Headache Classification and the Distinction Between Migraine and Muscle Contraction Headache

Carmen Vinci

Master of Science

Saint Mary's University

ISBN 0-315-77045-7

Table of Contents

List of Tables

Acknowledgements

I would like to thank those individuals who helped me with various aspects of my research. I would like to express special gratitude to Dr. Doug Cane without whose expertise, guidance, patience, and good humour, I would not have been able to complete this research project. I would also like to voice my appreciation to the Saint Mary's University Psychology Department for its support of my endeavours, and especially to Dr. Irmingaard Lenzer and Dr. Phil Street for serving on my thesis committee. I also wish to acknowledge my gratitude to the Victoria General Hospital for allowing me to conduct my research in the Pain Management Unit. Lastly, I would like to express my thanks to my husband Tom, for his steadfast support and encouragement throughout even the most difficult of times.

Headache Classification and the Distinction between Migraine and Muscle Contraction Headache

Carmen Vinci

Submitted in partial fulfillment of the
requirements for the degree of
Master of Science
at Saint Mary's University
Halifax, Nova Scotia

Approval: _____
Thesis Advisor

Approval: _____
Committee Member

Approval: _____
Committee Member

Date: October 1, 1992

Abstract
Headache Classification and the Distinction
Between Migraine and Muscle Contraction Headache

Carmen Vinci

September 25, 1992

The present study examined the ability of certain quantitative and qualitative measures of headache to distinguish between muscle contraction and migraine headache. Data were obtained from pain questionnaires administered to clients attending a hospital based Pain Management Clinic. Thirty-four complete files were available for analysis. Of these, 28 were completed by females; 6 by males. The average age of clients was 38 years. A cluster analysis performed on this sample suggested a two cluster solution corresponding to the traditional muscle contraction/migraine classification. The following variables were found to significantly distinguish between clusters: the number and type of adjectives used by subjects to describe headache; scores on the pain rating indices of the McGill Pain Questionnaire; the identification of physical factors as contributors to headache; the average intensity of headache; and a measure of disability due to headache. It was concluded that this study provided partial support for the traditional nomenclature of headache disorder and that future studies should replicate these results with a larger sample.

In 1962 the National Institute of Neurological Diseases and Blindness in the United States set up an hoc committee to derive a nomenclature for headache disorders (Ad Hoc Committee on the Classification of Headache). This committee divided headache problems into several distinct types based upon a combination of etiology and symptomatology. They identified 15 different categories of headache disorders. Of these, Blanchard and Andrasik note (1985) that the four comprising the majority of diagnosed headache disorder are:

(1) Vascular headache of migraine type: these include "classic" migraine, "common" migraine, and "cluster headache";

(2) Muscle-contraction headache;

(3) Combined headache (vascular and muscle contraction);

(4) Headache of delusional, conversion, or hypochondriacal states.

Diamond and Dalessio reviewed the Ad Hoc Committee's 15 categories and reduced them to three general categories of headache disorder (1978). These three categories were: (1) Traction and inflammatory headache (headaches resulting from organic disease of the brain, blood vessels, eyes, meninges, teeth, nose and sinuses); (2) Vascular headache, including classic and common migraine, as well as hemiplegic and opthalmoplegic migraine, rare headache syndromes which are variants of complicated migraine; cluster headache, toxic

vascular headache, hypertensive headache; and (3) Muscle contraction headache.

Recently the International Association for the Study of Pain (IASP) has distinguished "Primary Headache Syndromes" from "Craniofacial Pain of Musculoskeletal Origin" (1982). The primary headache syndrome designation includes classic migraine, common migraine, and cluster headache. Acute and chronic tension headache falls under the designation of Craniofacial pain of musculoskeletal origin. The IASP recommended that the term "mixed headache" be avoided. In its view this term is mostly used to refer to either migraine with inter-paroxysmal headache or to chronic tension headache, both which should be documented separately.

Whatever their differences, what is common to the above mentioned diagnostic schemes is the distinction they draw between migrainous headache and muscle contraction or tension headache. This distinction is based both upon proposed differences in the symptomatology of these two headache phenomena, as well as upon proposed etiological differences regarding these headache experiences. In the following sections evidence supporting the distinction between migraine and muscle contraction headache is reviewed.

Symptomatology of Migraine and of Muscle Contraction Headache

Muscle contraction headache (MCH) is typically reported as the experience of a dull, band-like, persistent pain which can last for days or months (Dalessio, 1978). A distinction has been made between episodic tension headache and chronic tension headache (Oleson, 1988).

Episodic tension headache occurs in discrete episodes which are separated from one another by headache-free periods. In most patients these headaches last less than 12 hours, although in some cases they may last for one or several days. Usually bilateral, the pain is usually described as dull and aching, which is assumed to imply pain intensity of mild to moderate levels.

Chronic tension headache shares the same characteristics with episodic tension headache except that more chronic tension headache sufferers experience pain over all or most regions of their cranium (holocrania) rather than localized pain. As well, these patients generally report pain as more severe and experience more associated symptoms such as photophobia, irritability, anorexia or nausea. These symptoms are not as "marked" as they are in migraine.

Headache is perhaps the cardinal symptom of the "symptom complex" of migraine attacks (Dalessio, 1978). That is, migraine represents a syndrome or process in which headache is only one symptom. The other symptoms composing the migraine

syndrome are largely prodromal or premonitory symptoms (Lance, 1982). That is, they indicate the onset of migraine headache. Some prodromal migraine symptoms are neurological in nature, such as the experience of an aura. The aura is usually visual in nature and is characteristically a scintillating scotoma (Friedman, 1982). A scotoma is an area of depressed vision within the visual field which is surrounded by an area of normal vision or of less depressed vision. The scintillating scotoma of migraine is a bright and flickering zigzag line stemming from the centre of the visual field and moving out toward the periphery (Edmeads, 1982). Auras and other focal neurological symptoms may precede the headache phase of a migraine attack by ten to sixty minutes.

Not all prodromal symptoms are as sharply defined as are auras or other focal neurological symptoms. For example, up to 24 hours prior to the headache phase of a migraine attack, some migraine patients report the experience of mood changes, drowsiness, or alterations in appetite or thirst (Lance, 1982).

Classic migraine is often distinguished from common migraine on the basis of premonitory or prodromal symptomatology. In classic migraine the prodromes are sharply defined, contralateral, and neurological (usually visual) in nature. In common migraine the prodromes are not sharply defined and may precede the attack by several hours or days.

Common migraine headache may last longer than classic migraine headache and can occur bilaterally.

During the headache phase the migraineur, whether suffering from classic or common migraine, is often nauseated and may vomit; as well, she will usually try to avoid sensory stimuli of all types, by for example, lying down in a quiet, dark room (Dalessio, 1978).

Etiology of Muscle Contraction Headache

It has been hypothesized that possible extracranial sources of head pain include the skin of the scalp and its blood supply, as well as the neck muscles. Upper cervical nerve roots which supply the occipital region and the upper connect with the spinal tract and the nucleus of the trigeminal nerve. Pain from structures above the tentorium is referred to the anterior two-thirds of the head by way of the trigeminal nerve. This may permit referral of pain from the upper neck to the head and vice versa (Friedman, 1982).

Muscle contraction headache has traditionally been thought to arise from sustained contraction of head and neck muscles (Wolff, 1963). Relevant cranial arteries measured during MCH show marked diminution in amplitude. Therefore, vasoconstriction with diminished blood flow and muscle ischemia is thought to occur as a consequence, giving rise to head pain (Friedman, 1982).

Tunis and Wolff (1954) studied the temporalis muscle and the frontal branch of the superficial temporal artery, which supplies the temporalis muscle region, in MCH sufferers as well as in nonheadache subjects. Muscle action potentials were recorded from the temporalis muscle region and pulse wave tracings were recorded from the temporal artery. Ten MCH sufferers recorded during headache and when headache-free were compared with ten nonheadache subjects. It was found that relative to nonheadache subjects, headache subjects during headache-free intervals manifested arterial vasoconstriction. Moreover, during right temporal MCH these same subjects manifested even greater vasoconstriction. Short-lived temporal artery constriction alone did not result in headache, nore did brief contraction of the temporalis muscle alone. One conclusion drawn from this study was that the co-occurrence of these two factors (artery constriction and muscle contraction) is required to produce the experience of MCH.

Evidence regarding the status of the temporal artery both during and between muscle contraction headache indicates that for MCH sufferers, it is constricted both between and during headache. It appears that MCH sufferers are relatively vasoconstricted between headaches and severely constricted during headache. Data from drug research appears to be consistent with these findings. Vasoconstrictive drugs either are ineffective in treating MCH, or exacerbate this type of headache (Cohen, 1978).

Etiology of Migraine Headache

It is generally thought that migraine pain is associated with cranial and cerebral vasoconstriction during the prodromal phase and with cranial and cerebral dilatation during the headache phase (Bakal, 1982). Specifically, the internal and external carotid and scalp arteries are constricted during the prodromal phase of migraine attack. The visual phenomena which can occur in the prodromal phase are caused by constriction of the posterior cerebral artery which causes ischemia in the occipital lobes. Following the prodromal phase, the external cerebral arteries dilate and hemicrania occurs. External carotid dilation by itself is not responsible for pain. Migraine pain results from a sterile inflammation which surrounds the dilated artery (Dalessio, 1972).

The pulse waves of the temporal arteries of ten migraine patients were recorded during headache and at the midpoint of headache-free intervals. These recordings were compared with those obtained from headache-free control subjects. Results indicated that during headache-free intervals migraineurs were more vasodilated than were controls. Migraineurs experienced a further increase in dilation during headache (Tunis & Wolff, 1953).

A classic study by Graham and Wolff in 1938 recorded the magnitude of pulse amplitudes from the superficial temporal and occipital arteries in migraine subjects during headache.

These subjects were given ergotamine tartrate, a potent vasoconstrictor. Decreases in the magnitudes of pulse amplitudes ranged from 84% to 16% with corresponding reports of decline in the subjective intensity of headache.

Dalessio (1978) has noted that subjects with severe and frequent migraines are often observed to manifest temporal artery prominence on one or both sides of the head, even when they are free from headache. In general it appears that migraine patients have relatively dilated temporal arteries between headaches which dilate even more during headache. Data from drug studies also appear to corroborate these findings. The typical drug therapy for migraine headache consists of the administration of some type of vasoconstrictive agent. The patient is warned to avoid vasodilatory agents such as alcohol which will exacerbate their headache (Cohen, 1978).

There is evidence that 36 to 72 hours prior to onset of migraine headache, the temporal artery becomes unstable and progresses through a sequence of vasodilation followed by vasoconstriction about six hours before pain begins. Headache pain results when the temporal artery again undergoes vasodilation (Tunis & Wolff, 1953).

In summary, some studies suggest that relative to nonheadache subjects, MCH subjects manifest a propensity toward vasoconstriction during headache-free intervals. During headache the vasoconstriction is even more severe. Other

studies suggest that migraine subjects manifest a propensity toward vasodilation during headache-free intervals relative to nonheadache control subjects. During headache the vasodilation increases. Thus, Cohen asserts that an important difference between migraine and MCH can be found in the status of the temporal artery (constricted or dilated) during headache and in headache-free intervals (1978).

Dietary triggers appear to be important in the etiology of migraine pain. Phenylethylamine, present in chocolate and some cheeses, seems to trigger migraine in susceptible individuals (Sandler, Youdim, & Hanington, 1974). Tyramine, found in chocolate, cheese, sherry, and red wine, has also been identified as a migraine trigger (Caviness & O'Brien, 1980; Kohlenberg, 1982). Aspartame has just recently been added to the list of foods which migraineurs should avoid (Koehler & Glaros, 1988). There is little or no evidence that dietary factors play any role in the etiology of MCH.

Lastly, various physical systems have been hypothesized to play a role in the genesis of migraine headache, including the autonomic nervous system, the vascular system, and the central nervous system.

The observed instability of the temporal artery prior to migraine headache has led some researchers to hypothesize that migraineurs are characterized by a generally unstable autonomic nervous system (Selby & Lance, 1960). A number of studies have provided support for this hypothesis (Dalessio,

1975; Gotoh, Komatsumotot, Nobuo, & Gomi, 1984; Havanka-Kannianinen, Tolonen, & Myllyla, 1986; Lance, 1981; Rose & Capildeo, 1983; Rubin, Graham, Pasker, & Calhoun, 1985). Other studies have hypothesized that migraine essentially represents a blood disorder (Couch & Hassanein, 1977; Deshmuck & Meyer, 1977; Gawel, Burkitt & Rose, 1979; Hanington, Jones, Amess & Wachowicz, 1981; Hilton & Cumings, 1972; Kalendovsky & Austin, 1975; Muck-Seler, Deanovic & Dupelj, 1979). Still other studies hypothesize that migraine is primarily a neurogenic disorder which produces the vascular changes characteristic of this headache syndrome (Edmeads, 1982).

Other Factors Differentiating Migraine and MCH

In addition to potential differences in etiology and symptomatology, other factors such as differences in responses to medications and other substances, differences in responses to non-pharmacological treatments, and differences in the subjective experience of headache, support the distinction between migraine and MCH.

There is evidence that both migraine and MCH respond differently to certain medications. Typically, therapy for migraine headache involves the use of some form of a vasoconstrictive agent. If the patient does not obtain relief or cannot tolerate the medication, an agent that increases the threshold of pain is prescribed. The patient is told to avoid

alcohol and other vasodilatory agents. In contrast MCH is typically treated with analgesic medication. Furthermore, vasoconstrictors appear to exacerbate MCH while alcohol and other vasodilators, such as amyl nitrite, tend to provide temporary relief for MCH sufferers (Cohen, 1978).

Ergot compounds and calcium channel blockers have been shown to be more effective in alleviating migraine than muscle contraction headache (Featherstone, 1985). The effects of three calcium channel blockers, Nimodipine, Verapamil, and Nifedipine, on classic migraine, common migraine, cluster, and combined headache was investigated by Meyer & Hardenberg (1983). Calcium channel entry blockers were found to provide effective prophylactic treatment for both the prodromal and the headache phase of classic migraine. They also appeared to effectively control common migraine pain and cluster headache pain. However, they had little or no effect upon MCH pain in those subjects suffering from combined headache. Patients with combined headache reported relief of the vascular component of their headache pain but continued to experience MCH pain. Patients with MCH alone who were treated with Verapamil and/or Nifedepine reported no benefit. The authors concluded that head pain in muscle contraction headache is not vascular in nature.

A frequently employed non-pharmacological treatment for muscle contraction headache pain is biofeedback training of the muscles thought responsible for the pain. Wolff found

that the resting levels of frontalis EMG activity were significantly higher in subjects with frequent muscle contraction headache than in those with few or no headaches (Wolff, 1963). When MCH sufferers were treated with relaxation training, as well as with EMG biofeedback training, a decrease in the frequency of muscle contraction headache was found (Mullaney, 1973). Friedman (1982) found that myographic potentials recorded from the head and neck muscles of muscle contraction headache sufferers during headache show vigorous contractions. EMG biofeedback training has been found to be effective for individuals with pure muscle contraction headache symptoms but not individuals with combined headache or MCH with vascular concomitants (Philips, 1977). Thus, MCH and migraine headache appear to respond differently to biofeedback training.

The clinical utility of distinguishing between muscle contraction and migraine headache has also been cited as support for the traditional classification of headaches (Blanchard & Andrasik, 1985). Different rates of success with different treatments for different headache diagnostic groups have been found. As well, different variables have been found to predict successful outcome for the two headache groups. Stronger predictions can be made when patients are differentiated by type of headache than when they are combined into one group.

There is evidence that migraineurs describe their headache experience differently than do MCH sufferers. The responses of 100 patients seeking treatment for headache on the McGill-Melzack Pain Questionnaire (MMPQ, 1975) were analyzed and it was found that migraine headache patients endorsed significantly more affective words than did MCH sufferers. Migraine sufferers also reported more intense pain when recalling headache "at its worst" than MCH patients. It was also found that migraineurs used multiple vascular pain descriptors such as "pounding" and "shooting", but few muscular descriptors. In fact, this study found that migraineurs describe their headaches in traditionally vascular terms, while tension headache patients describe their headache in traditionally muscular terms (Allen & Weinmann,1982).

In 1988 the International Headache Society (IHS) devised a classification system for headache designed to create operational diagnostic criteria which would improve the reproducibility and validity of headache diagnosis. A recent study diagnosed the headache type of 84 patients using the Ad Hoc Committee's diagnostic criteria. These classifications were then compared to those derived using IHS criteria. One criterion proposed by the IHS scheme is the effect which routine physical activity has upon headache pain. Aggravation of pain by activity is thought to be indicative of migraine headache, and lack of aggravation is taken to denote the experience of MCH. This study found that the exacerbation of

headache pain by routine physical activity was found to be the best criterion used to distinguish between migraine and tension-type headache. The authors state that this criterion was better at distinguishing between these two headache events than unilaterality or pulsating pain quality. It was argued that this difference between migraineurs and MCH patients suggests that these headache problems may not have the same pathophysiology (Iversen, Langemark, Anderson, Hansen, & Oleson, 1990).

The Continuum Model of Headache Classification

An alternative view to the traditional model of headache classification hypothesizes that differences among headache sufferers are not qualitative, but are quantitative occurring along a continuum of pain intensity and pain frequency. This implies that there are no distinct categories of headache, such as muscle contraction headache and migraine headache. Instead there is only chronic headache, which varies in terms of both frequency and intensity (Blanchard & Andrasik, 1985).

Evidence supporting a continuum model of headache classification has been provided by a number of researchers. A brief review of this evidence follows.

In 1972 Ziegler, Hassanein, and Hassanein, administered a questionnaire to 289 patients suffering from recurrent headache. The questionnaire elicited information about the age of onset of headache episodes, duration, phenomena

associated with headache, recurrence patterns, family history, and other variables. A factor analysis of these data revealed no single factor which contained the variables assumed to be characteristic of migraine, but not of MCH, namely, unilateral pain, nausea, scotomata, and scalp tenderness.

Heredity is also assumed to play a role in migraine pain, but not in MCH, yet Ziegler et al., found that a positive family history for headache was characteristic of the entire headache population, including MCH sufferers.

Waters (1974) examined the prevalence of different symptoms associated with migraine in the general population. Results indicated that while classic migraine was thought to be experienced by a small percentage of headache sufferers, almost all people who had experienced headache were also familiar with at least one of the classic migraine symptoms. A positive relationship was also found between reported severity of symptoms and the prevalence of the three migraine symptoms. For example, of the subjects who rated their headaches as mild, 30% had unilateral pain; 5% had a pre-headache warning; and 20% experienced nausea. Of those rating their headaches as "unbearable", 55% had unilateral pain; 65% had warning signs; and 75% had nausea. Based on these findings, it was proposed that headache be thought of as extending across a continuum of severity ranging from mild headaches with no migrainous features, to severe headaches characterized by numerous migrainous features (Bakal, 1982).

A number of studies have evaluated the hypothesis that migraine headache can be differentiated from MCH on the basis of the quality of their symptomatology. Four representative studies will be briefly reviewed. The prevalence of musculoskeletal, vascular, and autonomic symptoms in headache sufferers in the general population was investigated by Kaganov, Bakal and Dunn (1981). Questionnaires with a stamped return envelope were mailed to 1551 households in the city of Calgary using a random sampling procedure. Subjects were asked to indicate how often they considered their headaches to be a problem. This item was used to distinguish headache sufferers along a continuum of perceived severity from those who never or hardly ever considered their headaches a problem to those who often or always considered their headaches a problem. Results indicated that persons who never or seldom considered their headaches to be a problem experienced musculoskeletal symptoms more frequently than they experienced vascular symptoms while those who often or always considered their headaches to be problematic experienced vascular symptoms more frequently than musculoskeletal symptoms. More specifically, it was found that while vascular symptoms were more strongly correlated with severe headache than musculoskeletal symptoms, reports of both types of symptomatology were positively correlated with headache severity. The majority of respondents showed no tendency to

experience headache in terms of distinct muscle contraction or migraine symptom clusters.

Bakal and Kaganov (1979) examined the prevalence of musculoskeletal, vascular, and autonomic symptoms in a group of chronic headache patients and in a group of occasional headache sufferers. A 14-item questionnaire was administered to the subjects asking the extent to which their symptoms were perceived to be present across all headache attacks. Respondents were also asked to indicate to what extent they considered their headaches to be a problem. In general, the data indicated that the primary difference between the chronic and non-chronic headache sufferer was in terms of the frequency with which the symptoms occurred rather than in terms of the kind of symptoms present. In a replication of this study, Thompson, Haber, Figueroa, and Adams (1980) classified subjects into headache types. The results indicated that migraineurs and MCH sufferers reported similar symptoms.

Bakal and Kaganov (1977) asked MCH and migraine headache patients to indicate whether their headaches were typically throbbing or of a dull, aching nature. Forty percent of both types of patients reported the experience of throbbing headaches. Fifty-two per cent of patients from each headache group reported associated visual disturbances. The results of this study, then, revealed few differences in the ways in

which migraineurs and individuals with MCH described their symptoms.

These studies indicate that considerable overlap exists between migraine and MCH sufferers regarding the quality of their reported symptomatology. Therefore, they do not support the hypothesis that migraine headache can be differentiated from MCH based on differences in symptomatology.

The hypothesis that migraineurs can be differentiated from MCH patients on the basis of the location of pain during headache has been examined by Bakal and Kaganov (1977). Patients were provided with a self-observation form devised to record location and intensity of pain. This form contained two depictions of a person's head, one showing a profile from the left, and the other a profile from the right. The cranium and the face were marked into different regions, and each region was represented by a number. Patients were asked to use all location numbers necessary to describe the pain and to write them down in the appropriate cells on the form, showing the time of day and pain intensity.

Based on the traditional classification of headache, it was hypothesized that bilateral pain from the neck areas and back of the head along with bilateral pain from the forehead would be associated with MCH. Migraine headache was hypothesized to be associated with reports of unilateral pain in the eye region and unilateral pain from the forehead. As well, bilateral pain from the eyes and sides of the head were

included. Contrary to the results predicted by the traditional classification of headaches, an analysis of variance found no significant differences between these two groups of headache patients with respect to the frequency of involvement of particular locations, or the intensity of pain at each location.

Another hypothesized difference between the experience of migraine and MCH sufferers is that, contrary to the experience of migraineurs, MCH sufferers will exhibit excessive EMG activity in relevant muscle groups. However, Bakal and Kaganov (1977) found conversely that migraineurs had significantly higher frontalis EMG activity than did MCH patients both during an attack and during headache-free intervals. These results led the researchers to question the assumption that migraine headaches are essentially vascular in nature. It was suggested that sustained muscle activity may be a predisposing factor for both groups of headache patients, and that severe headache may reflect a patient's inability to cope with less severe headaches.

It was also found that nausea and vomiting were more prevalent in migraineurs than in MCH sufferers. However, it was argued that this does not necessarily mean that migraine is essentially distinct from muscle contraction headache. Instead, it may simply reflect the fact that migraine is a more severe form of headache, and that the more severe the headache is, the greater the number of physiological systems

that will be called into play, such as the gastrointestinal system.

Generally, migraine headache is thought of as occurring suddenly and without provocation, while muscle contraction headache is viewed as developing more slowly, and usually in response to stress. On this basis, it would be predicted that, in general, sufferers of MCH, in response to daily life stress, would experience their headaches later in the day. Bakal, Demjen and Kaganov (1981) monitored the headache activity of patients suffering from migraine, muscle contraction, and mixed headache and found that the majority of headache attacks experienced by patients began upon wakening or within a few hours upon wakening. Therefore, the results of this study did not support the hypothesis that migraine and MCH could be differentiated on the basis of their pattern of onset.

In a review of EMG studies, Haynes, Cuevas and Gannon (1982) note that studies which have evaluated the relationship between reports of headache and EMG levels have reported both positive and negative correlation coefficients while other studies found no relationship. Philips (1978) concluded that tension headache sufferers cannot be differentiated from migraineurs with respect to muscular tension. Based on a review of the EMG literature, Takeshima and Takahashi (1988) cite EMG studies which show that EMG activity and muscle tenderness increases more in migraineurs than it does in MCH

sufferers. Regardless of headache type, headache patients manifested continuous electrical activity on neck and temporalis EMG measures resulting from sustained spasm. In contrast, control subjects did not exhibit this pattern (Pozniak-Patewicz, 1976).

The proposal that migraine is distinct from muscle contraction headache on etiological grounds has also been challenged. Evidence of platelet abnormalities has been found for MCH sufferers as well as for migraineurs. Anthony and Lance (1989) compared platelet serotonin concentrations in muscle contraction headache sufferers with those in migraineurs and controls. While a significant difference was found between both headache groups and controls, no significant differences were found between MCH sufferers and migraineurs during headache. Other studies have also found platelet abnormalities in MCH subjects (Rolf & Brune, 1981; Takeshima & Takahashi, 1987).

There is also evidence that both MCH sufferers and migraineurs experience autonomic nervous system disturbance. For example, the constriction and the dilatation of the pupil at rest and during stress has been hypothesized to be under the control of the autonomic nervous system. The study of the pupil under various conditions (pupillometry) has been established as a valid method of determining autonomic nervous system activity (Rubin, Graham, Pasker & Calhoun, 1985). Some pupillometry studies have found sympathetic hypofunction

(decreased function) in the iris of migraineurs, indicating an autonomic nervous system abnormality (Gotoh, Komatsumoto, Araki, & Gomi, 1984; Havanka-Kannianen, Tolonen & Myllyla, 1986; Rubin, Graham, Pasker, & Calhoun, 1985). However, sympathetic hypofunction in the iris of MCH patients has also been reported (Shimomura & Takahashi, 1986; Takeshima, Takao, & Takahashi, 1987).

Finally, some studies suggest that relaxation training and biofeedback techniques are equally effective in the treatment of both migraine and muscle contraction headache (Reich, 1989). Seven hundred and ninety three migraine and MCH patients were randomly assigned to one of four different treatment groups. These four treatment groups consisted of: relaxation training, biofeedback training, micro-electrical therapy, such as the use of a transcutaneous nerve stimulator (TENS), and multimodal therapy, which combined two of the other treatment groups. All treatment conditions were found to signficantly reduce the frequency and intensity of both types of headaches. These treatment gains were still evident at a three year follow-up of these patients.

Bakal and Kaganov (1977) provided frontalis EMG and deep muscle relaxation training to migraine and MCH subjects. They found that significant reductions occurred in headache frequency and severity for both headache groups. Bakal, Demjen, and Kaganov (1981) treated 45 chronic headache patients, including MCH, migraine, and combined headache

patients, with cognitive-behavioral techniques. They found a significant overall treatment effect for all diagnostic groups which was maintained at a six month follow-up evaluation. Cohen (1978) concluded that more and more evidence is accumulating in support of the hypothesis that the same forms of non-pharmacological treatments may be equally effective for migraine as they are for MCH.

The Present Study

The traditional classification of headache considers migraine and MCH to comprise two distinct headache disorders. The continuum model of headache asserts that there are no distinct categories of headache. Instead there is only chronic headache, which varies in terms of both frequency and intensity. Evidence has been cited in support of both models of headache classification. Whether the severity model of headache is to be preferred to the more traditional view of headache classification remains an interesting issue for debate. The present study examined two dimensions hypothesized to distinguish between migraine and MCH. These were: the nature and location of pain, and the frequency and intensity of headache. It was hypothesized that with respect to these variables, significant differences would be found between migraine and MCH, lending additional support to traditional headache nosology. Data regarding these variables were analyzed by a cluster analysis which is a classification

procedure that attempts to divide a set of heterogeneous objects into relatively homogeneous groups. The purpose of this study was to evaluate whether obtained clusters correspond to traditional headache classifications or were better represented by the continuum model of headache classification. It was hypothesized that two significant clusters corresponding to migraine and MCH subtypes would be obtained.

It was hypothesized that one cluster would contain cases wherein: (a) muscle tension descriptors were used to describe headache; (b) headache was reported to be influenced by psychological variables; (c) the circumstances under which headache first appeared were adverse; (d) holocrania was reported or headache was reported to occur bilaterally; (e) headache intensity levels were reported as mild and/or moderately severe, and (f) disability due to headache was low. It was hypothesized that the other cluster would contain cases wherein: (a) vascular descriptors were used to describe headache pain; (b) headache was reported to be influenced by physical factors; (c) the circumstances under which headache pain first appeared were reported as neutral relative to physical and/or psychological stress; (d) headache pain was reported to occur unilaterally; (e) headache pain intensity levels were reported as quite severe, and (f) disability due to headache was high.

Method

Subjects

Headache sufferers in current attendance or who attended the Pain Clinic of the Victoria General Hospital in Halifax, N.S., during the past five years served as the initial subject pool. Inclusion criteria for analysis in this study were: subjects diagnosed with either muscle contraction or migraine headache, as noted in their medical chart by the Pain Clinic physician.

Exclusion criteria for analysis in this study were: subjects diagnosed as suffering from headache associated with post-traumatic stress disorder, headache arising from myofascial conditions, or headache arising from intracranial neoplasm, central nervous system infection, or severe systemic illness such as congestive heart failure, and headache associated with arthritis or hypertension (Ziegler, Hassanein & Hassanein, 1972). If any of these conditions were noted on a patient's medical chart, the patient was excluded from the study.

Measures

Information regarding the headache experience of these past and current patients was obtained from two sources. The first was the initial assessment note provided by the Pain Clinic physician and attached to the patient's medical chart.

The second was the Pain Evaluation Questionnaire (PEQ) which is administered to all patients attending Pain Clinic.

The medical note generally included a diagnosis of either migraine or muscle contraction headache. Patients whose medical chart note contained a diagnosis of migraine or MCH were identified and their PEQs were selected for analysis.

The PEQ asks patients to provide demographic data; social information; personal habits regarding caffeine and nicotine use; a brief medical history; pain location and pain quality (assessed by having them complete the descriptor portion of the McGill Pain Questionnaire); two measures of pain intensity; variables affecting pain; medications consumed; past and present treatments; types of treatment; the effect of pain on daily activity, sleep, and work. Finally, it asks about other medical or health problems which patients may be experiencing. (The PEQ is presented in Appendix A).

Headache quality and headache intensity were evaluated using scores on the descriptor portion of the McGill Pain Questionnaire (MMPQ), using traditional scoring methods as well as a new scoring method of the descriptor portion of the MMPQ which may discriminate better between headache phenomena than does traditional scoring of MMPQ items (Charter & Nehemkis, 1983). The MMPQ was devised in order to obtain information on three hypothesized dimensions of the pain experience: the sensory, the affective, and the evaluative (Melzack, 1975; 1983). The MMPQ comprises 78 adjectives,

divided into 20 subclasses. Each adjective is rank-ordered in terms of intensity within each subclass (Melzack & Torgerson, 1971). The sensory dimension of pain is represented by the first ten subclasses (sensory category); the affective by the next five (affective category); and the evaluative by the sixteenth (evaluative category).

Three basic measures can be obtained from the MMPQ. These are: the Pain Rating Index or PRI; the Present Pain Intensity or PPI; and the number of words chosen, or NWC. The PRI is the summation of the rank values for each category. The PPI is an indicator of overall pain intensity at the time the questionnaire is administered. Finally, the NWC is derived by summing the number of subclasses that a patient chooses to describe his pain, for all pain categories. A problem of this scoring method is its failure to achieve a common metric for the three categories of pain description.

A study using MMPQ scores on a population of patients suffering from cancer found that conventional scoring of the MMPQ failed to confirm the hypothesis that the affective dimension would be prepotent in the descriptions of pain offered by cancer patients (Charter & Nehemkis, 1983). When a revised MMPQ scoring system was employed, the original hypothesis was supported. Charter and Nehemkis obtain four measures with their revised scoring procedure. These measures are: the Average Pain Intensity, or APT; the Average Pain Intensity-Word Classes Chosen, or APC; the Percentage of Words

Chosen, or PWC; and lastly, the difference between the APT and the APC (APT-APC).

The APT is derived by summing the scale intensity values for each subclass in each category and dividing this value by the number of subclasses in each category (ie., 13 for the sensory category, six for the affective). Charter and Nehemkis incorporate three of the MMPQ subclasses into the sensory category. A remaining miscellaneous subclass is included in the affective category.

The APC is computed by summing the scale intensity values for each subclass and dividing this value by the number of subclasses chosen from each category. The PWC, a measure of the complexity of pain, is derived by summing the number of words chosen per category and dividing this sum by the number of subclasses in that category. The APT-APC difference score is a further measure of the complexity of pain.

Charter and Nehemkis state that the APC is a more sensitive measure than the APT or the traditional PRI score because it allows us to discriminate between patients who may indicate an overall low level of pain intensity as reflected in their PRI(R) or APT score, yet for whom the pain is very intense for the class in which they are experiencing the pain. They also note that the PWC specific to a category is a more useful measure than the traditional computation of NWC for it allows comparisons between different MMPQ categories. They

note as well that they have found the APT-APC difference score extremely useful as a measure of pain complexity.

In this study, APT, APC, PWC, and APT-APC scores were computed for each headache sufferer. As well, traditional MMPQ scores were computed.

The medical histories of patients were ascertained by asking them for the appoximate time at which they first experienced their pain problem. Also, the patient was asked to check off the circumstances under which the pain began: an accident at work or at home; an accident away from work; a time of personal or family stress; a time of stress or conflict at work; during or following an illness; or following surgery. The patient can also report either that "the pain 'just' began" or "cannot remember."

Pain location was determined by an examination of the location of markings patients make on a drawing of a male human body from the perspective of the front, and of the back. Patient markings on the left side of the head were coded with the number "1". Patient markings on the right side of the head were coded with the number "2". Bilateral markings were coded with the number "3".

Intensity of pain was partly assessed by the descriptor portion of the MMPQ and partly by patient response to a question concerning the degree of pain intensity relative to the patient's experience of pain at the moment, at its worst, and at its least. As well, patients were asked to rate the

intensity of their pain on a seven-point scale of intensity ranging from "no pain at all" to "excruciating" pain.

The severity of pain, in terms of its effects on daily activities, was measured by asking the patient the following: "Thinking back over the past month, how much did your pain interfere with the following aspects of your life? Circle the number that best describes how much your pain interfered with each of the following." Patients were then asked to report by how much (a) work outside the home, (b) house and yard work, (c) child care, (d) recreation or hobbies, (e) social activities, (f) close relationships, (g) sexual relationships, (h) eating, (i) sleeping, and (j) tolerance for frustration has been affected by their pain. They were asked to specify "Not at all, a little, a fair amount, or very much." The number of areas affected by headache was computed for each subject. The total score represented the total extent of disability suffered by the patient due to his experience of headache. Scores on this measure could range from 10 to 40.

Patients were also required to report the degree to which either psychological or physical factors affected their pain. The PEQ lists five factors which, for the purposes of this study, have been classified as "psychological". These were: "Feeling happy or good; Feeling tense, anxious, or stressed; Feeling calm or relaxed; Feeling sad or depressed;" and "Feeling angry or frustrated". The PEQ also lists 27 factors which have been classified as "physical". These variables

range from dietary factors such as alcohol and caffeine consumption to various activities such as walking and performing chores at home. Patients were asked to note for each factor whether it "usually increases pain (+2), sometimes increases pain (+1), has no effect on pain (0), sometimes decreases pain (-1), or usually decreases pain (-2)". For the purposes of this study, the number of physical factors which both usually increase and decrease pain was computed for each subject. The sum of these measures was used as a basis for determining the extent to which the subject's pain was influenced by physical factors. The number of psychological factors which both increase and decrease pain was determined for each subject. The sum of these measures was used as a basis for determining the extent to which the subject's pain was influenced by psychological factors.

Procedure

Patient files physically stored in the Pain Management Unit of the Victoria General Hospital and whose medical chart note contained a diagnosis of migraine or MCH headache were identified. If a reading of the medical chart note indicated that the subject met the requisite inclusion and exclusion criteria for participation in the study, the PEQ was selected for analysis.

Results

Data were obtained from 86 questionnaires administered to chronic pain patients attending the Pain Management Unit at the Victoria General Hospital. It was hypothesized that two significant clusters corresponding to migraine and MCH subtypes would be obtained from an analysis of these data. This hypothesis was evaluated by means of a cluster analysis, a classification procedure which attempts to divide a set of heterogeneous objects into relatively homogeneous groups. In cluster analysis it is assumed that group membership is unknown. Thus the data were analyzed without reference to whether it was obtained from the file of a migraine or an MCH sufferer.

Sample Characteristics

A total of 86 questionnaires were collected, of which 11 were discarded due to the fact that more than 50% of the questionnaire items were not completed. Of the remaining 75 questionnaires, 62 were completed by females and 13 by males. The average age of these clients was 38.6 years (S.D.= 8.97). Approximately half of the subjects (46%) in this sample had graduated from high school; 20% had completed Grade 10, and 25% had a Grade 8 or lower education. Due to the fact that the Pain Management Unit is a tertiary care facility, patients whose headaches are of a longstanding nature are typically seen there.

Cluster Analysis

Average linkage cluster analysis was the method chosen to analyze the data. This statistical procedure defines the distance between two clusters as the average of the distances between all pairs of cases in which one member of the pair is from each of the clusters. This method uses information about all pairs of distances, not only the nearest or the furthest. The agglomeration schedule produced by this procedure yields correlation coefficients which can be used as a measure of similarity between groups or clusters. This procedure yielded 34 cases for analysis; 55 cases were rejected due to missing values. The 34 cases available for analysis did not differ significantly from the original sample with regard to age or sex.

The agglomeration schedule for this analysis is presented in Table 1. An examination of this schedule revealed an 84% increase in the value of the correlation coefficients representing the last two stages of agglomeration. Hunter (1983) notes that there generally are no acceptable criteria for determining the number of clusters from a dendogram. She adds that typically, dendograms are examined for large changes in fusion levels. Citing Everitt (1980) on the matter, she comments that in many cases what counts as a "large" change is very subjective. For the purpose of this study, percentage increases in the values of correlation coefficients of 50%

Table 1

Cluster Analysis
Agglomeration Schedule
For N=34

Stage	Cluster 1	Cluster 2	Coefficient	Cluster 1	Cluster 2	Stage
1	20	29	84.000000	0	0	12
2	13	34	111.000000	0	0	14
3	16	33	119.000000	0	0	9
4	2	32	120.000000	0	0	13
5	7	24	124.000000	0	0	13
6	26	28	144.000000	0	0	17
7	21	25	160.000000	0	0	16
8	6	10	164.000000	0	0	14
9	9	16	181.500000	0	3	16
10	1	4	182.000000	0	0	28
11	12	14	189.000000	0	0	25
12	20	30	196.000000	1	0	24
13	2	7	203.000000	4	5	21
14	6	13	226.500000	8	2	19
15	3	15	231.000000	0	0	26
16	9	21	233.000000	9	7	19
17	26	31	237.000000	6	0	18
18	18	26	257.333344	0	17	25
19	6	9	261.850006	14	16	21
20	11	17	278.000000	0	0	23
21	2	6	288.611115	13	19	23
22	22	23	336.000000	0	0	31
23	2	11	365.307678	21	20	24
24	2	20	395.511108	23	12	28
25	12	18	484.250000	11	18	27
26	3	19	498.500000	15	0	27
27	3	12	539.833313	26	25	30
28	1	2	542.722229	10	24	29
29	1	5	659.700012	28	0	30
30	1	3	788.735474	29	27	31
31	1	22	1136.800049	30	22	32
32	1	8	1451.781250	31	0	33
33	1	27	2674.151611	32	0	0

or more were deemed to be sufficiently large to warrant a two or more cluster solution. Therefore, the results of this preliminary cluster analysis supported a two cluster solution. Based on this result, a quick cluster analysis, specifying the formation of two clusters, was performed on the sample. In this procedure cluster one contained 14 cases and cluster two contained 20 cases.

This thesis had hypothesized the formation of two clusters, distinguishable from each other in the following ways. Subjects in one cluster would predominantly describe their headache experience with muscular descriptors; subjects in the other cluster would predominantly describe their headaches with vascular descriptors. The descriptors chosen to represent the category of vascular descriptors were "sharp" and "throbbing"; the descriptor chosen to represent the muscular descriptors was "pressing". Headache was hypothesized to be influenced by psychological variables in one cluster and by physical variables in the other cluster. The circumstances under which headache first appeared were hypothesized to be adverse in one cluster but relatively neutral in the other cluster. Holocrania or bilateral headache would comprise the cases of headache in one cluster, while unilateral headache would comprise the cases of headache in the other cluster. Headache intensity levels were hypothesized as mild and/or moderately severe in one cluster, but quite severe in the other cluster.

Finally, disability due to headache was hypothesized to be low in one cluster whereas disability ratings would be high in the other cluster.

The quick cluster procedure employed in this study incorporates an analysis of variance. Though an analysis of variance carried out in this setting does not meet all of the standard conditions, it can provide preliminary information about differences that may exist between the two clusters.

An analysis of variance carried out between the two clusters found, at an .05 significance level, that a number of variables distinguished the two clusters. Relative to the other cluster, subjects in one cluster chose more sensory, affective, and miscellaneous words to describe their headache. The choice of affective words to describe headache represented the largest distance between final cluster centers. Subjects in one cluster evidenced a higher sensory pain rating index, a higher affective pain rating index, and a higher miscellaneous pain rating index than subjects in the other cluster. Relative to the other cluster, subjects in one cluster reported a higher average intensity of pain score. With regard to the number of reported physical factors increasing pain, as well as the sum of physical factors increasing pain, subjects in one cluster reported higher scores than subjects in the other cluster. Lastly, subjects in one cluster evidenced a higher

amount of disruption in their daily activities due to pain, as opposed to subjects in the other cluster. Therefore, the results of this analysis produced a two cluster solution with one cluster corresponding to the description of migraine headache and the other cluster matching the description of MCH. These results are summarized in Table 2.

TABLE 2

Means for Variables Which Significantly Differentiated The Obtained Clusters

Variables	Cluster 1	Cluster 2
Sensory Number of Words Chosen	8.4	6.0*
Affective Number of Words Chosen	4.1	2.9*
Miscellaneous Number of Words Chosen	3.9	3.2*
Pain Intensity	3.3	3.1
Pain Rating Index-Sensory	24.1	18.1*
Pain Rating Index-Affective	8.3	5.3*
Pain Rating Index-Miscellaneous	9.6	6.6*
Sensory APC Score	2.7	2.7
Affective APC Score	3.4	3.4
Evaluative APC Score	4.3	3.7
Average Intensity of Pain	6.2	4.9*
Worst Pain Score	4.8	4.9
Least Pain Score	2.3	1.9
Physical Factors Increasing Pain-Number	16.7	11.6*
Physical Factors Increasing Pain-Sum	27.7	14.9*
Psychological Factors Increasing Pain -Number	2.7	2.7
Psychological Factors Increasing Pain -Sum	4.5	3.9
Daily Activity Index	31.9	25.2*

Note: Differences significant at $p < .05$ are indicated with a *.

These results supported the traditional classification of headache disorders in that significant differences between clusters were found as hypothesized for the following variables: the identification of physical factors as contributors to the headache experience, the average intensity of headache, and a measure of disability due to headache. In addition, as already noted, six scores derived from the MMPQ were found to significantly distinguish between one cluster and the other.

Certain variables which were hypothesized to significantly distinguish between clusters were represented by nominal scores and therefore were not amenable to an analysis of variance. These variables were the two vascular and one muscular descriptors of pain; the circumstances under which the headache initially began; and the location of the headache. In order to determine whether these variables significantly distinguished between clusters, a Chi square analysis was conducted. The three headache descriptors were analyzed using 2x2 contingency tables. Since these tables contained one degree of freedom, Chi Square was computed using Yates' correction for continuity, which corrects the value of χ^2 to provide a better approximation to the exact multinomial probability (Hays, 1981). Yates' correction was not employed in the computation of Chi square for the remaining variables, circumstances of pain onset and location of pain.

As can be seen in Table 3, the independence of the two population distributions represented by cluster 1 and cluster 2 was not supported by the results. The value of the χ^2 obtained for any of these variables was not significant ($p > .05$). Thus, the choice of "throbbing" to describe headache did not differentiate one cluster from the other; the choice of "pressing" to describe headache did not differentiate the two clusters; the choice of "sharp" to describe headache did not differentiate the two clusters; the reported circumstances under which pain initially began did not differ significantly for the two clusters, and, lastly, location of pain in the head did not distinguish between the two clusters. Thus, the two clusters did not differ with respect to the frequency with which subjects used throbbing, pressing, and sharp to describe their headaches. In addition, the two clusters did not differ with respect to the circumstances under which the headache initially began, or with respect to the location of headache.

TABLE 3

Chi-Square Analysis of the Independence of Clusters One and Two

Variables	Df	N	Chi-square value
Throbbing	1	34	1.176
Sharp	1	34	.035
Pressing	1	34	1.12
Circumstances of Onset	7	34	12.01
Location	4	34	7.78

Note: $p > .05$ for all comparisons.

Additional Analysis

Given the small sample employed in this study, the power of the previous analysis was low. To increase the power of those analyses, a second analysis was conducted in which mean values were computed for certain variables and substituted for missing values. The variables on which the greatest number of missing values occurred included the number and sum of psychological factors increasing pain. Mean values were not computed for some variables due to a lack of recorded scores. Using this procedure the sample size was increased to 73.

An average linkage cluster analysis was performed upon this sample. The agglomeration schedule for this analysis is presented in Table 4. An examination of this schedule for this analysis revealed that the largest rise in coefficient values was 20%. This study assumed that only percentage increases in the values of correlation coefficients of 50% or more were deemed to be sufficiently large to warrant a two or more cluster solution. Therefore, these results supported the formation of a single cluster.

Table 4

Cluster Analysis
Agglomeration Schedule
For N=73

Stage	Cluster 1	Cluster 2	Coefficient
1	36	62	22.000000
2	17	64	23.000000
3	30	43	26.000000
4	14	52	27.000000
5	16	21	28.000000
6	51	67	30.000000
7	11	31	34.000000
8	17	19	35.500000
9	10	41	37.000000
10	28	53	39.000000
11	4	51	39.000000
12	12	45	41.000000
13	16	70	43.000000
14	29	42	44.000000
15	5	8	48.000000
16	10	37	50.500000
17	9	36	57.000000
18	5	26	57.000000
19	11	48	58.000000
20	4	23	59.333332
21	6	22	60.000000
22	17	50	63.666668
23	7	66	66.000000
24	16	30	66.000000
25	4	29	66.000000
26	13	69	69.000000
27	1	20	75.000000
28	24	27	76.000000
29	18	73	77.000000
30	14	39	78.500000
31	5	17	83.250000
32	4	71	83.333336
33	9	63	94.666664
34	10	54	97.000000
35	5	33	97.428574
36	12	35	100.500000
37	13	44	101.500000
38	4	14	106.380951
39	57	65	107.000000
40	28	72	109.500000
41	49	61	113.000000
42	2	9	113.000000
43	18	32	120.500000
44	40	60	121.000000
45	5	28	131.166672
46	10	24	132.000000
47	25	40	133.500000
48	3	57	136.500000
49	4	16	139.839996
50	7	11	144.666672
51	13	46	145.333328
52	1	4	154.166672
53	18	38	163.666672
54	7	10	176.166672
55	1	12	176.745102
56	47	49	187.500000
57	1	5	200.118179
58	6	18	206.250000
59	34	56	229.000000
60	3	68	239.666672
61	13	59	246.500000
62	47	58	287.666656
63	3	6	299.750000
64	13	25	305.000000
65	1	7	307.609985
66	1	2	342.985718
67	1	3	476.157440
68	13	34	608.125000
69	47	55	757.250000
70	1	13	896.157898
71	1	47	1125.041748
72	1	15	1239.152832

Discussion

The first analysis conducted in this study identified two clusters, lending support to the traditional classification of headache experience. The second analysis identified only one cluster, lending support to the continuum theory of headache. Further discussion of the nature of each of these analyses may shed light on the conflicting conclusions drawn from each.

The traditional classification of headache distinguishes between migraine and muscle contraction headache on the basis of proposed differences in symptomatology and etiology. Regarding symptomatology, muscle contraction headache appears to be described by clients as "dull" and "band-like", whereas migraine is typically described as "sharp" and "throbbing". Muscle contraction headache is usually bilateral and migraine usually occurs unilaterally. While MCH is thought of as a single symptom, migraine headache tends to be viewed as part of a syndrome, including prodromal and premonitory symptoms. Certain symptoms such as nausea, which can be present both in chronic MCH and migraine, are thought to take more severe forms in migraine attacks, leading to vomiting. The intensity of pain itself, is thought to be more severe in migraine than in MCH. In this regard an assumption made by proponents of traditional headache classification is that given the greater severity of symptoms associated with

migraine, greater disability is incurred by the sufferer of migraine headaches relative to the sufferer of MCH. Regarding etiology, while MCH is commonly thought to be produced by sustained contraction of head and neck muscles resulting from stressful events, the etiology of migraine is thought to be far more complex. In summary, MCH is thought to be largely influenced by psychological factors while migraine is thought to be largely influenced by physical factors.

The results of the first analysis conducted in this study provide some support for the above mentioned distinctions between MCH and migraine headache. First, consistent with traditional classification, the two clusters differed significantly with regard to the number of physical factors identified as increasing pain, as well as the sum of physical factors affecting pain. Second, the reported average intensity of pain distinguished the two clusters, as the traditional classification of headache would predict. Third, the index of daily activity, the measure used in the study to assess extent of disability associated with headache, was found to significantly distinguish between two clusters. Fourth, certain categories of descriptors for pain used in the MMPQ were found to significantly distinguish between two clusters. These were the sensory, the affective, and the miscellaneous number of words chosen.

However, several of the differences between migraine

and MCH described by the traditional classification of headache were not supported by the results of the first analysis. For example, the unilaterality or bilaterality of headache did not significantly distinguish one cluster from the other. The adjectives "sharp", "throbbing" and "pressing" which were hypothesized to distinguish between headache types, did not significantly differentiate one cluster from another. Lastly, the nature of the circumstances under which headache first appeared did not differentiate clusters.

Overall, these results provide some support for the subjective impression of many health professionals working in the field of headache that a distinction should be made between migraine and MCH. As well, they support the practice of many health professionals who routinely take such a distinction for granted and prescribe specific treatments for headache based upon this distinction.

The results of the second analysis conducted in this study were more consistent with the continuum theory of headache experience in that only one cluster was obtained. This analysis however, included a treatment for missing values wherein average values were substituted for a significant number of missing values in the data set. The averaging effect of such a treatment may have imposed a false homogeneity among variables. Therefore the validity of these results appeared questionable in that this

treatment for missing variables may have masked the effects of significant differences between clusters in the study. Therefore, these results were not accepted as overriding the results of the initial analysis which provided support for the traditional classification of headache experience.

Limitations of the Present Study

Several limitations of the present study should be noted. First, the size of the sample employed in this initial analysis was small while the number of variables under study was high. In this case the power of the study to determine significant differences between clusters was low.

Second, the sample was comprised almost entirely of women. Consequently, the extent to which these results generalize to men is unknown. Therefore, conclusions about headache experience in men cannot be drawn from the present study.

Third, the present study employed a sample drawn from a tertiary care facility. Therefore, the subjects in this study had likely experienced significant headaches for an extended period of time. The extent to which these results would apply to a sample of subjects whose headaches extended over a wider range of severity requires further study.

Fourth, this study did not examine all of the variables which have been hypothesized to differentiate headache

experience. For example, the presence of nausea or vomiting and the experience of auras are hallmarks of migraine headache for many health care professionals and may prove to be worthwhile variables to investigate. In addition, it would be worthwhile to include the length of time that a client has suffered from headache as a variable for consideration. Had some of these other variables been examined, additional significant differences between clusters may have been found.

Fifth, the stability of the cluster solution obtained in this study is unknown. Therefore, it is not clear if the same cluster solution would be obtained if the analysis was repeated with a different sample. Further study is required to address this issue.

Finally, this study only examined cross-sectional data regarding headache experience. It did not study longitudinal data regarding headache which could provide useful information regarding the utility of each theory.

Implications for Future Research

Given the low power of the present study, it would be useful to replicate the study with a larger sample size which would serve to strengthen its power to detect significant differences between clusters. A larger sample size should also obviate the need to resort to using

unsatisfactory treatments for missing values, as the larger the sample, the more complete the data set.

It is noteworthy that despite the low power of the study, certain variables distinguished between clusters, namely, the physical factors influencing headache, certain intensity measures of headache, and the degree of disability associated with headache. It may be useful for future researchers to concentrate their efforts in further delineating the contributions of these particular variables to the headache experience, as well as to headache nosology.

Further research may also include additional variables thought to have an impact upon the headache experience but which were not investigated in this study. For example, the presence of nausea or vomiting and the experience of auras are hallmarks of migraine headache for many physicians and may prove to be worthwhile variables to investigate in future research. In addition, it would be worthwhile to include the length of time that a client has suffered from headache as a variable for consideration in future studies.

The use of more accurate history taking may be well advised in future research examining the validity of the continuum theory of headache and the traditional classification of headache. Proponents of a traditional approach may argue that the experience of the client who presents with solely migrainous symptoms lends support to the view that migraine represents a phenomenal experience

distinct from MCH. This assertion could be tested by accurate record keeping on the part of a researcher. If the more severe symptoms of headache make a stronger impression on a client and his family, he/she may not accurately recall his true headache experience. As well, history taking on the physician's part may be inadequate in this regard, masking the client's experience with the milder symptoms of the headache continuum. In this case the use of a standard questionnaire which would carefully search the client's headache history for details regarding the possible experience of muscle contraction symptoms in the past, may be useful.

The results of standardized and careful history taking would then be reviewed: if a large number of health professionals found that their migrainous clients indeed had a significant history of muscle contraction headache symptomatology prior to the onset of migrainous symptoms, the continuum theory would be further supported. If not, the traditional classification of headache would be supported.

Proponents of the traditional approach would also argue that another shortcoming of the continuum model occurs when patients manifest combined headaches, or the simultaneous or concurrent symptoms of both migraine and MCH. Within the traditional model, the client is simply manifesting two distinct headache experiences at the same time. However, if

these headache experiences are not distinct but represent two extremes of a continuum, this scenario poses a problem for the continuum model. The use of longitudinal study in headache research may be of value in such cases. Proponents of the continuum model may argue that with regard to clients who appear to manifest symptomatology associated with both ends of the continuum, it may be that this headache experience represents a transitional period between the predominance of MCH symptomatology and purely migrainous symptomatology. Such a hypothesis could be tested by means of longitudinal study of clients diagnosed with combined headache. First, if the headache history of clients solely manifesting either MCH symptoms or combined headache is monitored over time, and a natural progression from a predominance of milder to more severe symptoms is observed, the continuum theory of headache would accrue support. If such a pattern was not observed to obtain, the adoption of the traditional classification of headache may be the better theory to adopt.

On the other hand, longitudinal study of headache may incorporate specific interventions the aim of which would be to halt the assumed natural progression from less severe to more severe symptoms on a continuum model of headache. In the case of clients solely presenting with MCH symptomatology, whether children or adults, instruction in relaxation training and other stress management techniques

may keep symptoms from escalating in severity. In the case of clients presenting with combined headache, long term observation of their symptoms may reveal that relaxation training significantly reduces, or eliminates migrainous symptoms from their headache experience. If such results were obtained, support would be garnered for the continuum theory of headache. However, if clients continue to manifest both types of symptomatology, traditionalists may argue that this result lends support to the argument for distinct types of headache.

Finally, with regard to proposed treatments for headache, because of the numerous limitations noted previously, the results of the present study should not be used to prematurely select treatment strategies. In the future, more powerful and rigorous longitudinal studies may enable the clinician to decide not only between different orientations to headache experience, but between different treatment strategems. For example, if longitudinal studies support the continuum theory of headache, the child or adult presenting with mild symptoms of MCH may be cautioned that if left unattended, these symptoms may escalate in severity, or that they may develop migrainous symptoms. Treatment may expand considerably beyond a prescription for analgesics and an admonition to feel less stressed. The use of analgesics may be discouraged altogether and the child may be referred to a specialist in relaxation training.

References

Ad Hoc Committee on Classification of Headache. (1962). Classification of Headache. Journal of the American Medical Association. 179, 717-718.

Allen, R.A., & Weinmann, R.L. (1982). The McGill-Melzack Pain Questionnaire in the diagnosis of headache. Headache, 22, 20-29.

Anthony, M., & Lance, J. (1989). Plasma serotonin in patients with chronic tension headaches. Journal of Neurology, Neurosurgery, and Psychiatry, 52, 182-184.

Bakal, D. (1982). The psychobiology of chronic headache. New York: Springer Publishing Company.

Bakal, D.A. & Kaganov, J. (1977). Muscle contraction and migraine headache: Psychophysiologic comparison. Headache, 17, 208-215.

Bakal, D.A., & Kaganov, J.A. (1979) Symptom characteristics of chronic and non-chronic headache sufferers. Headache, 19, 285-289.

Bakal, D. A. , Demjen, S., & Kaganov, J. A. (1981). Cognitive behavioral treatment of chronic headache. Headache, 21, 81-86.

Blanchard, E. B., & Andrasik, F. (1985). Management of chronic headaches: A psychological approach. New York: Pergamon Press.

Caviness, V.S., & O'Brien, P. Current concepts: Headache. New England Journal of Medicine, 302, 446-449.

Charter, R.A., & Nehemkis, A.M. (1983). The language of pain intensity and complexity: New methods of scoring the McGill Pain Questionnaire. Perceptual and Motor Skills, 56, 519-537.

Cohen, M.J. (1978). Psychophysiological studies of headache: Is there similarity between migraine and muscle contraction headache? Headache, 18, 189-196.

Couch, J.R., & Hassanein, F.R. (1977). Platelet aggregability in migraine. Neurology, 27, 843-848.

Dalessio, D.J. (1972). Wolff's Headache and other head pain. New York: Oxford University Press.

Dalessio, D.J. (1975). Classification and mechanism of migraine. Headache, 19, 114-121.

Deshmukh, S.V., & Meyer, J.S. (1977). Cyclic changes in platelet dynamics and the pathogenesis and prophylaxis of migraine. Headache, 17, 101-108.

Edmeads, J. (1982) Migraine as a model of Neurogenic Ischemia. Pain, 287-288.

Everitt, B. (1980) Cluster analysis. London: Heineman.

Featherstone, H.J. (1985). Migraine and muscle contraction headaches: A continuum. Headache, 25, 194-198.

Friedman, A.P. (1982). Overview of migraine. In Advances in Neurology. New York: Raven Press.

Friedman, A.P. (1979). Nature of headache. Headache, 19, 163-167.

Gawel, M., Burkitt, M., & Rose, F.C. (1979). The platelet release reaction during migraine attacks. Headache, 19, 323-327.

Gotoh, F., Komatsumoto, S., Araki, N., & Gomi, S. (1984). Noradrenergic nervous activity in migraine. Archives of Neurology, 41, 951-955.

Graham, J.R., & Wolff, H.G. (1938). Mechanism of migraine headache and action of ergotamine tartrate. Archives of Neurology and Psychiatry, 39, 737-763.

Hanington, E., Jones, R.J., Amess, J.A.L., & Wachowicz, B. (1981). Migraine: a platelet disorder. The Lancet, 720-723.

Havanka-Kanniainen, Tolonen, U., & Myllyla, V.V. (1986). Autonomic dysfunction in adult migraineurs. Headache, 26, 425-430.

Haynes, S.N., Cuevas, J., & Gannon, L.R. (1982). The psychophysiological etiology of muscle-contraction headache. Headache, 22, 122-132.

Hilton, B.P., & Cumings, J.N. (1972). An assessment of platelet aggregation induced by 5-hydroxytryptamine. Journal of Clinical Pathology, 24, 250-258.

Hunter, M. (1983). The Headache Scale: A new approach to the assessment of headache pain based on pain descriptors. Pain, 16, 361-373.

International Association for the Study of Pain. (1986). Pain, Supplement 3. Elsevier Science Publishers: Amsterdam.

Iversen, H.K., Langemark, M., Andersson, P.G., Hansen, P.E., & Olesen, J. (1990). Clinical characteristics of migraine and episodic tension-type headache in relation to old and new diagnostic criteria. Headache, 30, 514-519.

Kaganov, J., Bakal, D. & Dunn, B. (1981). The differential contribution of muscle contraction and migraine symptoms of problem headache in the general population. Headache, 21, 157-163.

Kalendovsky, Z., & Austin, J.H. (1975). Complicated migraine -its association with increased platelet aggregability and abnormal plasma coagulation factors. Headache, 15, 18-35.

Koehler, S.M., & Glaros, A. (1988) The effect of aspartame on migraine headache. Headache, 28, 10-13.

Kohlenberg, R.J. (1982). Tyramine sensitivity in dietary migraine: a critical review. Headache, 22, 30-34.

Lance, J.W. (1981). Headache. Annals of Neurology, 10, 1-10.

Lance, J.W. (1982). Advances in Neurology, 33. New York: Raven Press.

Melzack, R. (1975). The McGill Pain Questionnaire: Major properties and scoring methods. Pain, 1, 277-299.

Melzack, R. (1983). Pain measurement and assessment. New York: Raven Press.

Melzack, R., & Torgerson, W.S. (1971). On the language of pain. Anesthesiology, 34, 50-59.

Meyer, J.S., & Hardenberg, J. (1983). Clinical effectiveness of calcium entry blockers in prophylactic treatment of migraine and cluster headaches. Headache, 23, 266-277.

Muck-Seler, D.M., Deanovic, Z., & Dupelj, M. (1979) Platelet serotonin (5-HT) and 5-HT releasing factor in plasma of migrainous patients. Headache, 19, 14-17.

Mullaney, D.J. (1973). EMG biofeedback and tension headache: A controlled outcome study. Psychosomatic Medicine, 35, 484-496.

Norusis, M.J. (1988). SPSS/PC + advanced statistics, 2.0, SPSS: Chicago.

Olesen, J. (1988). Clinical characterization of tension headache. Basic Mechanisms of Headache, Amsterdam: Elsevier.

Philips, C. (1978). Tension headache: Theoretical problems. Behavior Research and Therapy, 16, 249-261.

Philips, C. (1977). The modification of tension headache pain using EMG biofeedback. Behavior Research and Therapy, 15, 119-129.

Pozniak-Patewicz, E. (1976). "Cephalgic" spasm of head and neck muscles. Headache, 16, 261-265.

Reich, B.A. (1989). Non-invasive treatment of vascular and muscle contraction headache: a comparative longitudinal clinical study. Headache, 29, 34-41.

Rolf, L.H., Wiele, G., & Brune, G.G. (1981). 5-Hydroxytryptamine in platelets of patients with muscle contraction headache. Headache, 21, 10-11.

Rose, C.F., & Capildeo, R. (1983). Migraine: Definition and classification. Cephalalgia, 3, 225-229.

Rubin, L.S., Graham, D., Pasker, R., & Calhoun, W. (1985). Autonomic nervous system dysfunction in common migraine. Headache, 25, 40-48.

Sandler, M., Youdim, M.B.H., & Hanington, E. (1974). A phenylethylamine oxidising defect in migraine. Nature, 250, 335-337.

Selby, G., & Lance, J.W. (1960). Observations on 500 cases of migraine and allied vascular headache. Journal of Neurology, Neurosurgery, and Psychiatry, 23, 322-327.

Shimomura, T., Nishikawa, S., & Takahashi, K. (1986). Hereditary dysrhythmic headache. Headache, 26, 33-36.

Takeshima, T., & Takahashi, K. (1988). The relationship between muscle contraction headache and migraine: a multivariate analysis study. Headache, 28, 272-277.

Takeshima, T., Takao, Y., & Takahashi, K. (1987). Pupillary sympathetic hypofunction and asymmetry in muscle contraction headache and migraine. Cephalalgia, 7, 257-262.

Thompson, J.K., Haber, J.D., Figueroa, J.L., & Adams, H.E. (1980). A replication and generalization of the "Psychobiological" model of headache. Headache, 20, 199-203.

Tunis, M.M., & Wolff, H.G. (1954). Cranial artery vasoconstriction and muscle contraction headache. Archives of Neurology and Psychiatry, 425-433.

Waters, W.E. (1974). The Pontypridd Headache Survey. Headache, 14, 81-90.

Wolff, H.G. (1963). Headache and other head pains. New York: Oxford University Press.

Ziegler, D.K., Hassanein, R.S., & Hassanein, K. (1972). Headache syndromes suggested by factor analysis of symptom variables in a headache prone population. Journal of Chronic Diseases, 25, 353-363.

APPENDIX A

THE PAIN EVALUATION QUESTIONNAIRE

Pain Evaluation Questionnaire

Please print or write clearly:

Name: _____ Today's Date: _____

Address: _____

_____
(postal code)

Telephone: (home) _____ (work) _____

GENERAL INFORMATION

Age: (in years) _____ Date of Birth _____

Sex: _____ Male _____ Female

Education: _____

Occupation: _____

Family Doctor: (name) _____ Tel. No. _____

Address: _____

_____

MEDICAL HISTORY

When did you first experience the pain problem for which you are seeking help?

_____

Under what circumstances did the pain begin? (check as many as apply)

___ an accident at work

___ an accident at home

___ an accident away from home or work

___ a time of personal or family stress

___ a time of stress or conflict at work

___ during or following an illness

___ following surgery

___ the pain "just began"

___ cannot remember

Please describe the circumstances in which the pain began.

_____

_____

_____

WHERE IS YOUR PAIN?

Please mark, on the drawings below, the areas where you feel pain. Then, near the areas you have marked, put an E if the pain is external or an I if the pain is internal. Put EI if the pain is both external and internal. Also, if you have one or more areas which can trigger your pain when pressure is applied to them, mark each "trigger point" with an X.

COMMENTS: _____

_____

If you have more than one type of pain, which one gives you the most trouble?

_____

_____

WHAT DOES YOUR PAIN FEEL LIKE?

Some of the words below describe your present pain. Please circle those words that best describe your pain. Use only a single word in each appropriate category--the one that applies best. Leave out any category that is not suitable. If you have more than one pain problem, please focus on the pain problem which gives you the most trouble.

1		2		3		4	
1	Flickering	1	Jumping	1	Pricking	1	Sharp
2	Quivering	2	Flashing	2	Boring	2	Cutting
3	Pulsing	3	Shooting	3	Drilling	3	Lacerating
4	Throbbing			4	Stabbing		
5	Beating			5	Lancinating		

5		6		7		8	
1	Pinching	1	Tugging	1	Hot	1	Tingling
2	Pressing	2	Pulling	2	Burning	2	Itchy
3	Gnawing	3	Wrenching	3	Scalding	3	Smarting
4	Cramping			4	Searing	4	Stinging

9		10		11		12	
1	Dull	1	Tender	1	Tiring	1	Sickening
2	Sore	2	Taut	2	Exhausting	2	Suffocating
3	Hurting	3	Rasping				
4	Aching	4	Splitting				

13		14		15		16	
1	Fearful	1	Punishing	1	Wretched	1	Nagging
2	Frightful	2	Gruelling	2	Blinding	2	Troublesome
3	Terrifying	3	Cruel			3	Miserable
		4	Vicious			4	Intense
		5	Killing			5	Unbearable

17		18		19		20	
1	Spreading	1	Tight	1	Cool	1	Nagging
2	Radiating	2	Numb	2	Cold	2	Nauseating
3	Penetrating	3	Drawing	3	Freezing	3	Agonizing
4	Piercing	4	Squeezing			4	Dreadful
		5	Tearing			5	Torturing

People agree that the following five words represent pain of increasing intensity:

1	2	3	4	5
mild	discomforting	distressing	horrible	excrutiating

To answer each question below, write the number of the most appropriate word in the space beside the question.

1. Which word describes your pain right now? _____

2. Which word describes it at its worst? _____

3. Which word describes it when it is least? _____

PRESENT PAIN PATTERN

Your answers to the following questions will help us to understand the nature of your pain at the present time. In answering these questions, think about how you have felt during the past month. If you have more than one pain problem, please answer these questions in terms of the pain problem that gives you the most trouble.

1. Please put an x beside the statement that best describes your pain at the present time. Choose only one statement.

___ My pain is continuous, steady, constant.

___ My pain is periodic, intermittent, comes and goes.

___ My pain is brief, momentary, transient.

For each of the questions below, circle the number that best describes how you have been feeling during the past month.

2. How much of the time were you in pain?

1	2	3	4	5	6	7
not at all						all of the time

3. On average, how intense was your pain?

1	2	3	4	5	6	7
no pain at all						excruciating

4. On average, how much energy did you have?

1	2	3	4	5	6	7
no energy at all						full of energy

PRESENT PAIN PATTERN (cont'd)

Pain often varies in intensity throughout the day. During the past month how has your pain varied during a typical day? On each of the scales below, circle the number that best describes how intense your pain is during that part of the day.

When you first wake up.

1	2	3	4	5	6	7
no pain at all						excruciating

Early morning.

1	2	3	4	5	6	7
no pain at all						excruciating

Late morning.

1	2	3	4	5	6	7
no pain at all						excruciating

Early afternoon.

1	2	3	4	5	6	7
no pain at all						excruciating

Late afternoon.

1	2	3	4	5	6	7
no pain at all						excruciating

Early evening.

1	2	3	4	5	6	7
no pain at all						excruciating

Late evening.

1	2	3	4	5	6	7
no pain at all						excruciating

Bedtime.

1	2	3	4	5	6	7
no pain at all						excruciating

WHAT AFFECTS YOUR PAIN?

Please read through the list below and circle the number that best describes how your pain is affected by each thing. Again, if you have more than one pain problem please focus on the problem that gives you the most trouble.

	USUALLY INCREASES PAIN	SOMETIMES INCREASES PAIN	NO EFFECT ON PAIN	SOMETIMES DECREASES PAIN	USUALLY DECEASES PAIN
Alcohol	+2	+1	0	-1	-2
Coffee	+2	+1	0	-1	-2
Certain foods	+2	+1	0	-1	-2
Eating	+2	+1	0	-1	-2
Heat	+2	+1	0	-1	-2
Cold	+2	+1	0	-1	-2
Dampness	+2	+1	0	-1	-2
Massage	+2	+1	0	-1	-2
Pressure	+2	+1	0	-1	-2
Movement	+2	+1	0	-1	-2
Staying still	+2	+1	0	-1	-2
Standing	+2	+1	0	-1	-2
Lying down	+2	+1	0	-1	-2
Sitting or driving	+2	+1	0	-1	-2
Chores at home	+2	+1	0	-1	-2
Walking	+2	+1	0	-1	-2
Activity at work	+2	+1	0	-1	-2
Distraction (TV, reading etc.)	+2	+1	0	-1	-2
Urinating, bowel movements	+2	+1	0	-1	-2
Feeling happy or good	+2	+1	0	-1	-2

WHAT AFFECTS YOUR PAIN? (cont'd)

	USUALLY INCREASES PAIN	SOMETIMES INCREASES PAIN	NO EFFECT ON PAIN	SOMETIMES DECREASES PAIN	USUALLY DECEASES PAIN
Feeling, tense anxious, or stressed	+2	+1	0	-1	-2
Feeling calm or relaxed	+2	+1	0	-1	-2
Feeling sad or depressed	+2	+1	0	-1	-2
Feeling angry or frustrated	+2	+1	0	-1	-2
Being touched	+2	+1	0	-1	-2
Bright lights or loud noises	+2	+1	0	-1	-2
Reading too long	+2	+1	0	-1	-2
Mild exercise	+2	+1	0	-1	-2
Sexual relations	+2	+1	0	-1	-2
Coughing or sneezing	+2	+1	0	-1	-2
Feeling overtired	+2	+1	0	-1	-2
Lifting or reaching	+2	+1	0	-1	-2

During the past month how much control do you feel you've had over your pain?

1	2	3	4	5	6	7
No control at all						A great deal of control

During the past month how much control do you feel you've had over your life?

1	2	3	4	5	6	7
No control at all						A great deal of control

During the past month how well have you been able to cope with your problems?

1	2	3	4	5	6	7
Extremely poorly						Extremely well

During the past month how hopeful have you felt about the future?

1	2	3	4	5	6	7
Not at all hopeful						Extremely hopeful

Over the past year has anything (not releated to pain) happened to you or to someone close to you that has caused you added stress or difficulties?

_____ No _____ Yes

Have you ever received counselling, treatment, or mediciation for emotional difficulties, problems with your nerves, stress at work, or problems in your marriage or family? (include any present treatments)

_____ No _____ Yes

If yes, please describe briefly what the problems were, when they occurred, who treated you, and what treatment you received.

_____

_____

_____

_____

Have you tried anything on your own to reduce or eliminate the pain?

_____ No _____ Yes

If yes, please describe. _____

_____

_____

To understand fully how pain affects you, think of a recent incident when pain was particularly bad. Close your eyes for a moment and picture that situation. Relive in your mind that incident. What I would like you to do is to tune into the thoughts, images, and feelings you had in this situation. Please describe these thoughts and feelings in as much detail as possible. What were the thoughts and feelings you had when you first noticed the pain?

What were the feelings and thoughts that occurred to you as you tried to cope with the pain? (Please be specific.)

Do you have such thoughts and feelings in other situations?

Are there any thoughts or images that you find helpful in managing or reducing your pain? (Describe in as much detail as possible.)

CURRENT TREATMENTS

What medications have you been taking for your pain during the past year?

Prescription medication:

Name or Type of Drug	Dosage or Strength	Amount Taken Daily	Amount Taken Weekly	Effects	Date Started	Date Stopped

Non-prescription medication (aspirin etc.):

Name or Type of Drug	Dosage or Strength	Amount Taken Daily	Amount Taken Weekly	Effects	Date Started	Date Stopped

What other types of treatment are you presently receiving or using for your pain?

Treatment	Effects

Are you scheduled for surgery or any other medical procedures for your pain in the future?

_____ No _____ Yes

If yes, please state what the procedure is and when you expect to have it done.

_____

_____

PAST TREATMENT

Since your pain began, what doctors have you consulted?

TYPE OF DOCTOR	WHEN?	WHAT TREATMENT DID YOU RECEIVE?	WHAT WERE THE RESULTS?
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____

Since your pain began, what other health professionals have been treated by?

	WHEN?	WHAT TREATMENT?	RESULTS?
PSYCHOLOGIST	_____	_____	_____
CHIROPRACTOR	_____	_____	_____
ACUPUNCTURIST	_____	_____	_____
OTHER	_____	_____	_____

(please specify) _____

Have you ever felt that your condition was not been taken seriously or that you were being treated as if you were faking or exaggerating?

_____ No _____ Yes

If yes, please explain: _____

_____

Since your pain began, have you ever been treated by a physiotherapist?

_____ No _____ Yes

If yes, when? _____

Approximately how many miles did you have to travel for physiotherapy?

_____ miles

Did you attend physiotherapy more than once? _____ No _____ Yes

If yes, how many times a week did you attend physiotherapy? ____ times.

How long did you receive physiotherapy? _____ less than 2 months

_____ 2 to 6 month

_____ more than 6 months

Overall, how helpful was physiotherapy? Circle the number that best describes how helpful physiotherapy was.

1	2	3	4	5
not at all helpful		somewhat		extremely helpful

Please indicate with a checkmark the extent to which the following treatments were helpful. If you have never had a particular treatment put a checkmark in the column marked "Never Had".

	Never Had	Helpful	No Effect	Made Pain Worse	Not Sure of Effect
Hot Packs	___	___	___	___	___
Ice	___	___	___	___	___
Electrical Treatments	___	___	___	___	___
Deep Heat (Short Wave)	___	___	___	___	___
Ultrasound	___	___	___	___	___
Home Exercises Given by Physiotherapist	___	___	___	___	___
Mobilizations (Moving of joints by physiotherapist)	___	___	___	___	___
Back Education	___	___	___	___	___
Traction	___	___	___	___	___
Biofeedback	___	___	___	___	___
Relaxation Exercises	___	___	___	___	___
Acupuncture	___	___	___	___	___
Other: (please specify)	___	___	___	___	___

_____

Have you ever worn a neck brace? _____ No _____ Yes

If yes, what type of brace did you wear?

_____ Harris Brace _____ Sacroiliac Brace

_____ Depuy _____ Other (please specify) _____

_____ Camp _____ Don't know

Was the brace helpful? _____ No _____ Yes

How long did you wear it? _____ (weeks, months)

Are you presently wearing a neck brace? _____ No _____ Yes

Have you ever worn a neck collar? _____ No _____ Yes

If yes, was it helpful? _____ No _____ Yes

How long did you wear it? _____ (weeks, months)

Are you presently wearing a collar?

_____ No

_____ Yes, on occasion. When? _____

_____ Yes, all the time

Have you ever had a Transcutaneous Electrical Nerve Stimulator (TENS) unit for use at home?

_____ No _____ Yes

If yes, how long did you use it? _____

Have you ever been given specific exercises by a physiotherapist to do at home?

_____ No _____ Yes

If yes, how often did you do them? _____ Never

_____ Occasionally

_____ Regularly (at least 3x times a week)

Are you presently using these exercises? _____ No _____ Yes

If not, please explain. _____

_____

How important do you think exercise is for your particular pain problem?

1	2	3	4	5
not at all important		somewhat		extremely important

Which of the following best describes you?

_____ I participate in a physical activity on a regular basis (e.g., swim or walk at least three times a week).

_____ I enjoy occasional physical activity (less than three times per week).

_____ I am not involved in physical activities.

Please add any additional comments that you think may be helpful to the physiotherapist.

_____

_____

_____

_____

_____

PAIN AND DAILY ACTIVITIES

1. Thinking back over the past month, how much did your pain interfere with the following aspects of your life? Circle the number that best describes how much your pain interfered with each of the following.

	NOT AT ALL	A LITTLE	A FAIR AMOUNT	VERY MUCH
a. Work outside the house (if applicable)	1	2	3	4
b. House and yard work	1	2	3	4
c. Child care (if applicable)	1	2	3	4
d. Recreation or hobbies	1	2	3	4
e. Social Activities	1	2	3	4
f. Close Relationships	1	2	3	4
g. Sexual Relationships	1	2	3	4
h. Eating	1	2	3	4
i. Sleeping	1	2	3	4
j. Tolerance for frustration .	1	2	3	4

2. If and when your pain interferes with your daily life, do you find that you need to stop what you are doing? (check one)

_____ NEVER _____ SOMETIMES _____ OFTEN

3. How many hours (not counting bedtime) do you spend lying down or resting because of pain? _____ hours

4. How long can you usually do the following activities before changing to another activity? Give your answer in hours or minutes.

	HOURS	MINUTES
Sitting	_____	_____
Walking	_____	_____
Standing	_____	_____
Reading	_____	_____
Driving	_____	_____

PAIN AND SLEEP

Thinking back over the past month, how has pain affected your sleep? For each of the following questions, circle the number that best describes the extent to which pain has affected your sleep.

	Almost Never	Sometimes	Often	Almost Always
1. How often do you have trouble falling asleep because of the pain?	1	2	3	4
2. How often do you need medication to fall asleep?	1	2	3	4
3. How often are you awakened by pain during the night?	1	2	3	4
4. How often are you awakened by pain in the morning?	1	2	3	4

What do you usually do when you are awakened by pain during the night?

_____ I stay in bed and try to go back to sleep.
_____ I get up to empty my bladder, then go back to bed.
_____ I get out of bed and stay up for awhile.
_____ I take medication.
_____ Other (please indicate): _____

_____

If married, what does your husband or wife do when you wake up at night with pain? (please be specific): _____

_____

_____

What is the average number of hours of sleep you get per night?
_____ hours.

Has this changed over the last year? _____ No _____ Yes

If yes, how? _____

_____

_____

PAIN AND WORK

1. Check as many as apply. At the present time are you:

_____ working full-time for someone else?
_____ working part-time for someone else?
_____ self-employed full-time?
_____ self-employed part-time?
_____ the main one taking care of the home?
_____ the main one taking care of the children?
_____ retired ..... for how long? _____
_____ unemployed ..... for how long? _____

If you are presently working, please describe your work

_____

_____

If at present you are unemployed, retired, or working part-time, is this due to your pain condition? _____ No _____ Yes

If yes, how has pain interfered with your ability to work?

_____

_____

_____

If you have worked for someone else or are doing so at present, have your employers been helpful and understanding of your pain problems?
_____ No _____ Yes

Please explain: _____

_____

_____

If you are not working at present, have you tried to return to work (either to a previous job or starting a new one)?

_____ No _____ Yes

If yes, when? _____

What type of work? _____

For how long? _____

What happened? _____

Are you presently concerned about financial problems or major debts?
_____ No _____ Yes

Please explain: _____

_____

_____

What are your present sources of income?

_____

_____

Are you presently receiving

worker's compensation	___ No	___ Yes
disability pension	___ No	___ Yes
insurance benefits	___ No	___ Yes

In the past have you received any financial benefits or compensation for your pain or injury? _____ No _____ Yes

If yes, what kind? _____

Are you presently applying for financial benefits or compensation resulting from your pain or injury? _____ No _____ Yes

If yes, what kind _____

Are you presently involved in legal action that is related to your pain or injury? _____ No _____ Yes

If yes, what is the nature of this action? _____

_____

OTHER HEALTH PROBLEMS

Please list any other health problems (other than pain problems) that you currently have:

Date of Onset	Type of Problem and Treatment
_____	_____
_____	_____
_____	_____

Please list all other medications you are taking at present (other than those for pain):

_____

_____

_____

Please list any health problems or major illnesses you have had in the past:

Date	Type of Problem or Illness
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____

Please list any surgery you have had that was not related to your pain:

Date	Type of Surgery
_____	_____
_____	_____
_____	_____
_____	_____

PERSONAL HABITS

1. How much tea or coffee do you drink per day? _____

2. How much pop do you drink per day? _____

3. How much do you smoke per day? _____

4. How much alcohol do you drink per week? _____

SOCIAL INFORMATION

Present marital status: (Check one)

_____ Single _____ _____ Remarried--How long? _____

_____ Engaged--How long? _____ _____ Separated--How long? _____

_____ Cohabitating--How long? _____ _____ Divorced--How long? _____

_____ Married--How long? _____ _____ Widowed--How long? _____

Partner's occupation: _____

Number of children (boys) _____ (ages) _____
(girls) _____ (ages) _____

With whom do you live? _____

Whom should we contact in an emergency?

Name: _____ Relationship to you: _____

Address: _____ Telephone No.: _____

Thank you for completing this questionnaire. Is there any other information that you feel may help us better understand you and you present problem?

_____

_____

_____

_____