scales from 1 (never) and 7 (always). This instrument is flexible and users decide how many individuals they wish to rate. Participants in this study were asked to select three individuals. Raw scores are obtained for each type of support for each individual on the questionnaire. Scores can be summed across individuals to give separate measures of emotional support and practical support. These scores are then divided by the number of individuals to give a mean for each type of support. Three separate indices can be derived for both emotional and practical support these are: *actual level of support, ideal level of support* and the calculated *discrepancy* between actual and ideal. This discrepancy provides an index of likely satisfaction with available support in each area. This questionnaire has good test-re-test reliability and criterion validity.

- The Multidimensional Health Locus of Control Scale (Wallston, Wallston & DeVellis, 1978) is a multidimensional, 18-item three-factor scale derived from Rotter's internal/external locus of control studies and is widely used in health behaviour research. Factor scores are generated for three subscales: (1) Internal; (2) Powerful Others; and (3) Chance. Internal refers to the belief that the locus of control for health is internal and that one stays or becomes healthy or sick as a result of his or her behaviour. Powerful Others refers to the expectancy that primarily doctors and other health professionals determine health. The Chance subscale refers to generalised expectancies that factors, which determine health, are such things as luck, fate, or chance. On each item individuals are asked to rate their belief in a statement regarding responsibility for health. Individuals answer on a six point scale from *strongly disagree* to *strongly agree.* Raw scores for each scale are summed to give a total score for that scale. Scores range from 6-36 for each factor scale. Higher scores for each scale indicate greater adherence to this particular belief. The test-retest reliability of the measure is established (Wallston, & Wallston, 1981). The internal consistency of the individual scales are all below $\alpha = .8$ and this raises some question about the unity of each of the scales.

On average these questionnaires took approximately 20-25 minutes to complete.

2.3 Procedure

All participants were followed over a 10-week period. Over this 10-week period participants were asked to record information relating to the headaches and medication usage on a daily basis. At the end of the 10- week period all participants were again asked to complete the same series of questionnaires as described above.

2.3.1 Protocol for Groups

- **Treatment as usual group (TAU) (n=12)**

Participants assigned to this group were followed up as usual by medical practitioners at the Headache Clinic. They completed the questionnaires at week one and again at week ten (see figure 2)

Week 1

Participants were given a set of questionnaires to complete and a headache diary. Time was taken to explain how to fill in the diary over the next 10 weeks.

Week 10

Participant were re-contacted and given the second set of questionnaires. Participants were asked to hand in their diaries. No further contact was made following this.

- **Treatment as usual plus cognitive behavioural therapy group (TAU+ CBT) (n=12)**

Participants assigned to this group were followed up as usual by medical practitioners at the Headache Clinic. In addition, they underwent a 10-week course of cognitive behavioural therapy. Individuals completed the questionnaires at week one and again at week ten.

Throughout the 10 weeks all participants were asked to fill in a daily headache diary recording daily headache frequency (number of headaches per day), headache duration (hours), pain severity (0-10) and over the counter medication usage (number of tablets per day)

2.3.2 Therapy Protocol (See Fig 1)

Session One

This session was dedicated to performing an initial psychological assessment. This involved noting the presenting problem, identifying the background history to this problem along with current coping strategies. Details were taken on educational history, family background and current circumstances. A formulation of the presenting problem was developed based on this initial information. At this session participants were asked to fill in the first series of questionnaires and time was taken to explain the way in which the diaries should be completed for the following ten weeks.

Session Two

This was a session used to give a comprehensive overview of the development of chronic pain using a biopsychosocial diathesis-stress model. This allowed the therapist to deliver information relating to the psychological factors involved in the development and maintenance of chronic pain without excluding the obvious physical basis of the headaches. Information included, role of anxiety, fear and avoidance behaviours (including reduction in physical activity), cognitive thinking errors relating to pain and role of significant others. Also during this session issues relating to over the counter medication usage in chronic pain were addressed. This was an "add- on" and requested by the specialist at the headache clinic. On the advice of the specialist emphasis was placed on stopping over the counter medication usage due to its rebound effects. A motivational interviewing approach was taken to explore reasons why individual participants found it difficult to stop over the counter medication usage. Time was spent exploring ways in which to reduce medication usage

Session Three (Relaxation)

Building on the information given in week two, relating to the impact that anxiety and tension have on chronic pain, this session was dedicated to learning relaxation techniques. This was done in session by use of a progressive muscular relaxation audio tape which was copied for home use purposes. The use of diaphragmatic breathing was also demonstrated during this session. Participants were instructed to use these techniques through the following weeks on a daily basis.

Session Four (Pacing and Activity)

This session emphasised the danger of over-activity and how this may lead to downward trend in overall activity levels. Time was taken to explain how the over-activity rest cycle develops and strategies were identified that could be used to overcome this. Advice was given on achieving a consistent baseline of activity and the need to pace activity levels. Further information was given on goal setting and ways to plan increases in overall activity levels. This session also incorporated information relating to the importance of maintaining activity, particularly mild exercise.

Session 5 (Thoughts and Pain)

A focus was placed on the relationship between stress and tension and how much pain is experienced. Information was provided on the way that thoughts act as a mediator between situations/events and feelings and behaviour. A distinction was drawn between negative automatic thoughts and positive automatic thoughts in particular relation to pain. Participants were instructed and given written information on how to identify negative automatic thoughts and thinking errors along with techniques to counter them. Homework was set during this session in which participants had to keep a diary of thoughts relating to pain over the following week, they were instructed in the use of countering techniques where negative automatic thoughts were identified.

Session 6 (Review)

This early part of this session was used to evaluate the diaries from the week before. The rest of this session was used to discuss any aspects of the previous sessions participants had questions about and did not clearly understand. If problems were identified during this session time was spent collaboratively seeking a solution.

Session 7 (Review)

This session was used to review the participants progress. As in session 6 this session was also used to discuss any aspects of the previous sessions that participants had questions about or did not clearly understand. If problems were identified during this session time was spent collaboratively seeking a solution.

Session 8 (Review)

This session was used to review progress to date. Participants were asked to fill out another set of questionnaires and to hand in their diaries. This session marked the end of contact.

Figure 8: Graphic Representation of Procedure for Treatment As Usual +CBT

Flow Chart of Study CBT Group

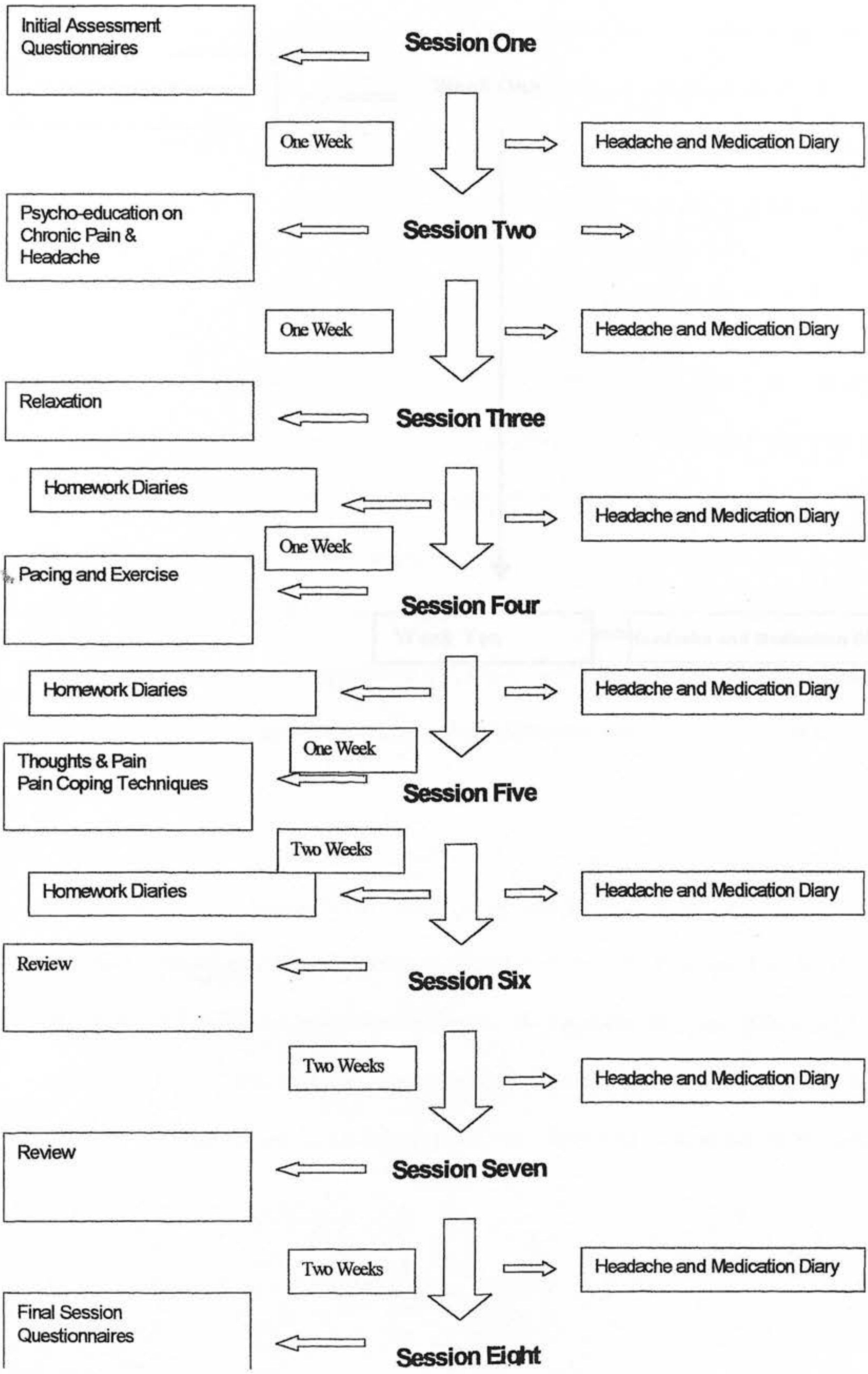
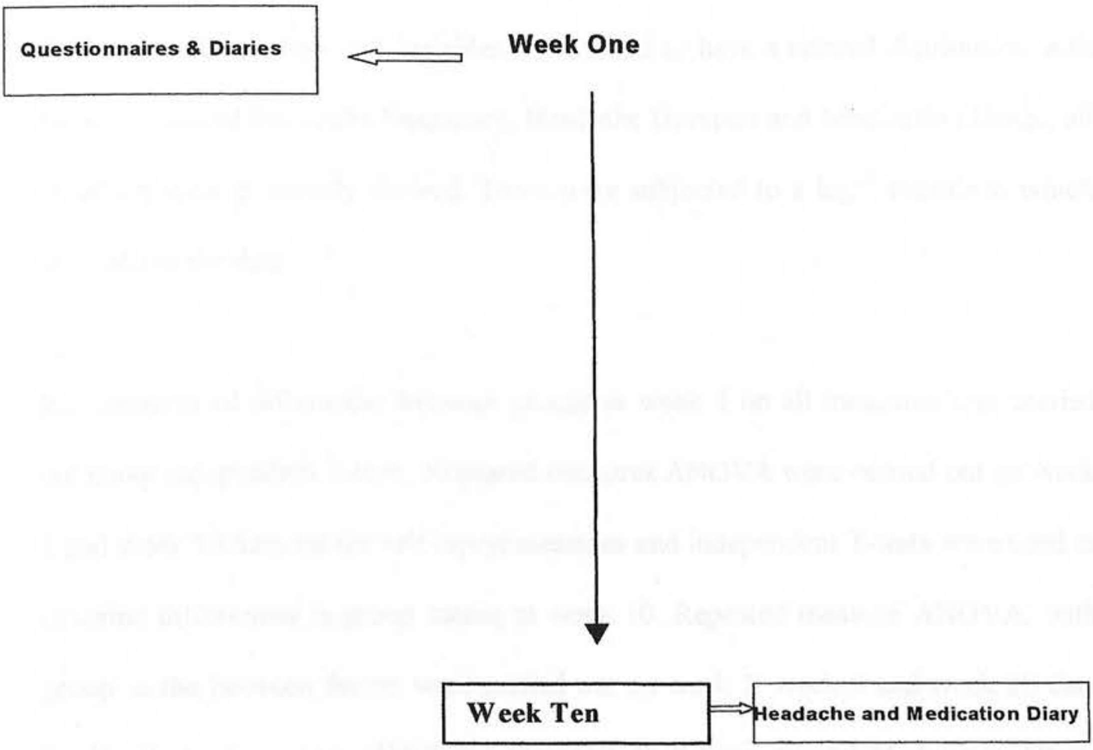

Initial Assessment Questionnaires
Session One
One Week
Headache and Medication Diary
Psycho-education on Chronic Pain & Headache
Session Two
One Week
Headache and Medication Diary
Relaxation
Session Three
Homework Diaries
Headache and Medication Diary
One Week
Pacing and Exercise
Session Four
Homework Diaries
Headache and Medication Diary
One Week
Thoughts & Pain
Pain Coping Techniques
Session Five
Two Weeks
Homework Diaries
Headache and Medication Diary
Review
Session Six
Two Weeks
Headache and Medication Diary
Review
Session Seven
Two Weeks
Headache and Medication Diary
Final Session Questionnaires
Session Eight

Figure 9: Graphic Representation of Procedure Treatment as Usual Participants


Flow Chart of Study Control Group

Questionnaires & Diaries
Week One
Week Ten
Headache and Medication Diary

2.4 Statistical Analysis & Result Reporting

2.4.1 Analyses

Statistical analyses were carried out using SPSS Version 10.1. Data were screened for normal distribution. All variables were found to have a normal distribution with the exception of Headache Frequency, Headache Duration and Medication Usage, all of which were positively skewed. These were subjected to a log^{10} transform which normalised the data.

Examination of differences between groups at week 1 on all measures was carried out using independent T-tests. Repeated measures ANOVA were carried out on week 1 and week 10 data for the self report measures and independent T-tests were used to examine differences in group means at week 10. Repeated measure ANOVA, with group as the between factor, were carried out on week 1, week 5 and week 10 data for Headache Frequency, Headache Duration, Pain Severity and Medication Usage independent T-tests were used to examine differences in group means at week 5 and 10.

Regarding the use of parametric statistics for repeated measures, the sample size in this study meant that the central limit theorem could not be utilised to specify the normality of the distribution about the mean. A non-parametric analysis would have employed the Friedman Test. However, this alternative will not accommodate a between subjects factor. In summary, parametric statistics have been used where no non-parametric alternative could be discerned. Given the small sample size an appeal is made citing the reported robustness of parametric tests when some of the assumptions are violated (Clark-Carter, 1997)

2.4.2 Power Analysis

Effect size was based results on the study by Blanchard et al. (1990) which examined the use of cognitive therapy for tension type headache. An effect size of 0.49 was calculated for the treatment group using pre and post treatment means. Using Cohen's Power Tables (1988) to achieve α .8 at .05 level number of subjects required is reported as n=18.

2.4.3 Reporting of Results

In this study we started with α =.05 and power 1-β=.80. As the sample size was too small, the level of power desired was not achieved and consequently the probability of making a Type II error became greater. In order to retain higher power, we have insisted on reporting the results with α= .10 (Cohen, 1992). Obviously, this increases the probability of making a Type I error, and to this end we have erred on the side of caution and referred to them as trends only. Reporting these trends seems justified under the rubric that if the a priori probability of the null hypothesis being false is high, then the probability of making a Type I error is correspondingly decreased, and a lower alpha level is reasonable. In this study we have assumed that the a priori probability is high, based on similar studies, involving larger sample sizes, finding significant differences in the same measures.

3 Results

3.1 Demographic data

Table 4 summarises overall demographic information and demographic information relating to treatment and control participants.

Table 4: Summary of Demographic Data

	Demographic Data		
	Treatment (n=12)	Control (n=12)	Overall (n=24)
Gender			
Male (n)	5 (41.7%)	4 (33.3%)	9 (37.5%)
Female (n)	7 (58.3%)	8 (66.7%)	15 (62.5%)
Age (yr.)			
Mean	38.17	42.5	40.33
SD	13.31	12.72	12.92
Education (yr.)			
Mean	15.17	14.58	14.88
SD	2.92	2.64	2.74
Onset (mths)			
Mean	20.08	19.08	19.58
SD	7.14	3.92	5.66

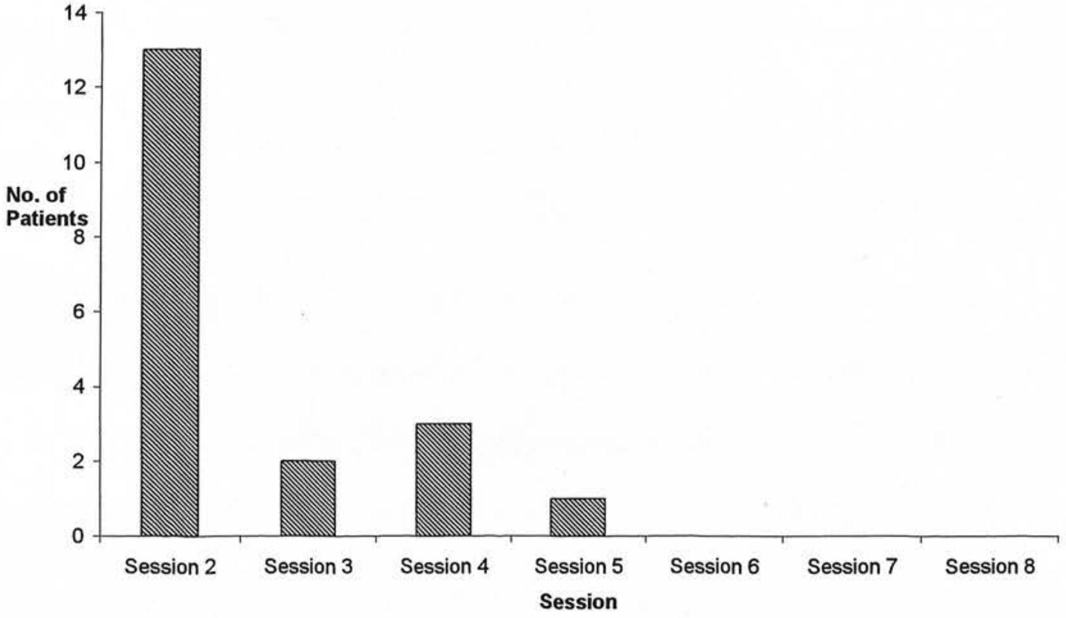
Between and within groups there were more females than males. Independent samples t-tests showed no significant difference between groups in terms of age (t= .815, df=22, p= n.s, 2-tailed), or educational attainment (t= .513, df=22, p= n.s., 2-tailed).

A measure was taken of the length of time individuals had been suffering from chronic pain prior to attending the headache clinic. This measure related to the time the individual had been suffering more than 15 headaches a month. An independent samples t-test revealed no significant difference between length of time since onset of pain between groups (t= .425, df=22, p= n.s., 2-tailed).

3.1.1 Attrition Rate for Therapy

Attrition rate was based on the number of treatment participants recruited into the study who either did not turn up for the initial assessment or attended and did not complete therapy. A total of 44 participants were assigned to the treatment group. Of these 32 (72 per cent) did not complete therapy, this further broke down into 13 (41 per cent) who did not attend the initial assessment and 19 who attended but did not complete therapy (59 per cent). Of these 19 the frequency distribution (graph 1) graphically illustrates the various points in time individuals left treatment.

Graph 1: Frequency Distribution of Patient Attrition (for those who attended first session) Over Treatment Sessions for TAU+CBT.



Retrospective analyses of the explanations for non-completion of therapy is broadly summarised in table 5 below.

Table 5: Explanations for Non Completion of Therapy

Explanation for Non Completion	Number of Patients (n=19)
No Explanation	14
Felt Therapy Was Not Helping	2
Difficulty Attending	3
Due to Pain	*2*
Due to Work Commitments	*1*

Data on patients who did not complete therapy were not included in subsequent analysis. This was due to the fact that patients left at a very early stage of the treatment program and data was therefore incomplete.

3.2 Self Report Measures

3.2.1 Pain Stages of Change Questionnaire

Following the protocol established by Jensen, Nielson, Romano, Hill & Turner (2000) each participant was classified into one of the four stages of change based on his or her highest PSOCQ scale score. In those cases where an individual exhibited two or more scale scores that were equal, the individual was placed into the "higher" of the two stages this again followed the precedent set by (Jensen et al. 2000). This only occurred two times (out of the 24 participant) at week 1 and four times at week 10. Distribution of participants at week 1 and week 10 is summarised in Table (6).

Table 6: Distribution of Participants in Each Stage of Change at Week 1 and Week 10

	Pain Stages of Change Questionnaire							
	Precontemplation		***Contemplation***		***Action***		***Maintenance***	
	Pre	***Post***	*Pre*	***Post***	*Pre*	***Post***	*Pre*	***Post***
Treatment	*0*	***0***	*6*	***1***	*3*	***0***	*3*	***11***
	(0 %)	***(0 %)***	*(50 %)*	***(8.3 %)***	*(25 %)*	***(0 %)***	*(25 %)*	***(91.6 %)***
Control	*0*	***0***	*8*	***10***	*0*	***1***	*4*	***1***
	(0 %)	***(0 %)***	*(66.6 %)*	***(83.3 %)***	*(0 %)*	***(8.3 %)***	*(33.3 %)*	***(8.3 %)***
Overall	*0*	***0***	*14*	***11***	*3*	***1***	*7*	***12***
	(0 %)	***(0 %)***	*(58.3 %)*	***(45.8 %)***	*(12.5 %)*	***(4.16 %)***	*(29.2 %)*	***(50 %)***

An analysis of the distribution of ranks between groups was conducted using a Mann-Whitney U Test which revealed no significant difference (U=66.0, p= n.s.) at week 1. A further analysis of the distribution of ranks was conducted at week 10 this revealed a significant difference (U=12.5, p=.001). This suggests that

Table 7: Means, Standard Deviations & Results from Repeated Measures ANOVA on Self Report Measures

	Overall (n=24)		*Treatment (n=12)*		*Control (n=12)*		*Main effects*		*Interactions*	
	Pre	**Post**	**Pre**	**Post**	**Pre**	**Post**	**F**	**p**	**F**	**p**
Pain stage of change										
Precontemplation	2.70 ± .42	2.47 ± .33	2.85 ± .41	2.37 ± .28	2.55 ± .39	2.57 ± .36	8.02	.01	9.42	.006
Contemplation	3.48 ± .50	3.26 ± .60	3.48 ± .40	2.92 ± .41	3.47 ± .60	3.61 ± .56	3.52	.07	9.20	.006
Action	3.18 ± .51	2.58 ± .47	3.30 ± .45	2.23 ± .27	3.07 ± .56	2.92 ± .36	27.86	.0001	16.14	.001
Maintenance	3.13 ± .66	3.41 ± .60	3.02 ± .83	3.83 ± .44	3.24 ± .46	2.99 ± .40	8.32	.009	29.73	.0001
Health LOC										
Internal	20.88 ± 6.1	23.83 ± 3.1	22.25 ± 5.4	25.9 ± 2.2	19.50 ± 6.6	21.75 ± 2.3	6.29	.02	.36	ns
Chance	20.46 ± 5.9	21.08 ± 5.5	20.75 ± 4.6	19.08 ± 5.7	20.17 ± 7.1	23.08 ± 4.8	.22	ns	2.98	.10
Powerful others	18.71 ± 7.8	18.13 ± 7.5	19.5 ± 6.4	17.75 ± 6.1	17.92 ± 9.2	18.5 ± 9.0	2.33	ns	9.31	.006
BDI	19.5 ± 9.4	17.54 ± 8.9	19.0 ± 10.9	15.33 ± 9.0	20.0 ± 8.0	19.75 ± 8.6	1.80	ns	1.37	ns
BAI	14.42 ± 9.8	12.08 ± 10.2	13.75 ± 10.4	9.83 ± 10.6	15.08 ± 9.6	14.3 ± 9.6	2.96	.10	3.22	.08
SOS – emotional										
Actual	10.07 ± 2.31	10.31 ± 2.66	10.56 ± 1.80	11.0 ± 2.37	9.58 ± 2.71	9.61 ± 2.84	.10	ns	.08	ns
Ideal	12.13 ± 1.51	12.47 ± 1.56	12.64 ± 1.42	12.47 ± 1.70	11.61 ± 1.48	12.47 ± 1.47	.56	ns	1.23	ns
Discrepancy	2.06 ± 2.09	2.17 ± 2.36	2.08 ± 2.17	1.47 ± 2.07	2.03 ± 2.11	2.86 ± 2.51	.07	ns	2.84	.10
SOS – practical										
Actual	9.58 ± 2.44	9.71 ± 2.54	9.58 ± 2.54	10.44 ± 2.53	9.58 ± 2.44	8.97 ± 2.43	.02	ns	.83	ns
Ideal	11.96 ± 1.83	12.01 ± 1.89	12.33 ± 2.24	12.17 ± 2.24	11.58 ± 1.30	11.86 ± 1.55	.03	ns	.12	ns
Discrepancy	2.47 ± 1.86	2.31 ± 2.03	2.94 ± 1.89	1.72 ± 1.95	2.0 ± 1.79	2.89 ± 2.01	.17	ns	6.88	.02

some participants had shifted classification over the 10 week period. Further examination of Table 6 suggests that the treatment group shows reduction in the distribution of participants in the contemplation and action stage and an increase in numbers in the maintenance stage at week 10. The control group shows a surprising opposite trend with a reduction in the distribution of participants in the maintenance stage and an increase in the contemplation stage at week 10.

3.2.2 Repeated Measures on Pain Stages of Change Questionnaire

Analyses revealed no significant differences between the treatment and control group at baseline (week one)

Separate repeated measure ANOVA's were carried out on each of the sub scales of the Pain Stages of Change Questionnaire comparing data at week one and week ten, to assess the main effect of time on measures. Interaction effect between treatment and control subjects were also assessed using repeated measures ANOVAs. Results of the repeated measure ANOVA's can be viewed in Table 7 and are described in text.

3.2.2.1 Precontemplation Scale

A significant main effect was observed with a significant decrease in scores found over time. A significant between group interaction was characterised by a reduction in means for scores for the treatment group and no change in means for the control group (see Table 7).

3.2.2.2 Contemplation Scale

There was no effect of time on this variable. However, a significant interaction was characterised by a small increase in means for the control group and a decrease in means for the treatment group (see Table 7). An independent t-test revealed a significant difference in means (t=3.47, df=22, p=.002, 2-tailed) at week 10 with a higher mean for control.

3.2.2.3 Action Scale

A significant main effect was observed with a decrease from baseline to post-treatment. A significant interaction was characterised by a greater decrement in means for the treatment group compared to the control group (see Table 7). An independent t-test revealed a significant difference in means (t=5.31, df=22, p=.000, 2-tailed) at week 10 with a higher mean for control.

3.2.2.4 Maintenance Scale

A significant main effect was observed along with a significant interaction. The interaction was characterised by an increase in means for the treatment group and an overall decrease in means in the control group (see Table 7) An independent t-test revealed a significant difference in means (t=4.89, df=22, p=.000, 2-tailed) at week 10 with a lower mean for control

3.2.2 Multidimensional Health Locus of Control

Table 7 shows overall means and standard deviations on the Internal, Chance and Powerful Others sub-scales as well as means and standard deviations for the control and treatment groups on these sub-scales at week 1 and week 10.

3.2.2.1 Internal Sub Scale

A significant main effect was found with an increase in this scale from week 1 to week 10 (see Table 7). There was no significant interaction between groups. An independent t-test revealed a significant difference in means (t=4.582, df=22, p=.000, 2-tailed) at week 10 with a higher mean for treatment group.

3.2.2.2 Chance Sub Scale

No significant main effect or interaction was found (see Table 7). An independent t-test revealed no significant difference in means (t=1.86, df=22, p= n.s., 2-tailed) at week 10.

3.2.2.3 Powerful Others Scale

No significant main effect was observed for this subscale although a significant interaction was revealed which was characterised by an increase in means for the control group and a decrease in means for the treatment group (see Table 7). An independent t-test revealed no significant difference in means (t=.240, df=22, p= n.s., 2-tailed) at week 10.

3.2.3 Beck Depression Inventory

Means and standard deviation for the Beck Depression Inventory at week 1 and week 10 can be seen in Table 7. No main effect or interaction effect were observed for this scale (see Table 7). An independent t-test revealed no significant difference in means (t=1.22, df=22, p= n.s., 2-tailed) at week 10.

Using available cut-off scores participants were classified into one of four groups based on their overall score: minimal (0-13), mild (14-19), moderate (20-28) and severe (29-63). Table 8 shows the distribution of participants in each of the groups at week 1 and week 10.

Table 8: Distribution of Participants Across Categories of Severity at Week 1 and Week 10 on the Beck Depression Inventory

	Beck Depression Inventory							
	Minimal		Mild		Moderate		Severe	
	Week 1	Week 10	Week 1	Week 10	Week 1	Week 10	Week 1	Week 10
Treatment	4	6	3	4	4	1	1	1
(n=12)	(33.3 %)	(50 %)	(25 %)	(33.3 %)	(33.3 %)	(8.3 %)	(8.3 %)	(8.3 %)
Control	2	2	4	4	4	3	2	3
(n=12)	(16.7 %)	(16.7 %)	(33.3 %)	(33.3 %)	(33.3 %)	(25 %)	(16.7 %)	(25 %)
Overall	6	8	7	8	8	4	3	4
(n=24)	(25 %)	(33.3 %)	(29.2 %)	(33.3 %)	(33.3 %)	(16.7 %)	(12.5 %)	(16.7 %)

An analysis of the distribution of ranks between groups was conducted using a Mann-Whitney U Test which revealed no significant difference (U=59.0, p= n.s.) at week 1. A further analysis of the distribution of ranks was conducted at week 10 this also revealed a significant difference (U=40, p=.05). This suggests that some participants had shifted classification over the 10 week period. Examination of the distributions in Table 8 suggests that the distribution of participants in the control groups remains relatively stable between week 1 and week 10. By contrast, the treatment group shows reduction in numbers in the moderate category and an increase in the minimal and mild categories between week 1 and week 10.

3.2.4 Beck Anxiety Inventory

Means and standard deviation for the Beck Anxiety Inventory can be seen in Table 9. There were no significant main effects and no significant interactions between groups over time. An independent t-test revealed no significant difference in means (t=1.32, df=22, p= n.s., 2-tailed) at week 10.

Using available cut-off scores participants were classified into one of four groups based on their overall score: minimal (0-7), mild (8-15), moderate (16-25) and severe (26-63). Table 9 summarises distribution of classification of participants at week 1 and week 10.

Table 9: Distribution of Participants Across Categories of Severity at Week 1 and Week 10 on the Beck Anxiety Inventory

	Beck Anxiety Inventory							
	Minimal		Mild		Moderate		Severe	
	Week 1	Week 10	Week 1	Week 10	Week 1	Week 10	Week 1	Week 10
Treatment (n=12)	4 (33.3 %)	6 (50 %)	3 (25 %)	4 (33.3 %)	3 (25 %)	1 (8.3 %)	2 (16.7 %)	1 (8.3 %)
Control (n=12)	3 (25 %)	2 (16.7 %)	3 (25 %)	4 (33.3 %)	4 (33.3 %)	4 (33.3 %)	2 (16.7 %)	2 (16.7 %)
Overall (n=24)	7 (29.2 %)	8 (33.3 %)	6 (25 %)	8 (33.3 %)	7 (29.2 %)	5 (20.8 %)	4 (16.7 %)	3 (12.5 %)

An analysis of the distribution of ranks between groups was conducted using a Mann-Whitney U Test which revealed no significant difference (U=65.5, p= n.s.). A further analysis of the distribution of ranks was conducted at week 10 this also revealed no significant difference (U=41, p=.078) although there may be a trend toward significance suggesting some participants had shifted classification during the 10 week period. Looking at Table 9 it appears that the control group appears stable in terms of its distribution of participants between week 1 and week 10. By contrast, the treatment group shows reductions in numbers in the moderate and severe categories and increases in numbers in the minimal and mild categories.

3.2.5 Significant Others Scale

Means and standard deviations for the actual level of support, ideal level of support and discrepancy sub-scales, that comprise the practical and emotional levels of support scales, for week 1 and week 10 can be seen in Table 7.

Repeated Measures

No main effects were found for any of the sub-scales (see Table 7). A significant interaction was found for the Practical Support Discrepancy score, which was characterised by an increase in mean for the control group and a decrease in mean for the treatment group. In addition independent t-tests revealed no significant differences in means at week 10 on any of the sub scales.

3.3 Treatment Measures

3.3.1 Treatment Measures (Week 1)

Table 10 displays the means and standard deviations at week 1 (in bold) and week 10 for Headache Frequency, Headache Duration, Headache Pain Severity and Medication Usage. Distribution of data was positively skewed for headache frequency, headache duration and medication usage. Data was subjected to log ^{10} transform for those data sets that were skewed. These transformations resulted in normal distributions for each of the data sets (see table 10)

Independent t-tests revealed no significant differences on Headache Frequency (t=1.77, df=22, p= n.s., 2-tailed), Headache Duration (t=.483, df=22, p= n.s., 2-tailed), Pain Severity (t=.189, df=22, p= n.s., 2-tailed) and Medication Usage (t=.371, df=22, p= n.s., 2-tailed) at week one.

Table 10: Raw (Skewed) and Transformed Data for Headache Frequency, Headache Duration & Medication Usage

	Raw Data				**Transformed Data**			
	Skewness		**Kurtosis**		**Skewness**		**Kurtosis**	
	Stat	**Std Error**	**Stat**	**Std Error**	**Stat**	**Std Error**	**Stat**	**Std Error**
Headache	**2.59**	**.472**	**7.0**	**.918**	**.572**	**.472**	**1.49**	**.918**
Duration	**1.43**	**.472**	**1.33**	**.918**	**-.065**	**.472**	**.064**	**.918**
Medication	**1.13**	**.472**	**1.51**	**.918**	**-.217**	**.472**	**.080**	**.918**

3.3.2 Treatment Repeated Measures

Preliminary analyses of distribution revealed that all week 5 and week 10 measures were normally distributed. Table 12 displays the means and standard deviations at week 1, week 5 and week 10 for Headache Frequency, Headache Duration, Headache Pain Severity and Medication Usage.

For the purposes of repeated measures data that was transformed at week 1 also had to be transformed at week 5 and week 10. Following this transform data maintained its normal distribution. Repeated Measures ANOVA's were conducted on the week 1, week 5 and week 10 data, with group (treatment vs. control) as a between factor. Results of the repeated measures ANOVA's can be seen in Table12.

Table 12: Summary of Main Effects and Interactions

	d.f.	**F**	**Sig.**
Main Effects			
Headache Frequency	**1,22**	**3.77**	**..04***
Headache Duration	**1,22**	**3.74**	**.04***
Pain Severity	**1,22**	**2.85**	**..08**
Medication Usage	**1,22**	**4.31**	**.03***
Interactions			
Headache Frequency* Group	**1,22**	**4.00**	**.034***
Headache Duration* Group	**1,22**	**3.13**	**.06**
Pain Severity* Group	**1,22**	**7.69**	**.003***
Medication Usage* Group	**1,22**	**2.65**	**.09**

* Significant at .05

Table 12: Means and Standard Deviations for Headache Frequency, Headache Duration, Pain Severity & Medication Usage at Week 1, Week 5, Week 5.

	Headache Frequency			Headache Duration			Pain Severity			Medication Usage		
	Week 1	Week 5	Week 10	Week 1	Week 5	Week 10	Week 1	Week 5	Week 10	Week 1	Week 5	Week 10
Treatment												
Mean	1.29	.986	.799	289.87	235.70	156.02	4.15	3.59	2.49	1.77	1.39	.640
SD	1.03	.776	.722	196.29	145.86	97.66	1.66	1.66	1.19	1.58	1.63	.886
Control												
Mean	.745	.658	.699	333.15	288.27	301.51	3.98	3.43	4.03	2.04	2.03	1.93
SD	.264	.350	.264	240.33	263.25	260.23	2.70	2.41	2.59	1.99	1.84	1.92

Independent t-tests were performed on week 5 and week 10 data to examine the presence of group differences. Results of these analyses can be viewed in Table 13.

Table 13: Results of Post Hoc Independent T-tests Conducted a Week 5 and Week 10

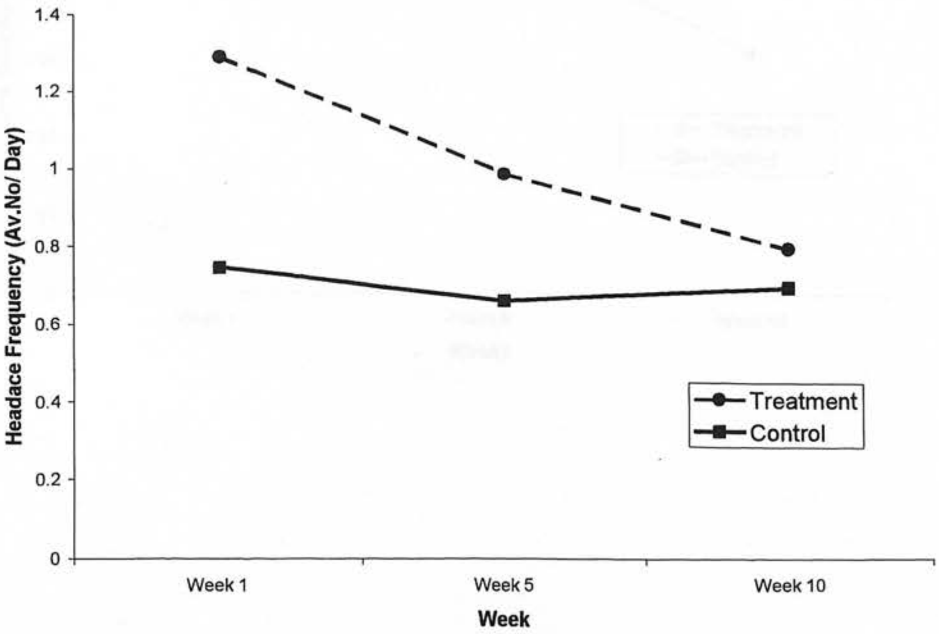
	t	*p*	*df*
Week 5 Comparison			
Headache Frequency	***1.34***	***n.s.***	***22***
Headache Duration	***.605***	***n.s.***	***22***
Pain Severity	***.197***	***n.s.***	***22***
Medication Usage	***.90***	***n.s.***	***22***
Week 10 Comparison			
Headache Frequency	***.452***	***n.s.***	***22***
Headache Duration	***1.813***	***.083***	***22***
Pain Severity	***1.878***	***.074***	***22***
Medication Usage	***2.105***	***.047****	***22***

***** Significant a .05, 2 tailed.***

3.3.2.1 Headache Frequency

Repeated measures analysis of variance revealed a significant main effect for headache frequency between pre-treatment and post-treatment. Inspection of the means showed that there was an overall reduction in headache frequency for both treatment and control participants. A significant interaction was also observed for Group * Headache Frequency with greater reduction of headache frequency over time for the treatment participants. Inspection of the means showed that treatment participants began with a higher headache frequency at week one compared with control participants; this discrepancy was analysed and found to be not significant (see Table 13). The means also demonstrate that treatment participants never achieve the absolute level of reduction found in the control participants but again this discrepancy is not significant

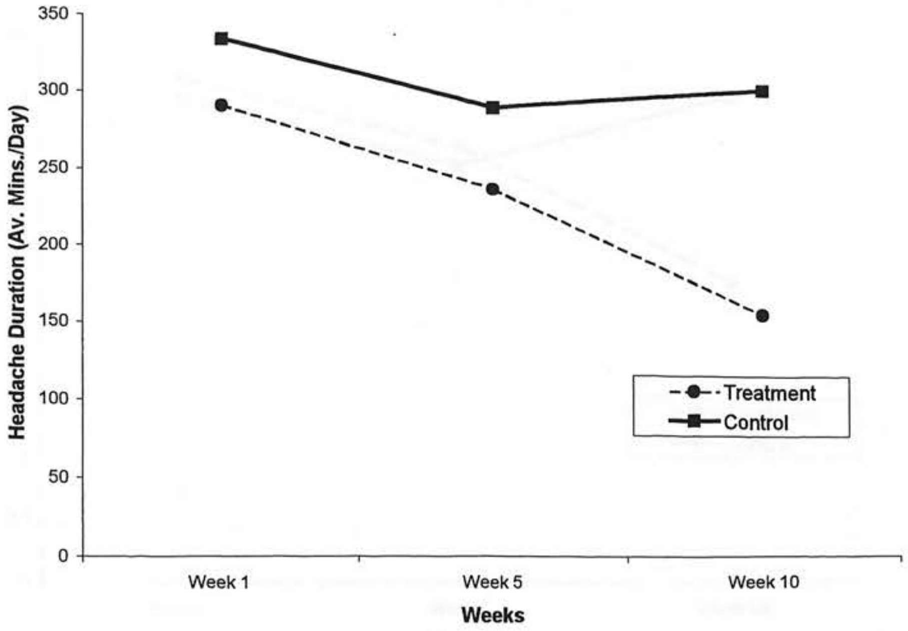
Graph 2: Plot of Average Daily Headache Frequency for TAU and CBT+TAU Groups at Week 1, Week 5 & Week 10



3.3.2.2 Headache Duration

Repeated measures analysis of variance revealed a significant main effect for headache duration. Inspection of the means showed that there was an overall reduction in headache duration for both treatment and control participants. There was a trend towards significance for the interaction Group* Duration implying both groups do not show similar reduction in headache duration over time. Independent t-tests revealed no significant difference between means at week 5 although there was a possible tend towards significance at week 10 (see table 13).

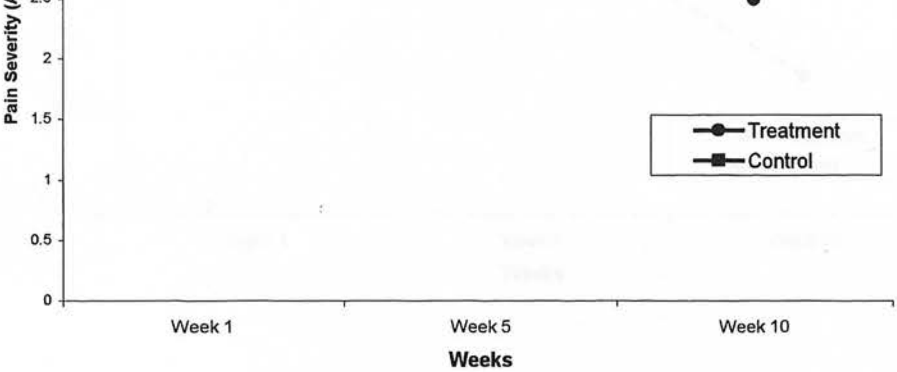
Graph 3: Plot of Average Daily Headache Duration for TAU and CBT+TAU at Week 1, Week 5 & Week 10



3.3.2.3 Headache Pain

Repeated measures analysis of variance revealed a main effect for headache pain. Inspection of the means showed that there was an overall reduction in headache pain severity for the treatment group but a small increase for the control group. A significant interaction was found for Group * Headache Pain Severity. Independent t-tests revealed no significant difference between group means at week 5 although there was a trend towards significance at week 10 (see Table 13).

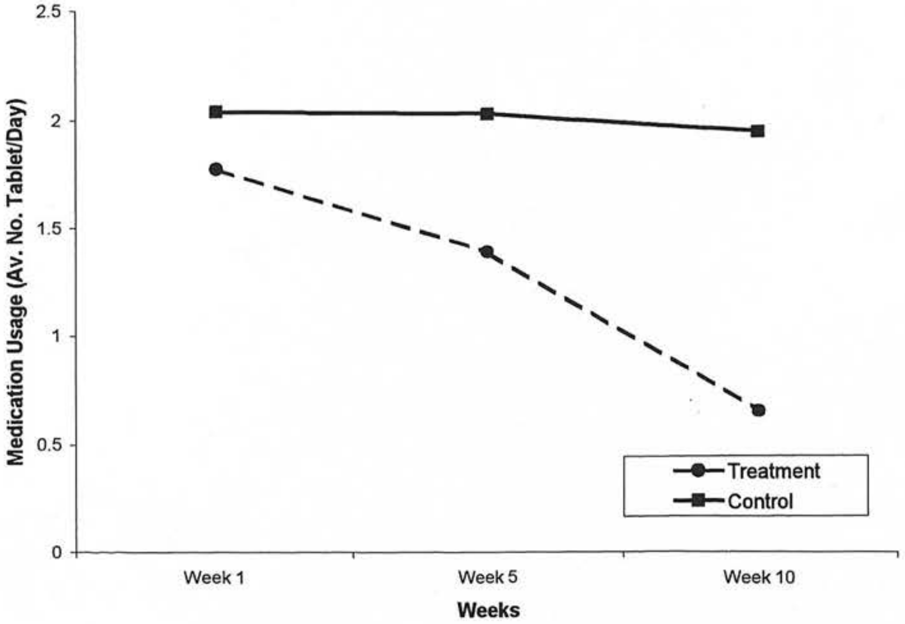
Graph 4: Plot of Average Daily Pain Severity for TAU and CBT+TAU at Week 1, Week 5 & Week 10



3.3.2.4 Medication Usage

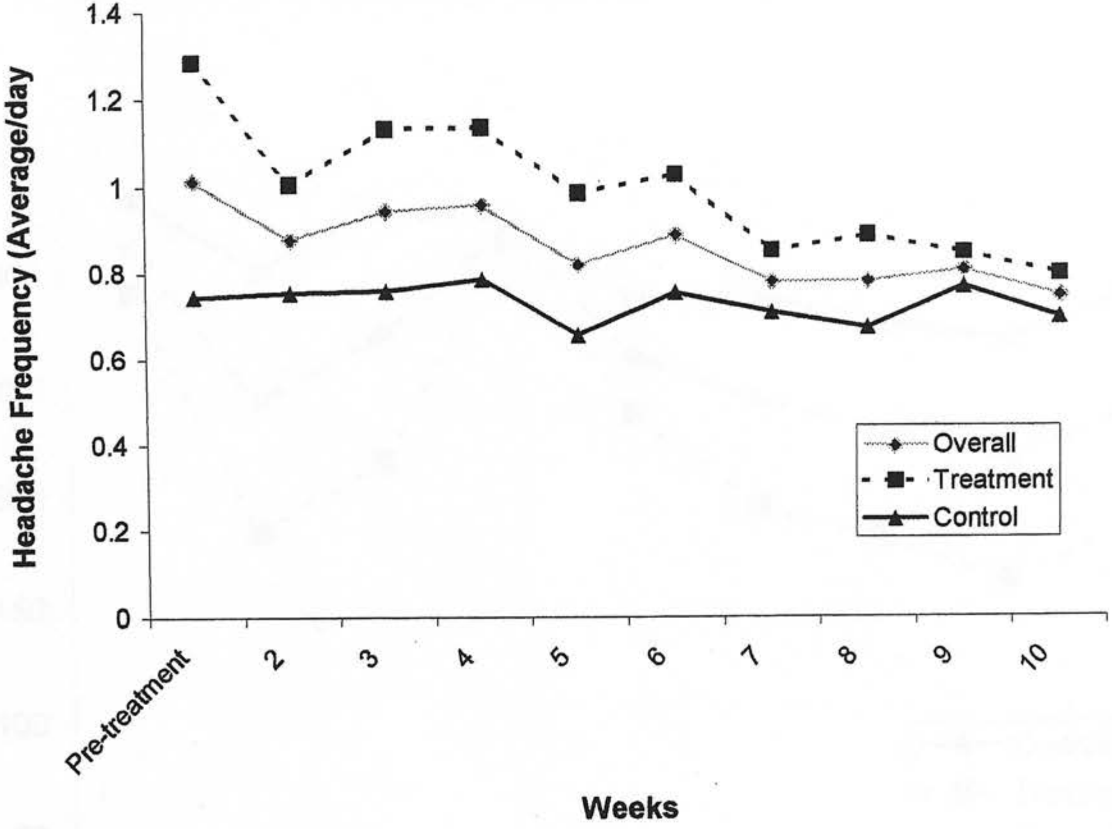
Analyses revealed a main effect for medication usage and a trend towards significance for an interaction for Group * Medication Usage (see Table 11). Independent t-tests revealed no significant difference in means between groups at week 5 but a significant difference between groups at week 10 with controls exhibiting a higher mean than the treatment group (see table 13)

Graph 5: Plot of Average Daily Medication Usage for TAU and CBT+TAU at Week 1, Week 5 & Week 10

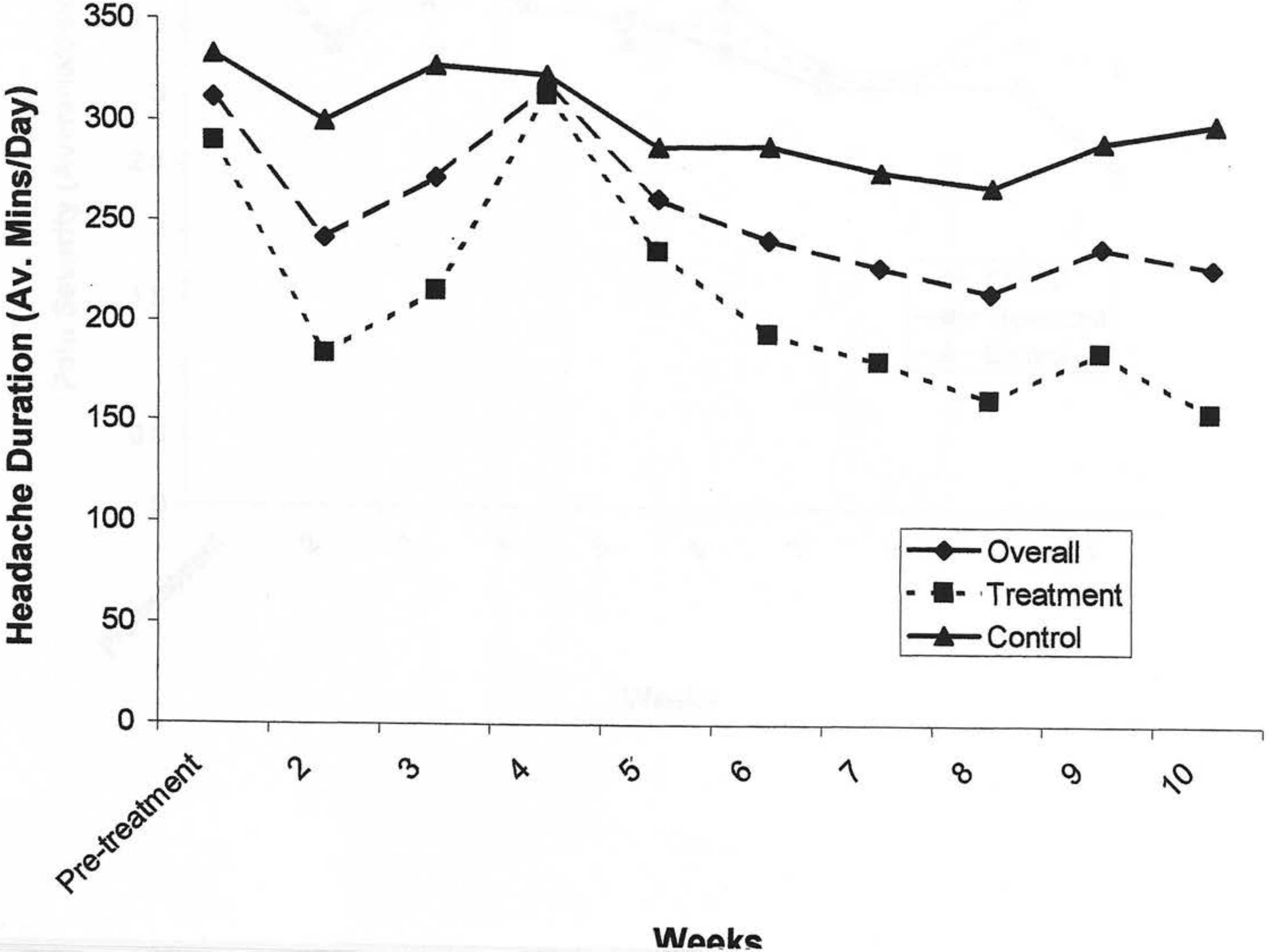


Graphs 6, 7, 8 and 9 have been included to encapsulate all data points collected. The graphs demonstrate that although there is some variation trends over time support results of the statistical analysis using the three data points of week 1, week 5 and week 10.

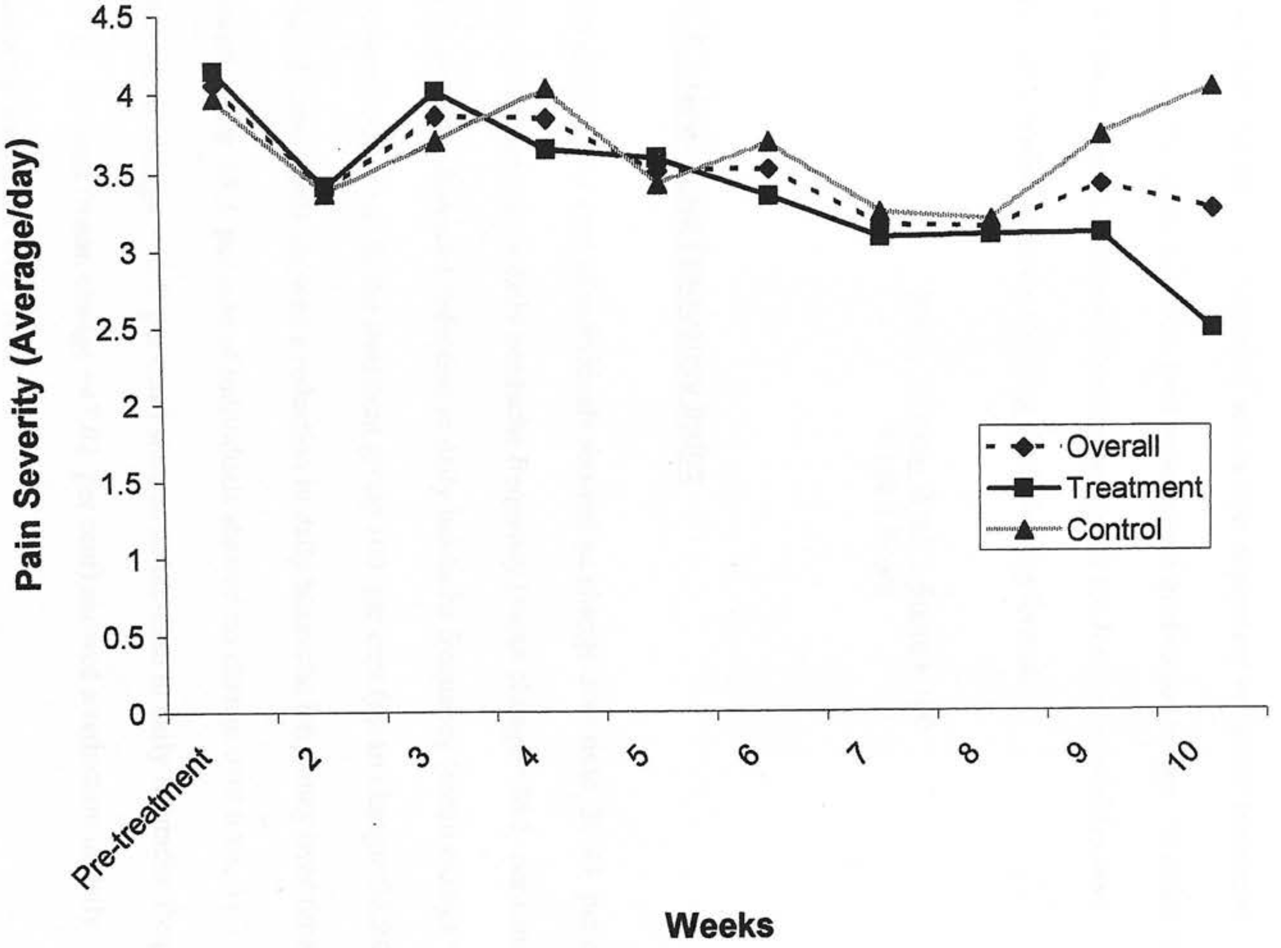
Graph 6: Plot of Headache Frequency over 10 weeks for Treatment And Control Groups



Graph 7: Plot of Headache Duration over 10 weeks for Treatment And Control Groups



Graph 8: Plot of Pain Severity over 10 weeks for Treatment And Control Groups



3.4 Treatment Effectiveness

3.4.1 Success of Treatment

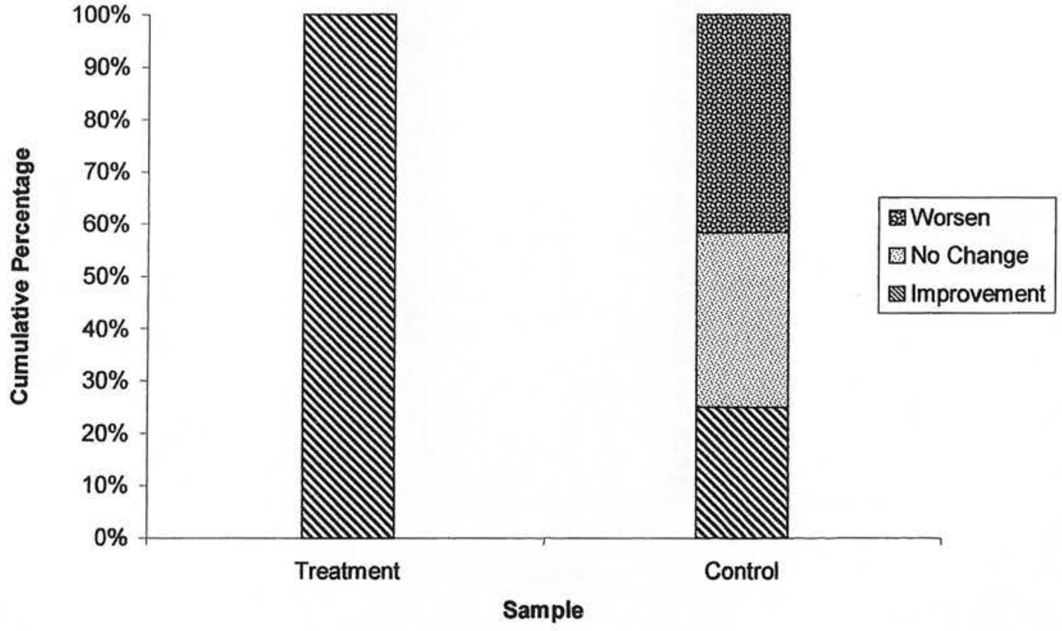
Treatment success was measured across four dependent variables: headache frequency, headache duration, pain severity and medication usage. An index of percentage change between week one and week ten for these variables was calculated within subjects by using the following formula:

$$\frac{\text{Week 1 Score- Week 2 Score}}{\text{Week 1 Score}} \times 100$$

3.4.1.1 Headache Frequency Index

Overall 16.7 per cent of individuals showed no change over time, 20.83 per cent showed an increase in daily headache frequency (mean change =26.5 per cent) and 62.5 per cent showed a reduction in daily headache frequency (mean change =40.1 per cent) over time. In the treatment group 100 per cent (mean change=38.24 per cent) of individuals showed a reduction in daily headache frequency over time. In the control group 33.3 per cent of individuals showed no change over time, 41.7 per cent (mean change =26.5 per cent) showed an increase in daily headache frequency and 25 per cent (mean change =47.62 per cent) showed a reduction in daily headache frequency over time.

Graph 10: Cumulative Percentage Graph of Treatment Success for Headache Frequency

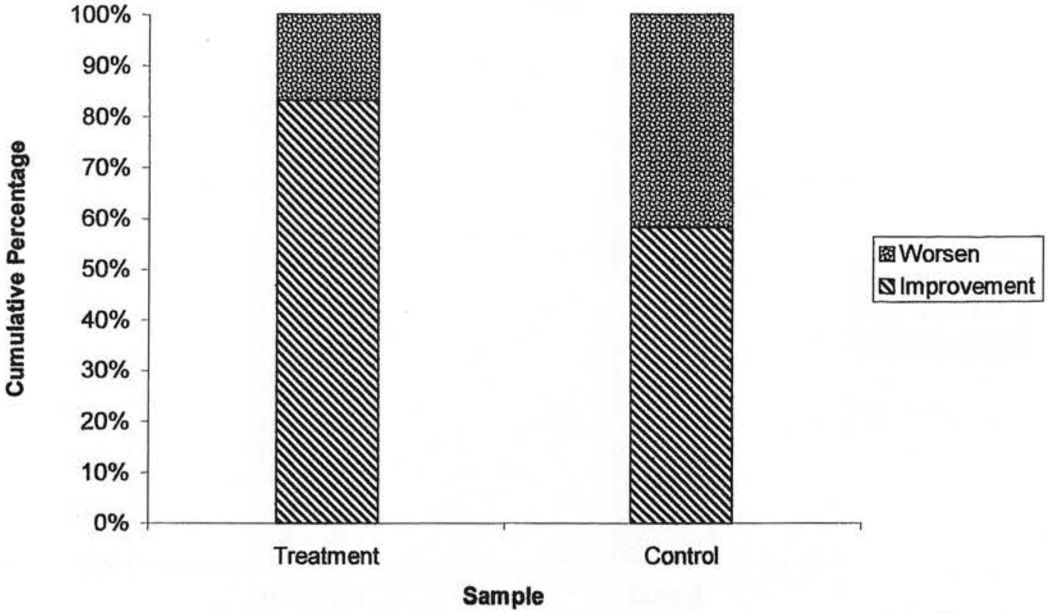


3.4.1.2 Duration of Headache Index

Overall 70.83% (mean change=47.84%) of individuals showed a reduction in headache duration while 29.17% (mean change =43.9%) demonstrated an increase in headache duration. In the treatment group 83.3% (mean change =55.09%) showed a reduction in headache duration while 16.7% (mean change =64.8%) demonstrated an increase in headache duration. In the control group 58.3% (mean change =37.5%)

showed a reduction in headache duration while 41.7% (mean change =35.67%) demonstrated an increase in headache duration.

Graph 11: Cumulative Percentage Graph of Treatment Success for Headache Duration

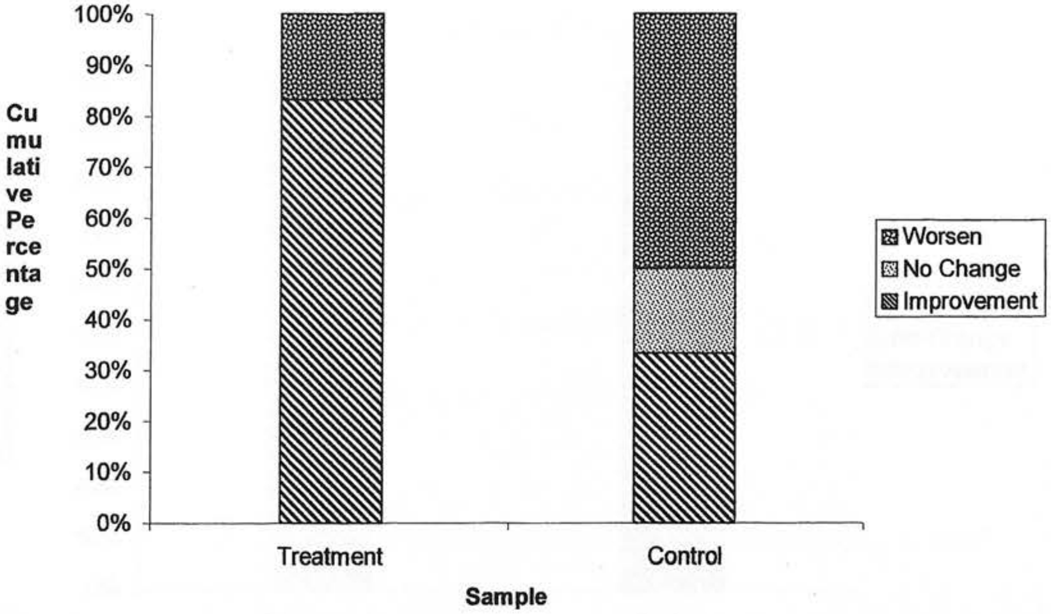


3.4.1.3 Pain Severity

Across groups 58.33% (mean change =41.1%) of individuals showed a reduction in pain severity over time, 8.3% showed no improvement and 33.3% (mean change =99%) demonstrated an increase in pain severity over time. In the treatment group 83.3% (mean change =42.7%) showed a reduction in pain severity while 16.7% (mean change =6.43%) demonstrated an increase in pain severity. In the control group 33.3% (mean change =37.08%) showed a reduction in pain severity, 16.7%

showed no change in pain severity and 50% (mean change =130.61%) demonstrated an increase in headache duration

Graph 12: Cumulative Percentage Graph of Treatment Success for Pain Severity

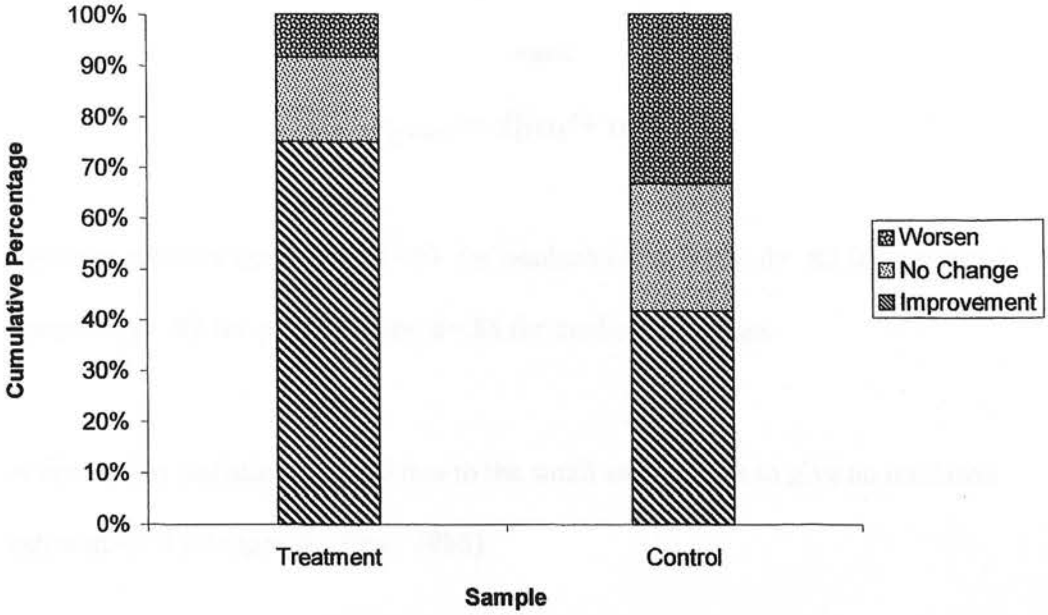


3.4.1.4 Medication Usage

Across groups 58.33% (mean change =57.5%) of individuals showed a reduction in medication usage over time, 20.83% showed no improvement and 20.83% (mean change =55.5%) demonstrated an increase in medication usage over time. In the treatment group 75% (mean change =69.4%) showed a reduction in medication usage, 16.7% showed no change medication usage severity and 8.3% (mean change =60%) demonstrated an increase in medication usage. In the control group 41.7%

(mean change =36.11%) showed a reduction in medication usage, 25% showed no change in medication usage and 33.3% (mean change =54.2%) demonstrated an increase in medication usage.

Graph 13: Cumulative Percentage Graph of Treatment Success for Medication Usage.



3.4.2 Effect Sizes

Effect sizes for the treatment group were calculated on all of the treatment measures using week 1 and week 10 data the equation for two dependent samples below (Rosnow & Rosenthal, 1996, Cohen 1988)

$$\mathbf{d = M_1 - M_2 / \sigma_{pooled}}$$

where

$$\mathbf{\sigma_{pooled} = \sqrt{}[(\sigma_1^2 + \sigma_2^2) / 2]}$$

Calculated effect sizes were d=.55 for headache frequency, d= .82 for headache duration, d=.81 for pain severity, d=.88 for medication usage.

A correction statistic was used due to the small sample size to give an unbiased estimate of d (Hedges & Olkin, 1985)

$$\textbf{Calculated value of } \boldsymbol{d} \times \left(1 - \frac{3}{\{4(N_1 + N_2) - 9\}} \right)$$

The correction statistic was calculated as being 0.95. All effect sizes were then adjusted using this statistic

Adjusted effect sizes were d=.52 (medium) for headache frequency, d= .78 (medium) for headache duration, d=.77 (medium) for pain severity and d=.84 (large) for medication usage.

4 Discussion

The aim of this research project was to examine the effectiveness of Cognitive Behavioural Therapy in chronic tension type headache. Findings are discussed in relation to previous literature, with methodological flaws evaluated. The relevance of these findings in terms of future research is discussed.

4.1 Discussion of main findings in relation to headache measures

4.1.1 Effectiveness of Cognitive Behavioural Therapy

We hypothesised that Cognitive Behavioural Therapy plus TAU would lead to a greater reduction in headache frequency, headache duration, headache pain severity and medication usage over time than compared to TAU alone.

Results of this study provide additional support for Cognitive Behavioural Therapy being an effective approach in chronic pain. This study also provides preliminary support for the inclusion of cognitive behavioural therapy in treating patients seen at a speciality medical setting.

In terms of findings on the headache specific measures there appears to be a general decrease in headache frequency, duration and medication usage over time. Measure of pain severity did not reach formal significance for a main effect but was considered to be approaching significance. These main effects arise due to the decrements in the treatment group. Scrutiny of the interactions from the repeated measures analyses demonstrate that for headache frequency and pain severity the treatment group show greater improvements, in terms of symptom reduction over time, than the control group. Other interactions were approaching significance and given a larger sample size may well have reached significance. In these interactions the trend is for the treatment group to show greater improvement over time compared to controls.

Considering the effect size for each of these measures it is clear that they are in general above the mean effect size of .5 seen in other studies looking at CBT and chronic pain (Holyroyd, 2002, Blanchard et al. 1990; Holyroyd et al. 1977). In addition, the amount of improvement in percentage terms is comparable with estimates derived from a meta-analysis by Goslin, Gray & Mc Rory (1999).

The presence of main effects, interactions and generally large effect sizes are unusual compared to other studies in the general chronic pain literature but not unexpected findings in this study. There are a number of factors, which might have led to these findings.

4.1.1.1 Patient Characteristics

Primarily, patient characteristics appear to play a large role in the variability of findings between studies in the general chronic pain literature. Evidence suggests that patients with rheumatoid arthritis (RA) engaged in CBT and behavioural treatments have non-significant to small effects on indicators of physical and psychological functioning at post-treatment, and non-significant effects at follow-up assessments (Hawley, 1995; Riemsma et al., 2002; see also, McCracken, 1991; Riemsma et al., 1999). By contrast, meta-analyses of CBT for various chronic pain disorders, such as chronic low back pain or osteoarthritis, have revealed more promising effects (Hawley, 1995; Morley et al., 1999; van Tulder et al., 2000). The limited effects of CBT for RA have frequently been ascribed to the heterogeneity of patients. On this basis, it has been argued that not all patients may receive benefits from generic treatments. Indeed it follows that the more heterogeneous the patient sample, the less likely that a generic treatment could encapsulate the varying needs of each individual. In view of the individual variability between patients with chronic pain, various attempts have been made to classify patients into more homogeneous subgroups and identify patients that may benefit from psychosocial interventions. For example, it has been repeatedly shown in various chronic pain disorders that subgroups of patients who are relatively well-adjusted might only receive limited benefits from CBT. Groups of patients with heightened distress levels and dysfunctional cognitive-behavioural factors benefit most from CBT (e.g. Turk and Rudy, 1988, 1990a; Turk, 1990; Main et al., 1992; Klapow et al., 1993, 1995; Strong et al., 1994; Turk and Okifuji, 1998; Gatchel, 2001). Retrospective analyses of CBT effects in fibromyalgia patients provided preliminary support for this view, indicating that patients characterized by high distress levels and dysfunctional cognitive-behavioural factors benefited more from CBT than those relatively well-adjusted or whose impairment was mainly related to social functioning (Turk et al., 1998).

In this study participants were relatively homogenous in that all suffered from a particular type of chronic pain and initial data suggest that the majority of the participants were in the clinical range for anxiety and depression. In addition, it is important to note that there may be something additional about the individuals in this study that we were unable to measure. A 72 per cent drop out rate was noted for the treatment group suggesting that the group completing treatment represented a minority of the patients referred in total. One suggestion is that these patients are qualitatively different from those individuals that dropped out that made them more receptive to cognitive behavioural therapy. This is not a question that can be answered here as the data for the participants who dropped out is limited. However this is a possible avenue of future research.

4.1.1.2 Treatment Specificity

A second factor involves the treatment specificity. Effect studies of CBT usually consist of generic treatments with multiple cognitive and behavioural modules, assuming that the different components are relevant and effective for patients. However, in view of the various problems from which patients with chronic pain suffer, treatment programs tailored to patients' clinical needs may increase the effectiveness of CBT in chronic pain patients (e.g. Turk, 1990; Fry and Wong, 1991; Turk and Okifuji, 1998; Gatchel, 2001). In addition, applying treatment modules matched to individual patient profiles and directed to the outcome from which patients suffer most, is likely to increase patient satisfaction with treatment and decrease attrition rates (see e.g. Turk and Rudy, 1990b). In this study the treatment programme was specific in terms of psychoeducation, which focused on a psychological theory of chronic headache and contained cognitive and behavioural components which made specific to headache. It is interesting to note that the numbers of studies which outline their protocol for therapy are few in number. This raises the issue of how well one CBT study can be compared with another. In addition, it raises a more fundamental issue about what is meant by CBT in various studies.

4.1.1.3 Timing of Treatment

It has been assumed that treatment effectiveness depends on the timing of treatment. For example, it has been suggested that patients develop a relatively stable way of coping with chronic pain over time (Sinclair and Wallston, 2001). It is hypothesised that dysfunctional cognitive-behavioral patterns may be less established and easier to modify at an earlier stage of the disease than later on. This would imply increased CBT effectiveness for patients suffering for a shorter time from their complaints (e.g. Philips and Jahan-shahi, 1985; DeVellis and Blalock, 1993; Peters et al., 2000; Sinclair and Wallston, 2001). In addition, interventions at an earlier stage of the disease have, by definition, a greater chance of having more long-term benefits and possibly preventing a worse long-term disease (Parker & Wright, 1995). Dysfunctional cognitive-behavioural factors, that predict a worse long-term disease outcome in chronic pain patients, have been shown to be already established in the initial years of the disease (Evers et al., 1997, 1998a, 2002; Smith et al., 1997; Kraaimaat et al., 1995; Sinclair and Wallston, 2001).

The patients in this study had a mean onset of chronic pain approximately a year and a half before being seen for cognitive behavioural therapy. Other studies report mean onset duration of headache as being 11.17 years (Johnson & Thorn, 1989), 10.7 years (Holroyd, Nash & Pingel, 1991), 15.6 years (Richardson & Mc Grath, 1989). Clearly patients in the present study are receiving cognitive behaviour therapy at an unprecedented early stage compared to reports in the literature and this may account for observed effect sizes.

4.1.1.4 Combining Treatments

Combing single treatment modalities can increase the effectiveness of intervention (Holroyd, 2002). The most common non-medication treatments for headache are biofeedback, relaxation training, and cognitive therapy. While evidence exists for their effectiveness as single treatment modalities, other studies have examined the effects of combining treatment modalities. One study (Blanchard et al. 1985) showed improvement in 39 per cent of 94 patients with headache using relaxation training alone. Adding biofeedback increased the portion of patients experiencing improvement to 56 per cent. In another study, Blanchard, Appelbaum, Guarnieri, Morrill & Dentinger (1987) reported that combining relaxation with EMG biofeedback led to improvement that was maintained at five years' follow-up.

Other studies have investigated cognitive psychotherapy alone and also in combination with other behavioural treatments for chronic tension-type headache.

Among these trials, at least 50 percent of patients had reduced symptoms when treated with progressive relaxation, cognitive therapy, or a combination of the two (Attanasio, Andraisk & Blanchard, 1987). This study used a combination of cognitive therapy and relaxation, which may have also contributed to the observed effect sizes.

4.2 Discussion of findings in relation to General Measures

4.2.1 Pain Stages of Change Questionnaire

Another question to be considered is in what way treatment achieves effectiveness, or more precisely, how treatment incurs change in individuals. The aim of cognitive therapy in chronic pain is to emphasise the adoption of a self- management approach (Gatchel &Turk, 1996). If treatment is successful, it is assumed that individuals are adopting a transfer of responsibility in the management of their own health. However, this requires a shift in competencies but also a shift in the way they view pain. Clearly, adopting a self-management programme necessitates a readiness to change. The transtheoretical model describes how people modify problem behaviour or acquire a positive behaviour. In chronic pain readiness to change is measured using the Pain Stages of Change Questionnaire. Studies that utilise the transtheoretical model generally aim to predict treatment success based on the particular stage a given individual occupies at a given point in time. No studies in the pain literature have examined the reverse and considered the effects of therapy on this model and how success in treatment may be reflected as progression through the stages of change during therapy.

We hypothesised that successful therapy should incur a shift towards the latter stages of the pain stages of change model for treatment participants. By contrast, control participants should show no such shift. This study found that over the course of therapy the treatment groups showed a shift towards the maintenance stages of the model. It may be hypothesised that the shift in the treatment group reflects an increased commitment to self management and this leads to greater adherence to recommendations of skill acquisition and practice, which may in turn lead to improved pain management. If therapy mediated this change then it was expected that controls would show no such shift in their stage of change. Indeed the control group who showed no such shift in fact some individuals in the control group showed a relapse in terms of the stage they occupied at week 1 and then at week 10.

In this study, individuals in both groups were generally classified in the contemplation, action or maintenance stages at week 1. This is perhaps not surprising as they are already attempting to seek out help in a specialist setting. Indeed, although speculative, many of these individuals may have had numerous investigations to identify the physical cause of the pain suggesting that they are already at least in a contemplation stage if not in an action stage of change.

4.2.2 Multidimensional Heath Locus of Control Questionnaire

We hypothesised that treatment success would reflect an increase in beliefs concerning the controllability of pain. This was partially supported by trends observed in the data. We employed the Multidimensional Health Locus of Control Questionnaire to examine changes over time in internal beliefs, chance beliefs and powerful other beliefs. TAU+CBT participants showed a similar pattern to controls on the internal scale with increased scores over time suggesting that both groups view health as more in their control. By week 10 the scores on the internal scale were significantly higher for the CBT+TAU group than the TAU group. An interaction on the powerful other scale was characterised by a reduction in means over time for the CBT+TAU group while the TAU group showed an increase over time.

Extending these findings, it may be further hypothesised that treatment serves to increase levels of self-efficacy in dealing with pain by changing locus of control beliefs towards internality and away from powerful others. In addition, it might be speculated that in a reciprocal relationship treatment success is dependent upon this change occurring.

4.2.3 Emotional Wellbeing

We hypothesised that treatment success should be reflected in greater emotional well being in TAU+CBT participants in contrast to theTAU participants who should show no such change in wellbeing. We employed two measures to assess wellbeing; the Beck Depression Inventory and the Beck Anxiety Inventory.

4.2.3.1 Beck Depression Inventory

Interestingly, results showed no effect of treatment on depression scores. There was no overall change in these scores over time and no interaction between the groups and scores over time. As previously noted, Romano and Turner (1985) reported that 40-50 per cent of chronic pain patients suffer from depression. In the majority of cases, the depression occurs as a reaction to the development of the chronic pain condition. Other studies have suggested that chronic pain may be a masked form of depression and that emotional dysregulation is expressed in somatic terms (Turk & Salovery, 1984). This latter issue leads to an interesting prediction. If chronic pain is a somatic externalisation of an underlying clinical depression then those therapies that a specifically tailored towards increasing self management of the pain condition only would be expected to have little impact upon the underlying depression. Conversely, if depression is simply a reaction to the onset of chronic pain, then these same therapies should have some degree of impact on overall depression as pain symptoms become more manageable. Results from this study suggest that improvements in the ability to manage pain, as demonstrated by reduction in the various headache measures, do not impact upon depression scores. Taken in isolation this would appear to lend support to the theory that chronic pain is a somatic externalisation of underlying depression.

4.2.3.2 Beck Anxiety Inventory

Results demonstrated an interaction between group and scores on the Beck Anxiety Inventory that was approaching significance. This might be expected to reach significance with a larger sample size. This trend suggests that therapy may have the effect of reducing anxiety scores to an extent not observed in the control group.

As with depression, studies exist that view anxiety as a premorbid characteristic of some individuals with chronic pain. Polatin et al. (1993) found that 95 per cent of those diagnosed with anxiety disorders in their sample of patients with chronic lower back pain had experienced anxiety disorders before the onset of pain. Based on this evidence, it is hypothesised that pain is a somatic expression of anxiety. Other studies such as that of Gatchel, Garoalo Ellis & Holt (1996) suggest that anxiety develops as a reaction to pain and increases in severity as the pain becomes chronic. They found that although chronic pain patients had much higher rates of overall psychopathology than did acute pain patients, anxiety disorders were diagnosed frequently in both groups.

Unlike results from the Beck Depression Inventory, these results suggest that anxiety, certainly for participants in this study, may be a reaction to the development of chronic pain rather than being a premorbid characteristic. Importantly though, while the therapy contained no specific elements relating to coping with depression there was a session devoted to relaxation. Relaxation is a specific skill in learning to deal with anxiety and this may explain the difference in effect of therapy over these two measures of wellbeing.

A final note may be that changes in depression scores occur after a latency in which patients consolidate their skills and continue to self-manage their pain consistently. Data from this study, which analysed shifts in classification (minimal, mild, moderate, severe) on the Beck Depression Inventory, suggest that a number of treatment participants descend through the classification stages following treatment, to lesser classification categories. On a critical note, this analysis removes much of the fine grained detail necessary for firm conclusions. However, it may indicate the beginnings of change not seen in more detailed analysis. Follow up data would clarify this issue of whether change in depression scores occurs following a latency period.

4.2.4 Significant Others Scale

We hypothesised that treatment success would be reflected by increases in social support satisfaction. This literature relating to social support in chronic pain is replete with studies that implicate social supports as discriminative cues for illness behaviour (Block et al.,1990, Fordyce, 1973, 1976). Literature also exists, albeit to a lesser extent, on the facilitative effects of social support in chronic pain (Wallston et al. 1983; Cohen and Syme; 1985; Cohen and Wills, 1985). Our study was interested in how a therapy, which emphasises the adoption of a self-management approach, affects levels of satisfaction of social support. We employed the Significant Others Scale that allowed an analysis of changes in satisfaction of emotional and practical support from significant other.

Analyses supported the hypothesis only for the measure of practical support. Participants in the treatment group reported less discrepancy between actual and ideal levels of practical support. This was characterised by an increase in the actual support available and a reduction in the ideal support required. Although the changes in means are small it might be hypothesised that a therapy which endorses a self-management approach reduces levels of actual support needed by the patient in the first instance. This has the paradoxical effect of increasing the amount of available support as the significant other as problems in the relationship are reduced. No studies have examined this complex relationship.

Given that the majority of measures associated with the Significant Others Scale did not show any significant differences at post treatment between groups this issue becomes an important point for speculative discussion.

A simple explanation may relate to the salience of the particular aspect of social support under scrutiny. Intuitively, it could be advocated that practical levels of social support are far more salient than changes in emotional levels of support (Flor, Turk & Scholz, 1987). Thus, the greater salience of changes in practical support increases the likelihood of their detection at an early stage. On the other hand, changes in emotional levels of support, being less salient, will tend to be less detectable at an early stage and may evolve more slowly over time. This leads to the argument that if emotional levels of support are tested over a long enough period of time group differences may become apparent. In essence, the lack of significant differences may be the result of the short interim period between testing at pre and post treatment.

Measurement issues may also account for lack of differences between groups. Social support is not a unitary concept and at present no measure exists which encompasses all aspects of the term. The Significant Others Scale measures the functionality of relationships in practical and emotional terms. It is also possible to examine social support from a structural perspective (Cohen & Ashby Wills, 1985). Structural measures seek to describe the existence of relationships. The lack of group differences may be attributable to the measure that we employed. Further, the greatest changes may not exist at a functional level but at the structural level at least in the early stages. The functional nature of the assessment tool used in this study means that such changes could not be detected.

Another important issue regarding measurement relates to the specificity or globality of the measure employed (Cohen & Ashby Wills, 1985). Specific measures assess a specific aspect of social support while global measures take a number of different aspects of social support and combine them. The Significant Others Scale is a moderately specific functional questionnaire. Again the lack of group differences may be attributable to the measure that we employed. We assessed specific aspects of social support where in fact gross changes may have occurred on a global level. The specific nature of the assessment tool used in this study once again meant that such changes could not be detected.

Finally, as with all self-report measures there are a number of problems with responder bias due to social desirability or inaccurate recall over time (Jensen & Karoly, 1991). It is therefore possible that while differences did exist the way in which the participants responded diluted such differences.

4.3 Methodological Problems

4.3.1 Sample Size

The numbers of treatment and control participants needed to achieve power were insufficient in this study. The effect of this is that some significant differences may have been lost. The small sample size was due to the high attrition rates observed. Attrition rate was based on the number of treatment participants recruited into the study who either did not turn up for the initial assessment or attended and did not complete therapy. A total of 44 participants were assigned to the treatment group this would have been sufficient to detect a small to moderate effect size, however, only 12 completed therapy. There are a number of possible reasons for the high attrition rate.

Firstly, individuals with chronic tension type headache are often involved in psychosocial difficulties and are experiencing substantial distress. As with somatic patients, however, they tend to use bodily symptoms to communicate, possibly because they have difficulties in expressing their feelings in words. Such patients prefer general medical services to mental health services (Simon, 1992) and use hospital services excessively (Fink, 1992). This difficulty in verbally expressing emotions makes talking therapies a threatening concept and will engender anxiety responses and consequent avoidance.

Secondly, many of these patients are most likely in the early stages of change in the transtheroretical model and would be classed as precontemplators or contemplators. If this is the case then clinical psychology has a particular role to play in terms of helping these individuals reach a stage at which they are thinking about change or beginning to take action. In addition, preliminary work may not need to focus on the headaches directly but on developing beliefs of self-efficacy in preparation for change.

Thirdly, it is possible that a high number of individuals may be deriving some secondary gain from maintaining illness behaviors. Reasons for this may be varied and could include ongoing compensation claims, benefit claims, avoidance of stressful situations such as work.

Finally, the expectations of individuals who are suffering from chronic pain and receiving a referral to psychology may be the most significant reason for attrition. An obvious question in this study relates to why the attrition rate was so much higher compared to other studies that involve psychological intervention. One possible answer is the nature of the problem being addressed. Chronic pain individuals present with an overtly physical complaint; unlike the majority of primary care cases. As such, chronic pain patients may view their problem as purely physical and interpret referral to psychology as symbolic of an invalidation of the physical nature of their complaint. As a defence to this and as a validation of their condition they seek to preemptively reject a psychological approach. In essence, this rejection is based on a misunderstanding of the reason for referral as well as the need of the patient to validate their present circumstances.

An overarching concern due to the high attrition rate is that the individuals that remained in the study were not representative of chronic tension type headache sufferers and that they responded to treatment for very specific reasons that would not be found in the population as a whole.

4.3.2 Single Therapist Design

This study employed a single therapist design, which is a potential methodological flaw. As this was a therapeutic intervention, this type of methodology makes it difficult to establish the effect of the treatment itself over the effect of the therapist. There is no method for separating out these effects in this study and it is therefore a requirement of further research to employ a multi-therapist design. In practical terms, however, this treatment is usually carried out by a single therapist attached to the headache clinic.

4.3.3 Follow Up Data

Due to time constraints this study did not include any follow up data. Consequently, the present study cannot determine the effectiveness of the therapy over a longer time period. There is no obvious reason why the treatment effects would not be maintained but this cannot be concluded without such data. In addition, there were some measures that did not show a significant change during the treatment period but showed evidence of the beginnings of change. Follow up data would be useful in terms of tracking these changes.

4.4 Future Research

4.4.1 Predicting Treatment Success

Although the present study was useful in identifying changes in general measures that are associated with treatment success, it was not possible to look at the general measures in terms of their ability to predict treatment success. In doing so, treatment might be targeted more effectively and individuals identified that are likely to benefit.

4.4.2 Developing Preventative Therapies in Chronic Pain

While this study adds to existing literature on the effectiveness of cognitive-behavioural programs (Compas et al. 1998; Morley et al. 1999), it was noted that the timing of therapy was a strong determinant of treatment success. Recent studies are now looking the specific effects of psychological preventive interventions.

Some early, secondary preventive efforts have shown promise. For example, Waddell, Newton. (1997) reviewed 10 trials of early interventions for acute back pain mainly in primary care settings and found that programs that encouraged maintaining daily activities produced better results than various control conditions. One reason for this success may be that these programs dealt with the fear and anxiety often associated with acute pain and believed to generate `fear-avoidance' behaviours that may produce disability (Vlaeyen and Linton, 2000).

More recently, Von Korff et al. (1998) have found that a lay-led, cognitive-behavioural program for patients with acute back pain significantly reduced worry and disability at follow-up relative to a treatment as usual control. Therefore, there is reason to believe that early, preventive interventions might be viable.

However, there has been some debate concerning the time point for preventive interventions and this reflects the relative lack of investigation in `non-patient' populations. For example, Frank et al. (1996a) argue that, statistically speaking, interventions for back pain prior to about 8 weeks sick leave have little value since the natural recovery rate is high. Indeed, Sinclair et al. (1997) found that an early intervention program based on exercise, mobilization and education administered throughout Ontario produced little benefit in relation to `usual' care.

However, from a psychological point of view, the development of fear, anxiety and other processes that may generate disability (Turk; Vlaeyen and Waddell), is probably activated quite early on. Consequently, very early, psychologically-oriented prevention might be valuable. Nevertheless, there are relatively few attempts at prevention in non-patient populations, and most attempts have not been psychologically orientated. At present no studies have considered preventative therapy for headache.

4.4.3 Comparison with Other Therapies

The treatment results reported in this study need to be compared with other treatment delivery formats such as group treatment, interpersonal therapy, limited contact treatment in medical setting, telephone based treatments and internet based treatments.

In addition, the results of this study need to be both replicated, with the removal of the methodological flaws, and compared with studies using other treatment modalities such as bio-feedback, massage, hypnosis and relaxation alone.

4.5. Conclusion

The aim of this research was to examine the effectiveness of Cognitive Behavioural Therapy on patients with Chronic Tension Type Headache referred from an acute medical setting. This study also aimed to examine the impact of therapy on other areas known to be implicated in the maintenance of chronic pain conditions. Results have shown that Cognitive Behavioural Therapy is useful in reducing a number of aspects of chronic tension type headache. Additional changes in more general psychological measures over time suggest that Cognitive Behavioural Therapy also successfully addresses issues that may mediate the experience of chronic pain.

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Appendix I

Headache Measures

Headache and Medication Diary (Week One)

Day	No. of headaches +/time	Average Pain Severity (0-10)	Anxiety Levels (0-10)	Coping Strategy. How effective? (0-10)	Medication used?	Dosage	No. of tablets taken	Comments
Monday								
Tuesday								
Wednesday								
Thursday								
Friday								
Saturday								
Sunday								

Appendix II

General Measures

Appendix 3

PAIN STAGES OF CHANGE QUESTIONNAIRE

This questionnaire is to help us to better understand the way you view your pain problem. Each statement describes how you *may* feel about this particular problem. Please indicate the extent to which you tend to agree or disagree with each statement. In each example, please make your choice based on *how you feel right now*, not how you have felt in the past or how you would like to feel.

Circle the response that best describes how much you agree or disagree with each statement

1 = Strongly disagree; 2 = Disagree; 3 = Undecided or unsure; 4 = Agree; 5 = Strongly agree

1. I have been thinking that the way I cope with my pain could improve	1	2	3	4	5
2. I am developing new ways to cope with my pain	1	2	3	4	5
3. I have learned some good ways to keep my pain problem from interfering with my life	1	2	3	4	5
4. When my pain flares up, I find myself automatically using coping strategies that have worked in the past, such as relaxation exercise or mental distraction technique	1	2	3	4	5
5. I am using some strategies that help me better deal with my pain problem on a day-to-day basis	1	2	3	4	5
6. I have started to come up with strategies to help myself control my pain	1	2	3	4	5
7. I have recently realised that there is no medical cure for my pain condition, so I want to learn some ways to cope with it	1	2	3	4	5

8. Even if my pain doesn't go away, I am ready to start changing how I deal with it	1	2	3	4	5
9. I realise now that it's time for me to come up with a better plan to cope with my pain problem	1	2	3	4	5
10. I use what I have learned to help keep my pain under control	1	2	3	4	5
11. I have tried everything that people have recommended to manage my pain and nothing helps	1	2	3	4	5
12. My pain is a medical problem and I should be dealing with physicians about it	1	2	3	4	5
13. I am currently using some suggestions people have about how to live with my pain problem	1	2	3	4	5
14. I am beginning to wonder if I need to get some help to develop skills for dealing with my pain	1	2	3	4	5
15. I have recently figured out that it's up to me to deal better with my pain	1	2	3	4	5
16. I have recently figured out that it's up to me to learn to live with my pain, but I don't see why I should have to	1	2	3	4	5
17. I have incorporated strategies for dealing with my pain into my everyday life	1	2	3	4	5
18. I have made a lot of progress in coping with my pain	1	2	3	4	5
19. I have recently come to the conclusion that it's time for me to change how I cope with pain	1	2	3	4	5
20. I'm getting help learning some strategies for coping better with my pain	1	2	3	4	5
21. I'm starting to wonder whether it's up to me to manage my pain rather than relying on physicians	1	2	3	4	5
22. I still think despite what doctors tell me, there must be some surgical procedure or medication that would get rid of my pain	1	2	3	4	5
23. I have been thinking that doctors can only help so much in managing my pain and that the rest is up to me	1	2	3	4	5
24. The best thing I can do is find a doctor who can figure out how to get rid of my pain once and for all	1	2	3	4	5
25. Why can't someone just do something to take away my pain?	1	2	3	4	5
26. I am learning to help myself control my pain without doctors	1	2	3	4	5
27. I am testing out some coping skills to manage my pain better	1	2	3	4	5
28. I have been wondering if there is something I could do to manage my pain better	1	2	3	4	5
29. All of this talk about how to cope better is a waste of my time	1	2	3	4	5
30. I am learning ways to control my pain other than with medications or surgery	1	2	3	4	5

Scoring of The Pain States of Change Questionnaire

PRECONTEMPLATION: Sum (11, 12, 16, 22, 24, 25, 29)/7
CONTEMPLATION: Sum (1, 7, 8, 9, 14, 15, 19, 21, 23, 28)/10
ACTION: Sum (2, 6, 20, 26, 27, 30)/6
MAINTENANCE: Sum (3, 4, 5, 10, 13, 17, 18)/7

To account for sporadic missing data, sums should be divided by the number of non-missing items. Any scale with more than 25% of its items missing should be considered missing.

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Below is a list of common symptoms of anxiety. Please carefully read each item in the list. Show how much you have been bothered by each symptom during the PAST WEEK, INCLUDING TODAY, by placing an X in a box in one of the columns next to each symptom.

	NOT AT ALL	MILDLY It did not bother me much	MODERATE It was very unpleasant but I could stand it	SEVERELY I could barely stand it
Numbness or tingling.	☐	☐	☐	☐
Feeling hot.	☐	☐	☐	☐
Wobbliness in legs.	☐	☐	☐	☐
Unable to relax.	☐	☐	☐	☐
Fear of the worst happening.	☐	☐	☐	☐
Dizzy or lightheaded.	☐	☐	☐	☐
Heart pounding or racing.	☐	☐	☐	☐
Unsteady.	☐	☐	☐	☐
Terrified.	☐	☐	☐	☐
	☐	☐	☐	☐
	☐	☐	☐	☐
Hands trembling.	☐	☐	☐	☐
Shaky.	☐	☐	☐	☐
Fear of losing control.	☐	☐	☐	☐
Difficulty breathing.	☐	☐	☐	☐
Fear of dying.	☐	☐	☐	☐
Scared.	☐	☐	☐	☐
Indigestion or discomfort in abdomen.	☐	☐	☐	☐
Faint.	☐	☐	☐	☐
Face flushed.	☐	☐	☐	☐
Sweating (not due to heat).	☐	☐	☐	☐

SIGNIFICANT OTHERS SCALE (B)

Name:..

Date:.. **Record Number:**..

Instructions

Please list below up to seven people who may be important in the individual's life. Typical relationships include partner, mother, father, child, sibling, close friends, plus keyworker. For each person please circle a number from 1 to 7 to show how well he or she provides the type of help that is listed.

The second part of each question asks you to rate how individuals would like things to be if they were exactly as they hoped for. As before, please put a circle around one number between 1 and 7 to show what the rating is.

Person 1 –	*Never*			*Sometimes*			*Always*
1 a) Can you trust, talk to frankly and share your feelings with this person?........	1	2	3	4	5	6	7
b) What rating would your ideal be?........	1	2	3	4	5	6	7
2 a) Can you lean on and turn to this person in times of difficulty?..	1	2	3	4	5	6	7
b) What rating would your ideal be?........	1	2	3	4	5	6	7
3 a) Does he/she give you practical help?........	1	2	3	4	5	6	7
b) What rating would your ideal be?........	1	2	3	4	5	6	7
4 a) Can you spend time with him/her socially?........	1	2	3	4	5	6	7
b) What rating would your ideal be?........	1	2	3	4	5	6	7

Person 2 –							
1 a) Can you trust, talk to frankly and share your feelings with this person?........	1	2	3	4	5	6	7
b) What rating would your ideal be?........	1	2	3	4	5	6	7
2 a) Can you lean on and turn to this person in times of difficulty?..	1	2	3	4	5	6	7
b) What rating would your ideal be?........	1	2	3	4	5	6	7
3 a) Does he/she give you practical help?........	1	2	3	4	5	6	7
b) What rating would your ideal be?........	1	2	3	4	5	6	7
4 a) Can you spend time with him/her socially?........	1	2	3	4	5	6	7
b) What rating would your ideal be?........	1	2	3	4	5	6	7

Person 3 –							
1 a) Can you trust, talk to frankly and share your feelings with this person?........	1	2	3	4	5	6	7
b) What rating would your ideal be?........	1	2	3	4	5	6	7
2 a) Can you lean on and turn to this person in times of difficulty?	1	2	3	4	5	6	7
b) What rating would your ideal be?........	1	2	3	4	5	6	7
3 a) Does he/she give you practical help?........	1	2	3	4	5	6	7
b) What rating would your ideal be?........	1	2	3	4	5	6	7
4 a) Can you spend time with him/her socially?........	1	2	3	4	5	6	7
b) What rating would your ideal be?........	1	2	3	4	5	6	7

PLEASE CIRCLE ONE NUMBER ONLY FOR EACH QUESTION

Person 4 – ....

		Never			Sometimes			Always
1	a) Can you trust, talk to frankly and share your feelings with this person?	1	2	3	4	5	6	7
	b) What rating would your ideal be?	1	2	3	4	5	6	7
2	a) Can you lean on and turn to this person in times of difficulty?	1	2	3	4	5	6	7
	b) What rating would your ideal be?	1	2	3	4	5	6	7
3	a) Does he/she give you practical help?	1	2	3	4	5	6	7
	b) What rating would your ideal be?	1	2	3	4	5	6	7
4	a) Can you spend time with him/her socially?	1	2	3	4	5	6	7
	b) What rating would your ideal be?	1	2	3	4	5	6	7

Person 5 – ....

1	a) Can you trust, talk to frankly and share your feelings with this person?	1	2	3	4	5	6	7
	b) What rating would your ideal be?	1	2	3	4	5	6	7
2	a) Can you lean on and turn to this person in times of difficulty?	1	2	3	4	5	6	7
	b) What rating would your ideal be?	1	2	3	4	5	6	7
3	a) Does he/she give you practical help?	1	2	3	4	5	6	7
	b) What rating would your ideal be?	1	2	3	4	5	6	7
4	a) Can you spend time with him/her socially?	1	2	3	4	5	6	7
	b) What rating would your ideal be?	1	2	3	4	5	6	7

Person 6 – ....

1	a) Can you trust, talk to frankly and share your feelings with this person?	1	2	3	4	5	6	7
	b) What rating would your ideal be?	1	2	3	4	5	6	7
2	a) Can you lean on and turn to this person in times of difficulty?	1	2	3	4	5	6	7
	b) What rating would your ideal be?	1	2	3	4	5	6	7
3	a) Does he/she give you practical help?	1	2	3	4	5	6	7
	b) What rating would your ideal be?	1	2	3	4	5	6	7
4	a) Can you spend time with him/her socially?	1	2	3	4	5	6	7
	b) What rating would your ideal be?	1	2	3	4	5	6	7

Person 7 – ....

1	a) Can you trust, talk to frankly and share your feelings with this person?	1	2	3	4	5	6	7
	b) What rating would your ideal be?	1	2	3	4	5	6	7
2	a) Can you lean on and turn to this person in times of difficulty?	1	2	3	4	5	6	7
	b) What rating would your ideal be?	1	2	3	4	5	6	7
3	a) Does he/she give you practical help?	1	2	3	4	5	6	7
	b) What rating would your ideal be?	1	2	3	4	5	6	7
4	a) Can you spend time with him/her socially?	1	2	3	4	5	6	7
	b) What rating would your ideal be?	1	2	3	4	5	6	7

PLEASE CIRCLE ONE NUMBER ONLY FOR EACH QUESTION

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11. Agitation

0 I am no more restless or wound up than usual.
1 I feel more restless or wound up than usual.
2 I am so restless or agitated that it's hard to stay still.
3 I am so restless or agitated that I have to keep moving or doing something.

12. Loss of Interest

0 I have not lost interest in other people or activities.
1 I am less interested in other people or things than before.
2 I have lost most of my interest in other people or things.
3 It's hard to get interested in anything.

13. Indecisiveness

0 I make decisions about as well as ever.
1 I find it more difficult to make decisions than usual.
2 I have much greater difficulty in making decisions than ~~I~~ used to.
3 I have trouble making any decisions.

14. Worthlessness

0 I do not feel I am worthless.
1 I don't consider myself as worthwhile and useful as I used to.
2 I feel more worthless as compared to other people.
3 I feel utterly worthless.

15. Loss of Energy

0 I have as much energy as ever.
1 I have less energy than I used to have.
2 I don't have enough energy to do very much.
3 I don't have enough energy to do anything.

16. Changes in Sleeping Pattern

0 I have not experienced any change in my sleeping pattern.
1a I sleep somewhat more than usual.
1b I sleep somewhat less than usual.
2a I sleep a lot more than usual.
2b I sleep a lot less than usual.
3a I sleep most of the day.
3b I wake up 1–2 hours early and can't get back to sleep.

17. Irritability

0 I am no more irritable than usual.
1 I am more irritable than usual.
2 I am much more irritable than usual.
3 I am irritable all the time.

18. Changes in Appetite

0 I have not experienced any change in my appetite.
1a My appetite is somewhat less than usual.
1b My appetite is somewhat greater than usual.
2a My appetite is much less than before.
2b My appetite is much greater than usual.
3a I have no appetite at all.
3b I crave food all the time.

19. Concentration Difficulty

0 I can concentrate as well as ever.
1 I can't concentrate as well as usual.
2 It's hard to keep my mind on anything for very long.
3 I find I can't concentrate on anything.

20. Tiredness or Fatigue

0 I am no more tired or fatigued than usual.
1 I get more tired or fatigued more easily than usual.
2 I am too tired or fatigued to do a lot of the things I used to do.
3 I am too tired or fatigued to do most of the things I used to do.

21. Loss of Interest in Sex

0 I have not noticed any recent change in my interest in sex.
1 I am less interested in sex than I used to be.
2 I am much less interested in sex now.
3 I have lost interest in sex completely.

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______ Subtotal Page 2

______ Subtotal Page 1

______ Total Score

BDI®-II

Date:

Name: ______________________ Marital Status: ________ Age: ______ Sex: ______

Occupation: ______________________ Education: ______________________

Instructions: This questionnaire consists of 21 groups of statements. Please read each group of statements carefully, and then pick out the **one statement** in each group that best describes the way you have been feeling during the **past two weeks, including today**. Circle the number beside the statement you have picked. If several statements in the group seem to apply equally well, circle the highest number for that group. Be sure that you do not choose more than one statement for any group, including Item 16 (Changes in Sleeping Pattern) or Item 18 (Changes in Appetite).

1. Sadness

0 I do not feel sad.
1 I feel sad much of the time.
2 I am sad all the time.
3 I am so sad or unhappy that I can't stand it.

2. Pessimism

0 I am not discouraged about my future.
1 I feel more discouraged about my future than I used to be.
2 I do not expect things to work out for me.
3 I feel my future is hopeless and will only get worse.

3. Past Failure

0 I do not feel like a failure.
1 I have failed more than I should have.
2 As I look back, I see a lot of failures.
3 I feel I am a total failure as a person.

4. Loss of Pleasure

0 I get as much pleasure as I ever did from the things I enjoy.
1 I don't enjoy things as much as I used to.
2 I get very little pleasure from the things I used to enjoy.
3 I can't get any pleasure from the things I used to enjoy.

5. Guilty Feelings

0 I don't feel particularly guilty.
1 I feel guilty over many things I have done or should have done.
2 I feel quite guilty most of the time.
3 I feel guilty all of the time.

6. Punishment Feelings

0 I don't feel I am being punished.
1 I feel I may be punished.
2 I expect to be punished.
3 I feel I am being punished.

7. Self-Dislike

0 I feel the same about myself as ever.
1 I have lost confidence in myself.
2 I am disappointed in myself.
3 I dislike myself.

8. Self-Criticalness

0 I don't criticize or blame myself more than usual.
1 I am more critical of myself than I used to be.
2 I criticize myself for all of my faults.
3 I blame myself for everything bad that happens.

9. Suicidal Thoughts or Wishes

0 I don't have any thoughts of killing myself.
1 I have thoughts of killing myself, but I would not carry them out.
2 I would like to kill myself.
3 I would kill myself if I had the chance.

10. Crying

0 I don't cry anymore than I used to.
1 I cry more than I used to.
2 I cry over every little thing.
3 I feel like crying, but I can't.

Subtotal Page 1

Continued on Back

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2 I have much greater difficulty in making decisions than I used to.

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1 I don't consider myself as worthwhile and useful as I used to.

2 I feel more worthless as compared to other people.

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2 I am too tired or fatigued to do a lot of the things I used to do.

3 I am too tired or fatigued to do most of the things I used to do.

21. Loss of Interest in Sex

0 I have not noticed any recent change in my interest in sex.

1 I am less interested in sex than I used to be.

2 I am much less interested in sex now.

3 I have lost interest in sex completely.

______ Subtotal Page 2

______ Subtotal Page 1

______ Total Score

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MULTIDIMENSIONAL HEALTH LOCUS OF CONTROL SCALE (FORM A)

Name: ....

Date: .... **Record Number:** ....

This is a questionnaire designed to determine the way in which different people view certain important health-related issues. Each item is a belief statement with which you may agree or disagree. Beside each statement is a scale which ranges from strongly disagree (1) to strongly agree (6). For each item we would like you to circle the number that represents the extent to which you disagree or agree with the statement. The more strongly you agree with a statement, then the higher will be the number you circle. The more strongly you disagree with a statement, then the lower will be the number you circle. Please make sure that you answer every item and that you circle **only one** number per item. This is a measure of your personal beliefs: obviously, there are no right or wrong answers.

Please answer these items carefully, but do not spend too much time on any one item. As much as you can, try to respond to each item independently. When making your choice, do not be influenced by your previous choices. It is important that you respond according to your actual beliefs and not according to how you feel you should believe or how you think we want you to believe.

	Strongly disagree	Moderately disagree	Slightly disagree	Slightly agree	Moderately agree	Strongly agree
1. If I get sick, it is my own behaviour which determines how soon I get well again.	1	2	3	4	5	6
2. No matter what I do, if I am going to get sick, I will get sick.	1	2	3	4	5	6
3. Having regular contact with my doctor is the best way for me to avoid illness.	1	2	3	4	5	6
4. Most things that affect my health happen to me by accident.	1	2	3	4	5	6
5. Whenever I don't feel well, I should consult a medically trained professional.	1	2	3	4	5	6
6. I am in control of my health.	1	2	3	4	5	6
7. My family has a lot to do with my becoming sick or staying healthy.	1	2	3	4	5	6
8. When I get sick, I am to blame.	1	2	3	4	5	6
9. Luck plays a big part in determining how soon I will recover from an illness.	1	2	3	4	5	6
10. Health professionals control my health.	1	2	3	4	5	6
11. My good health is largely a matter of good fortune.	1	2	3	4	5	6
12. The main thing which affects my health is what I myself do.	1	2	3	4	5	6
13. If I take care of myself, I can avoid illness.	1	2	3	4	5	6
14. When I recover from an illness, it's usually because other people (for example, doctors, nurses, family, friends) have been taking good care of me.	1	2	3	4	5	6
15. No matter what I do, I'm likely to get sick.	1	2	3	4	5	6
16. If it's meant to be, I will stay healthy.	1	2	3	4	5	6
17. If I take the right actions, I can stay healthy.	1	2	3	4	5	6
18. Regarding my health, I can only do what my doctor tells me to do.	1	2	3	4	5	6

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MULTIDIMENSIONAL HEALTH LOCUS OF CONTROL SCALE (FORM B)

Name:..

Date:.. **Record Number:**..

This is a questionnaire designed to determine the way in which different people view certain important health-related issues. Each item is a belief statement with which you may agree or disagree. Beside each statement is a scale which ranges from strongly disagree (1) to strongly agree (6). For each item we would like you to circle the number that represents the extent to which you disagree or agree with the statement. The more strongly you agree with a statement, then the higher will be the number you circle. The more strongly you disagree with a statement, then the lower will be the number you circle. Please make sure that you answer every item and that you circle **only one** number per item. This is a measure of your personal beliefs: obviously, there are no right or wrong answers.

Please answer these items carefully, but do not spend too much time on any one item. As much as you can, try to respond to each item independently. When making your choice, do not be influenced by your previous choices. It is important that you respond according to your actual beliefs and not according to how you feel you should believe or how you think we want you to believe.

	Strongly disagree	Moderately disagree	Slightly disagree	Slightly agree	Moderately agree	Strongly agree
1. If I become sick, I have the power to make myself well again.	1	2	3	4	5	6
2. Often I feel that no matter what I do, if I am going to get sick, I will get sick.	1	2	3	4	5	6
3. If I see an excellent doctor regularly, I am less likely to have health problems.	1	2	3	4	5	6
4. It seems that my health is greatly influenced by accidental happenings.	1	2	3	4	5	6
5. I can only maintain my health by consulting health professionals.	1	2	3	4	5	6
6. I am directly responsible for my health.	1	2	3	4	5	6
7. Other people play a big part in whether I stay healthy or become sick.	1	2	3	4	5	6
8. Whatever goes wrong with my health is my own fault.	1	2	3	4	5	6
9. When I am sick, I just have to let nature run its course.	1	2	3	4	5	6
10. Health professionals keep me healthy.	1	2	3	4	5	6
11. When I stay healthy, I'm just plain lucky.	1	2	3	4	5	6
12. My physical well-being depends on how well I take care of myself.	1	2	3	4	5	6
13. When I feel ill, know it is because I have not been taking care of myself properly.	1	2	3	4	5	6
14. The type of care I receive from other people is what is responsible for how well I recover from an illness.	1	2	3	4	5	6
15. Even when I take care of myself, it's easy to get sick.	1	2	3	4	5	6
16. When I become ill, it's a matter of fate.	1	2	3	4	5	6
17. I can pretty much stay healthy by taking good care of myself.	1	2	3	4	5	6
18. Following doctor's orders to the letter is the best way for me to stay healthy.	1	2	3	4	5	6

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