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THE EFFECTS OF AEROBIC EXERCISE ON MIGRAINE

by

DONNA-MARIE C. LOCKETT

**A thesis submitted to the
Faculty of Graduate Studies and Research
in partial fulfillment of the
requirements for the degree of
Master of Arts**

Department of Psychology

Carleton University

Ottawa, Ontario

September, 1990

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**The undersigned recommend to the Faculty of Graduate Studies
and Research acceptance of the thesis**

"The Effects of Aerobic Exercise on Migraine"

**submitted by Donna-Marie C. Lockett
in partial fulfillment of the requirements for the
degree of Master of Arts**

Thesis Supervisor

Chair, Department of Psychology

Carleton University

September 20, 1990

ABSTRACT

Previous research has implicated strenuous exercise as a trigger to migraines. Others have found that exercise, performed at the onset of a headache, may be effective in aborting the pain. However, the potential prophylactic effects of a long term exercise program have not yet been explored. An exploratory study was therefore conducted to examine the effects of a long-term cardiovascular exercise program on classical migraines. Twenty classical migraineurs were randomly assigned to a treatment or waiting-list control group. The treatment group ($n = 11$) received six weeks of aerobic fitness classes, while those in the control group ($n = 9$) were told that they would receive their classes upon completion of the study. Baseline, mid-treatment, post-treatment, and follow-up measures were taken on cardiovascular fitness, migraine activity and migraine impact. The Canadian Aerobic Fitness test was used as a measure of cardiovascular fitness. A headache diary was used to determine the Frequency, Intensity, and Duration of migraines. The Pain Severity, Life Control, Affective Distress, Support, and General Activity scales of the West Haven-Yale Multidimensional Pain Inventory were used to measure the impact of having migraines. The results of multiple repeated measures analyses of variance and multivariate analyses of variance suggest that the aerobic classes were effective in improving cardiovascular fitness. Pain Severity also improved significantly for the treatment group. Other variables, which indicated some improvement for the treatment group but which failed to reach significance, include Affective Distress, Frequency, Intensity and Duration of migraines. Such promising results would suggest that future research is warranted to further examine the effects of aerobic

activity on migraines.

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Introduction

"Take a clove of garlic, peel it, cut off both ends, make an incision in the temple, with a large lancet, producing a cavity under the skin which is large enough to introduce the garlic, and cover it completely. Apply some compresses on top, and tighten strongly, leave them thus for about 15 hours, then remove them, extract the garlic, leave the wound for two or three days, then apply cotton drenched with butter until it suppurates, then bandage with salve until it heals" (Abulcasis, 11th Century, as told in Thomas, 1887, pp. 5-6).

Such was the chosen form of treatment for migraines in the 11th century.

While scientific and clinical research in the etiology and treatment of migraine has advanced considerably in the past one thousand years, there is much about migraines which remains a mystery.

The Migraine

The concept of migraine first appeared in the writings of Aretaeus of Cappadocia, in the first century A.D. (Isler, 1987). According to Isler (1987), Aretaeus of Cappadocia described the migraine as heterocrania, meaning an affection of one or the other side of the skull, exactly up the midline, with blackness before the eyes, profuse sweating, nausea, vomiting, faintness, and photophobia.

Recent attempts to arrive at a conceptual definition of migraine have generally failed. Even the most widely used criteria, offered by the Ad Hoc committee for classifying headaches (Friedman, 1962), has repeatedly been used in defining migraines, when the word definition is not to be found in the text. In fact,

the chairperson of that committee, Arnold Friedman, later lamented about the lack of agreement on the definition of migraine (Friedman, 1975). Responding to this, Blau (1987) has offered the following definition of migraine:

"Episodic headaches lasting 2 to 72 hours with total freedom between attacks. The headaches must be associated with visual or gastrointestinal disturbances, or both. The visual symptoms occur as an aura before, and/or photophobia during, the headache phase. If there are no visual but only alimentary disturbances, then vomiting must feature in some attacks" (Blau, 1987, p. 25).

While problems of defining migraine have precluded a true account of its occurrence, most have estimated that between 5 and 25 percent of the population experiences some form of migraine headache (Atkinson & Appenzeller, 1982; Crisp, McGuiness, Kalucy, Ralph, & Harris, 1977; Dalsgaard-Nielson, 1970; Ogden, 1952; Ogunyemi, 1984; Selby & Lance, 1960; Waters & O'Connor, 1975; Wilson, 1940). Women generally report experiencing more migraines than men on the order of 2-3 to 1 (Philips, 1977; Selby & Lance, 1960; Waters, 1974). The typical age of onset is 20 years, although migraines become relatively common as early as 10 years of age (Adams et al., 1980).

Migraine attacks may occur as frequently as two to three times a week (Kroner, 1983), but typically occur one to four times a month (Selby & Lance, 1960), with about half of those with acute migraine attacks also having frequent mild to moderate headaches between their migraines (Olesen, 1978).

The most common types of migraines include classical, common, and cluster. Of these, the classical migraine, or migraine with aura, is the most prototypical.

The Classical Migraine

The classical migraine occurs in approximately 10% of migraine patients and generally develops between the ages of 5 and 35 (Atkinson & Appenzeler, 1982). The Headache Classification Committee of the International Headache Society (1988), under the chair of Jes Olesen, has described the classical migraine as an:

"Idiopathic, recurring disorder manifesting with attacks of neurological symptoms unequivocally localized to cerebral cortex or brain stem, usually gradually developed over 5-20 minutes and usually lasting less than 60 minutes. Headache, nausea, and/or photophobia usually follow neurological aura symptoms directly or after a free interval of less than one hour. The headache usually lasts 4-72 hours, but may be completely absent" (pp. 20-21).

This description discusses, generally, the aura and headache phases of the migraine. Many (e.g. Atkinson & Appenzeller, 1982; Blau, 1980; Kroner, 1983) have included a third phase to the migraine process, the prodrome, which appears to precede both the aura and the headache. Some have also described a fourth phase, the postdrome, which follows the migraine (Blau, 1987). Thus, a classic migraine will typically (though not always) involve a prodrome, followed by an aura phase, culminating in the head pain, and terminating with the postdrome phase.

The migraine prodrome refers to symptoms with insidious onset, lasting anywhere from one to twenty-four hours and affecting mood, behaviour, wakefulness, and/or fluid balance (Atkinson & Appenzeler, 1982; Kroner, 1983). Prodromal symptoms include feeling extremely well before an attack, feeling low and depressed, irritable, anxious, sleepy, hungry with cravings for sweets and carbohydrates,

experiencing diarrhea, constipation, and/or nausea (Blau, 1980).

The aura typically involves a generalized decrease in sensory threshold (Adams et al., 1980, Kroner, 1983), and usually lasts 15 to 30 minutes (Atkinson & Appenzeller, 1982). The most common neurological symptoms associated with the aura are visual disturbances. Such visual disturbances may involve scintillating scotoma, flickering lights, blurred vision, spots, colors, and even blindness, or sudden homonymous hemianopia (Fisher, 1980). Other sensory disturbances, often associated with the migraine, include feelings of numbness, which gradually spread over the face and down to the limbs (Atkinson & Appenzeller, 1982), blushing, nausea, chills, and other sensations mediated by the autonomic nervous system (Kroner, 1983).

The prodrome and aura are usually, though not always, followed by extreme head pain. The pain of migraine is often present when the patient awakens in the morning, but may come on at any time of the day or night and may awaken the patient from his/her sleep (Atkinson & Appenzeller, 1982). The headache is usually gradual in onset, developing over several minutes to an hour or two, reaching a peak that persists for several hours or days, and then gradually subsiding over several hours (Atkinson & Appenzeller, 1982). During the attack, the migraine patient typically seeks rest in a dark, quiet place. Motion generally makes the pain worse, and the patient is often hypersensitive to light, noise, and sometimes, odors.

During the attack, pain is usually unilateral (Atkinson & Appenzeller, 1982; Kroner, 1983), but may also be experienced over the entire head (Kroner, 1983; Olesen, 1978; Selby & Lance, 1960). In approximately one-half of those with

unilateral headaches, either side may be affected, and this may vary from episode to episode (Atkinson & Alexander, 1982). For many, however, headaches usually consistently occur on the same side of the head (Selby & Lance, 1960).

After the headache has disappeared, many migraineurs feel 'washed out', 'drained', or, alternatively may feel exceptionally well, even 'high' (Blau, 1987). Such symptoms may last anywhere from one hour to four days (Blau, 1982), and have been associated with receding attacks back to normality, as well as with the use of medication (Blau, 1987).

The experience of migraine is highly individualized, with few people perceiving the same sensations and/or pain. Such variability has made it difficult to determine the precise mechanisms involved in migraines.

Etiology of Migraines

Several factors, both physiological and psychological, may contribute to the etiology of migraine.

Physiological Mechanisms in Migraines

While some physiologically oriented researchers have advocated a neural basis to migraines (e.g. Pearce, 1969; 1987), many others prefer to focus on the vascular mechanisms in the etiology of migraines.

The vascular theory of migraine dates back to the 17th century, when Thomas Willis (1684) speculated that headaches may be caused by dilatation of the blood vessels within the head. In 1872, Latham theorized that visual auras are due to a defective supply of blood to one side of the brain due to a contraction of cerebral arteries. In the 20th century, Rodella (1931) explained migraine as a crisis of the

cerebral vessels producing what he called 'angina cerebri'. He stated that three phases occur in the production of the attack. The first phase involves vascular spasms of the arteries, followed by vascular dilatation, and culminating in a build-up of discharged fluid in the pia matter. The vascular mechanisms in migraines were later clarified by Wolff and his colleagues.

Graham and Wolff (1938) reported increases in the amplitude of pulsations of the temporal and occipital arteries during migraine headaches. Schumacher and Wolff (1941) subsequently reported that migraine auras are due to cerebral vasoconstriction of the vessels within the territories of both internal and external carotid arteries. Wolff and his colleagues later concluded that the pain of migraine arose from the combined effects of dilatation of large cephalic arteries plus the action of substances that lowered pain thresholds, which accumulated in blood vessel walls and perivascular tissues, thereby lowering pain threshold (Wolff, Tunis, & Goodell, 1953). Then, in 1953, Tunis and Wolff demonstrated significantly greater variability in the contractile properties of cranial vessels in migraine sufferers compared with normal controls, even when headache free. Such variability has been attributed to abnormal autonomic nervous system responses (Amery, 1982; Appenzeller, 1978; Gotoh, Komatsumoto, Araki, & Gomi, 1984; Havanka-Kanniainen, Tolone, & Mylyla, 1986; Johnson, 1978; Nielsen & Owman, 1971; Nielsen, Owman, & Sporrang, 1971; Pashier, Van Der Helm-Hylkema, & Orlebeke, 1984).

The autonomic nervous system. Ellertson & Hammerborg (1982), Johnson (1978), and Sicuteri (1967) have maintained that the classical migraine may be the

result of exaggerated and poorly habituated cardiovascular responses to stimuli which activate the autonomic nervous system. Such sympathetic overactivity typically leads to vasoconstriction of the arteries, and decreased blood flow, and is experienced, at the beginning of the migraine, as the aura (Johnson, 1978). If the vasoconstriction is not modified by some treatment, then the blood vessels will ultimately dilate, possibly assisted by the presence of vasodilator metabolites accumulated during the constrictor phase (Sicuteri, 1967), and also perhaps by a reflex inhibition of the constrictor drive. Accompanying this vasodilation is a decrease in sympathetic functioning and an increase in blood flow (Havanka-Kanniainen, 1986; Johnson, 1978). This increased blood flow in vessels in turn may lead to an increase in pulsation of cranial arteries, which appears to be the chief cause of pain (Rall & Schleifer, 1980).

Evidence for the role of the autonomic nervous system in classical migraines may be found in studies which have exposed migraineurs to various stressors, and studies which have examined monoamine and neuropeptide activity during migraines.

Migraine subjects appear to be sensitive to stressors and environmental changes (C. D. Anderson, Stoyva, & Vaughn, 1982; Anthony, 1981; Drummond, 1982). This sensitivity is particularly evident in studies which have found that, once exposed to stressors, migraineurs are slower to recover than controls (C. D. Anderson et al., 1982; Anthony, 1981; Drummond, 1982). For example, Anthony (1981) recorded physiological reactions of migraine subjects, and found that migraine subjects continued to show increases in diastolic blood pressure after the termination of a mental arithmetic stressor.

The role of the autonomic nervous system in migraine is further indicated in studies which have explored serotonin and norepinephrine levels in migraineurs (Adams et al, 1980; Akil & Liebesking, 1975; Anthony, 1972; Anthony, Hinterberger, & Lance, 1967; 1969; Curran, Hinterberger, & Lance, 1967; Curzon, Barrie, & Wilkinson, 1969; Fog-Moller, Genefke, & Bryndum, 1978; Hsu et al., 1978; Lance, 1987; Poloni, Nappi, Arrigo, & Savoldi, 1974; Sicuteri, Anselmi, & Del Bene, 1975; Tenen, 1968). These monoamines produce constriction of the arteries, increase blood pressure, heart rate, and have other relevant autonomic functions (Weiner, 1980) which have been implicated in the early stages of the migraine attack (Johnson, 1978).

Serotonin, a naturally-occurring derivative of tryptophan, is found in platelets and in cells of the brain, and acts as a potent vasoconstrictor (Carlson, 1977). A release of platelet serotonin results in the vasoconstriction associated with the prodromal and aura stages of migraine (Adams et al, 1980; Anthony et al. 1967; 1969). A consequent depletion of platelet serotonin results in the extracranial vasodilatation characteristic of migraines and is experienced as the headache phase (Anthony et al., 1967; 1969; Curran et al., 1967). Some studies have shown that headaches may be averted by increasing plasma levels of serotonin (Anthony, 1972), or a serotonin precursor, L-tryptophan (Poloni et al., 1974), at the onset of the migraine attack. Others have found that reserpine, a drug which depletes stores of serotonin, may induce headaches in migraineurs (Curzon et al., 1969).

Norepinephrine has also been implicated in the pathophysiology of migraine. This adrenergic hormone is synthesized naturally by the adrenal medulla and acts

to increase blood pressure by vasoconstriction (Carlson, 1977). Studies have found that, during a migraine attack, levels of norepinephrine parallel those of serotonin (Fog-Moller et al., 1978; Hsu et al., 1978; Lance, 1987). For example, levels of norepinephrine tend to be higher in the hours preceding the headache, e.g. during the aura and prodrome (Fog-Moller et al., 1978; Hsu et al., 1978; Lance, 1987), progressively decrease as the headache intensifies, and reach their lowest values between one and two hours before the headache peak (Fog-Moller et al., 1978).

Other studies on the pathophysiology of migraines have implicated neuropeptides in the etiology of migraine (Cuello, Jessell, Kanazawa, & Iversen, 1977; Groves & Schlesinger, 1982; Ray & Wolff, 1940). Neuropeptides, made up of short chains of amino acids (Groves & Schlesinger, 1982), have been proposed as neurotransmitters in the central and peripheral nervous system (Snyder, 1980). Specific peptides have been implicated in the perception of pain.

The most likely candidate in pain perception is Substance P, which is widely distributed in neurons of the brain and spinal cord and acts to stimulate vasodilation and contraction of smooth muscles (Ray & Wolff, 1940). Substance P has been implicated as a possible neurotransmitter in sensory nerve fibers that carry pain information into the spinal cord (Cuello et al., 1977; Groves & Schlesinger, 1982; Ray & Wolff, 1940). Although the precise manner in which Substance P and monoaminergic pathways are effective in pain control are unknown, it appears that the depletion of monoamines, and subsequent vasodilation, could open the pain control gates, releasing Substance P into the system, and lead to the perception of pain (Sicuteri, 1967).

Other peptides appear to exert an inhibitory influence in the pain experience. These, labelled endogenous opiates or endorphins, are peptides with between 15 and 30 amino acids, released by the pituitary gland and acting on the central and peripheral nervous systems to reduce pain (Pomernats & Chiu, 1976). Several different types of endorphins have been isolated in the brain and pituitary gland (Hughes, 1975; Terenius & Wahlstrom, 1975). Of these, beta-endorphins appear to be the most potent.

Many studies have explored the role of beta-endorphins in modulating pain perception (Clement-Jones, Tomlin, Rees, McLoughlin, Besser, & Wen, 1980; Akil, Madden, Patrick, & Barchas, 1976; Mayer, Price, & Raffi, 1975; Mayer, Price & Raffi, 1977; Pomerantz & Chiu, 1976). In relation to migraines, most have found that during headache free periods, central nervous system beta-endorphin levels in migraine sufferers are far below the lower limit of the normal range (Agnoli, De Marinis, Martucci, Feliciani, & Cerbo, 1987; D.G. Anderson, Jamieson, & Man, 1974; Fettes, Gawel, Kuzniak, & Edmeads, 1985; Genazzani et al., 1984; Leppaluoto, Havanka-Kanniainen, Myllyla, & Hokkanen, 1983; Sicuteri, 1978; Sicuteri, 1982). Others, however, have not substantiated these differences (Bach, Jensen, & Bleguad, 1985). Despite the lack of consensus regarding the level of beta-endorphin during headache-free intervals, a pattern does appear to emerge during the migraine episode.

During a migraine episode endorphins, like the monoamines, appear to be reactive to stimuli which act on the sympathetic nervous system (Sicuteri, Spillantini, Panconesi, & Cangi, 1984). Accordingly, levels of beta-endorphin tend to be

elevated during the early phases of the migraine (Sicuteri, Boccun, Fanciullacci, & Gatto, 1983), and like the monoamines, they diminish as the migraine progresses, reaching a low during the headache phase (Sicuteri et al., 1984).

Naloxone, an endogenous opiate antagonist, has been found to reverse migraine aura and ameliorate the accompanying headache (Sicuteri et al., 1983). Others have found that acupuncture, a beta-endorphin stimulant, may relieve headaches (D.G. Anderson et al., 1974; Hansen & Hansen, 1985; Loh, Nathan, Scott, & Zilkha, 1984). Still others have concluded that beta-endorphin levels are inversely correlated with the amount of pain experienced, as judged by the Headache Diary (Leppaluoto et al., 1983; Genazzanni et al., 1984).

Taken together, the data suggest that migraine sufferers may have some autonomic nervous system imbalance, with increased sympathetic activity signaling the beginning of the migraine attack, and some decrease in activity early in the attack. Tunis and Wolff (1953) have hypothesized that these changes may be induced by environmental and psychic factors.

Psychological Mechanisms in Migraines

Various personality traits have been implicated in the etiology of migraines. For example, migraineurs are reported to be ambitious and preoccupied with success, perfectionistic, compulsive and orderly, rigid and inflexible, resentful and unable to express aggressive feelings in a constructive manner (Alvarez, 1974; it is Friedman, von Storch & Merritt, 1954; Harrison, 1975; Kudrow, 1974; Kudrow & Sutkus, 1979; Lachar, 1954; Rappaport, McAnulty, & Brantley, 1988; Wolff, 1937). Most of these conclusions are based on uncontrolled clinical interviews. Others

have not found evidence to support these personality traits (Andrasik, Blanchard, Arena, Teders, Teevan, & Rodichok, 1982; Bakal, 1975; Harrison, 1975; Sovak, Kunzel, Sternbach, & Dalessio, 1981).

To limit the etiology of migraine to either physiological or psychological factors would be premature. The migraine is most likely a response to some biological weakness, probably involving the autonomic nervous system, which manifests itself when the individual experiences, among other things, stimuli which trigger strong emotions.

Precipitants of Migraines

Some stimuli which have been implicated as precipitants of migraines include oral contraceptives and the menstrual cycle, (Carroll, 1971; Kudrow, 1975; Waters & O'Connor, 1971), glare (Selby & Lance, 1960), foods such as fats, fried foods, cheese, chocolates, and oranges (Amery & Vandenberg, 1987; Atkinson & Appenzeller, 1982; Selby & Lance, 1960; Vandenberg, Amery, & Waelkens, 1987), and stress (Amery & Vandenberg, 1987; Henryk-Gutt & Rees, 1973; Selby & Lance, 1960; Vandenberg et al., 1987). In fact, some 50% of migraineurs have reported that their migraine episodes are triggered by stress (Amery & Vandenberg, 1987; Henryk-Gutt & Rees, 1973).

The Organism under Stress

"The word "stress"...means different things to different people so that defining it is extremely difficult...the air traffic controller who knows that a moment of inattention may mean death to hundreds of people, the athlete who wants to win a race...The problems they face are totally different, but medical

research has shown that in many respects, the body responds in a stereotyped manner, with identical biochemical changes, essentially meant to cope with any type of increased demand upon the human machinery" (Selye, 1974, pp. 25-26).

Thus, stress may be defined as that which activates the sympathetic nervous system and triggers the stress response, whether these stimuli be environmental, psychological, alimentary, or biochemical.

An organism which perceives stress reacts with a mobilization of all bodily resources. Metabolic rich blood is shunted away from the vegetative organs and toward the organs involved in the response, e.g. heart, muscles, etc. Physiological responses to stress include elevated heart rate and blood pressure (Pittner & Houston, 1980), increases in monoamines and cortisol levels (Frankenhauser, 1980; Glass et al., 1980; Mason, 1968), and increases in endorphin levels (Grossman, 1983; 1984; Grossman, Gaillard, McCartney, Rees, & Besser, 1982). Such a reaction is equivalent to what Selye (1950; 1976) recognized as the alerting response of the General Adaptation Syndrome. Under conditions which are severe, there may be a reversal in the direction of the stress responses (Teichner, 1968). Such a reversal is said to occur when the stress reactions reach a critical or threshold and trigger a signal which in effect produces reactions that are completely opposite to those initial responses. For example, the parasympathetic nervous system takes over, slowing the heart and other autonomic organs, and increasing cerebral dilation and blood flow in the brain (Teichner, 1968). The vigour of the compensatory reaction in the new direction will be dependent upon the level of activation. The higher the initial

reaction, the more dramatic will be the reversed compensatory reaction (Teichner, 1968).

This series of cerebrovascular responses parallels the pattern of response exhibited during the classic migraine. Indeed, there is a tendency for the headache phase of the classic migraine to appear not at the peak of stress, but during a period of relaxation immediately following stress (Henryk-Gutt & Rees, 1973).

The Impact of Migraines

Few studies have examined the consequences of suffering with migraines. Migraines may not only affect one's physical well being, but may also have an impact on one's psychological well being, as well as on social, interpersonal, and occupational functionings.

Physical impact. Physically, any illness, including migraine may be incapacitating (Hamburg, Hamburg, & DeGoza, 1953; Hobfoll, 1988; Moos & Tsu, 1977; Visotsky, Hamburg, Goss, & Lebovitz, 1961). Certainly, the characteristics of the migraine appear to describe a condition in which any sensory stimulus is intolerable. Indeed, Bond (1980) found that, during migraine episodes, most pain sufferers experience a reduction in tolerance for external stimuli such as lights or sound.

Impact on affect and control. Psychologically, migraineurs tend to be more emotional (Henryk-Gutt & Rees, 1973), and more depressed than controls (Andrasik et al., 1982; Alvarez, 1974; Friedman et al., 1954; Harrison, 1975; Kudrow, 1974; Kudrow & Sutkus, 1979; Lachar, 1954; Merikangas, Risch, Merikangas, Weissman, & Kidd, 1988; Wolff, 1937). Several explanations have been offered for the repeated

high incidence of depression in migraine sufferers (Couch, Ziegler, & Hassanein, 1975; Couch & Hassanein, 1979; Friedman, 1982; Merikangas, et al., 1988; Mohns, 1980). For example, depression may be due to the chronic incapacitation, that often accompany migraines, and the lack of control that one has over migraines. Such lack of control may threaten one's view of oneself as a healthy, normal individual, and may underscore one's vulnerability to illness and other problems (Cohen & Lazarus, 1979). Alternately, depression and migraine may be linked by virtue of shared neurochemical systems, such as low levels of catecholamines (Couch et al., 1975; Couch & Hassanein, 1979; Merikangas et al., 1988; Mohns, 1980).

Some have attempted to establish directionality in the relationship between migraine and psychological distress (Blanchard, Andrasik, Applebaum, Evans, Myers, & Barron, 1986; Blanchard et al., 1982; Cox & Thomas, 1981; Jurish, Blanchard, Andrasik, Teders, Neff, & Arena, 1983; Sovak et al., 1981; Teders, Blanchard, Andrasik, Jurish, Neff, & Arena, 1984). These studies have noted a significant drop in trait anxiety, depression, and symptomatology in migraine sufferers treated with biofeedback and relaxation training (Blanchard, et al., 1986; Blanchard et al., 1982; Cox & Thomas, 1981; Jurish, et al., 1983; Sovak et al., 1981; Teders et al., 1984). For example, Sovak et al. (1981) found significant reductions in MMPI scales designed to assess hypochondriacal and hysterical features, depression, and anxiety, for patients whose migraines were successfully treated with biofeedback. The researchers concluded that the depression which frequently accompanies the illness may be alleviated with a concurrent reduction in pain, and therefore may be secondary to the pain.

Interpersonal impact. Migraines may also have an impact on social and interpersonal relationships (Bond, 1980; Craig, 1980; Philips & Jahanshahi, 1985). Philips and Jahanshahi (1985) studied chronic headache sufferers and found that, as the pain persisted, the patients experienced an increasing disruption of social relationships and communication with others, leading to increasingly restricted ways of life. Craig (1980) has even suggested that headache patients often experience criticism and social isolation. This may be related to a refusal by the patients' environment and the medical profession to recognize them as sick (Bond, 1980). Because of this social devaluation of their state, patients cannot expect the degree of consideration that is usually evoked by the expression of clearly somatogenic pain (Bond, 1980).

Functional Impact. The few studies which have explored the functional impact of migraine have concluded that the head pain limits ones ability to enjoy free time or function properly at work (Beckstead, 1989; Bond, 1980; Levor, Cohen, Naliboff, & McArthur, 1986; Zenz, 1983). For example, Levor et al. (1986), who studied 33 headache patients, found that physical activity declined over the four days prior to, and including, the day of the migraine. Similarly, Beckstead's (1988) subjects showed a reduced range of socially-reinforcing activities as a result of having chronic headaches.

Treatment of Migraines

Given that migraines may lead to physical, psychological, as well as interpersonal distress, an intervention program is warranted which would address this interaction.

There have been numerous reports on the effects of a variety of medical and psychological treatments for migraines. According to a survey by DeLozier and Gagnon (1975), drug therapy is the most frequent method of treatment for migraines, with 72.8% of migraine sufferers receiving such intervention. Psychologically oriented therapies, in contrast, were prescribed for a mere 23.1% of migraineurs.

Most physicians prescribe drugs which are effective in relieving pain only once the migraine has begun (Dalessio, 1980). For example, Ergotamine, a vasoconstrictor which decreases blood flow and pulsations of cranial arteries (Rall & Schleifer, 1980), is effective only once the headache phase is activated (Ziegler, 1987). However, Ergotamine is ineffective if the pain has achieved a certain level (Ziegler, 1987).

Other drugs currently employed in treating an acute migraine attack are Dihydroergotamine (Raskin & Raskin, 1984), Isometheptene Naveate (Yuill, Swinburn, & Liversedge, 1972), antiemetics, such as Metaclopramide (Tfelt-Hansen, Olesen, & Aebelholt-Krabbe, 1980), analgesics, such as Aspirin, Acetaminophen, Ibuprophen (Tfelt-Hansen & Olesen, 1980), Dilaudid, Meperidine, or Morphine (Ziegler, 1987), and sedatives, such as Diazepam (Ziegler, 1987).

Some migraineurs, and their physicians, prefer to use medication aimed at preventing migraines. Beta-adrenergic blocking drugs are a common form of preventive treatment (Ziegler, 1987). Beta-blocking drugs which have been found to be effective include Atenolol (Forssman, Linblad, & Zbornikova, 1983; Stensrud & Staastad, 1980), Metoprolol (Andersson et al., 1983; Kangasniemi & Hedman,

1984), Nadolol (Ryan, Ryan, & Sudilowsky, 1983), Propranolol (Featherstone, 1983; Malvea, Gwon, & Graham, 1973; Weber & Reinmuth, 1972; Wideroe & Vigander, 1974), and Timolol (Standness, 1982). The anti-migraine effects of beta-blockers appear to persist for some time after drug withdrawal (Diamond, Kudrow, Stevens, & Shapiro, 1982). The mechanisms by which beta-blockers work are not yet understood, however, it has been speculated that they exert their effect by moderating autonomic nervous system reactions, e.g. lowering resting heart rate (Edvinsson, 1984), thus preventing exaggerated cranial vasoconstriction and subsequent vasodilation (Diamond & Dalessio, 1978).

Other drugs which act as prophylactics to migraines include alpha-adrenergic antagonists, such as Methysergide (Fanciullacci, Boccuni, Pietrini, & Gatto, 1984; Douglas, 1980; Rall & Schleifer, 1980), antidepressants, such as Amitriptyline (Taub, 1973), anti-inflammatory drugs, such as Naproxen (Ziegler & Ellis, 1985), anticonvulsant drugs, such as Phenobarbital (Ziegler, 1987), and calcium-channel blockers, such as Flunarizine (Fanciullacci et al., 1984; Godfraind & Dieu, 1981; Van Nueten, 1978) (see Ziegler, 1987 for review).

Although drugs are the most common form of therapy, they are usually directed at managing the disorder, controlling pain, and aborting the attack. As such, they fail to address psychological issues in migraines and often meet with limited success (Adams et al, 1980; Cutler, Aldredge, Fuller, & Siller, 1976; Fanchamps, 1975). In addition, the side effects of certain drugs, (Lucas & Falkowski, 1973), and their potential addictive quality (Worz, 1983), have often precluded their use. In view of this, many have relied upon psychological

interventions, which reduce the need for narcotics, analgesics, or vasoactive agents.

Psychological treatments for migraine are directed at achieving such divergent goals as insight, relinquishing control of body and symptoms, modification of pain perception, self-relaxation, self-regulation of temporal artery vasoconstriction, desensitization to stressful environmental stimuli, development of assertive behaviour, and modification of the environment (Adams, et al., 1980). Some reports have found psychotherapy to be effective in reducing migraine problems (Brenner, Friedman, & Carter, 1949). Others have found behaviour modification techniques, aimed at teaching patients to deal with stress using altered behaviours, to be effective using a wide range of techniques (Ziegler, 1987), including positive thinking (Mitchell & White, 1976), assertiveness training (Mitch, McGrady, & Iannone, 1976), desensitization (Mitchell & White, 1977), and relaxation training (Blanchard, Theobald, Williamson, Silver, & Brown, 1978). Biofeedback has proven to be a promising treatment directed at modifying both the peripheral pain mechanisms, and the assumed overactive autonomic nervous system of migraine sufferers (Adams, Webster, Graham, & Feuerstein, 1976; Blanchard et al., 1978; Feuerstein & Adams, 1977; Feuerstein, Adams, & Beiman, 1976; Medina, Diamond, & Franklin, 1976; Sturgis, Tollison, & Adams, 1978; Wickramaskera, 1973).

In a series of three studies, Blanchard and his colleagues (Blanchard, et al., 1982; Jurish et al., 1983; Teders et al., 1984) found that chronic headache patients showed significant improvements in terms of symptomatology, following biofeedback. They further found that this population is likely to show some improvement in

psychological functioning as a result of taking part in nonpharmacologic treatment. Such programs are based on the assumption that migraines are mainly an autonomic nervous system disorder, which can be triggered by a combination of psychophysical, neurochemical, environmental, cognitive, and affective factors (Wittchen, 1983).

Finally, some pre-experimental data suggest that brief, vigorous exercise, early in the migraine attack, may control headaches (Atkinson, 1977; Ekblom & Lindahl, 1970).

Aerobic Exercise

Definition of Aerobic Exercise

Aerobic exercises are those that require the muscles to have oxygen to burn the energy required to make them work. Oxygen is carried into the lungs with each breath of air and transported in the blood to the working muscles where it is used to drive the muscles. Carbon dioxide, the by-product of the oxygen used in combustion, is returned by the bloodstream and expelled during exhalation. Aerobic fitness, then, is a measure of the combined efficiency of the lungs, heart, bloodstream, and local muscles in getting the oxygen to the muscles and putting them to work (CASS & Fitness Canada, 1986). Aerobic activities are ones which can be continued for long periods, and utilize large quantities of oxygen, e.g. jogging, swimming, skipping, dancing (American College of Sports Medicine, (ACSM) 1990; Cooper, 1968).

Improvement in cardiovascular conditioning is directly related to frequency (ACSM, 1986; Atomi, Ito, Iwasaki, & Miyashita, 1978), intensity and duration of training (ACSM, 1986). Depending upon the quantity and quality of training,

improvement in cardiovascular conditioning typically ranges from 5 to 30% (Barry, Daly, & Pruett, 1966; Davis & Knibbs, 1971; Gaesser & Rich, 1984; Gossard, Haskell, & Taylor, 1986; Shephard, 1965; Shephard, 1977; Wright, Clarke, Niinimaa, & Shephard, 1976) with most of this improvement taking place in the first three to four weeks of exercise (Fried & Shephard, 1969; Hickson, 1981). According to ACSM (1990), the quantity and quality of training required to develop and maintain cardiovascular fitness suggest that one must exercise a minimum of twenty minutes, three days a week, at at least 50% of maximum capacity.

As quickly as aerobic capacity may be improved, however, it may be lost. A significant reduction in cardiovascular fitness occurs after two weeks of detraining (Coyle, Martin, & Bloomfield, 1985; Coyle, Martin, Sinacore, Joyner, Hagberg, & Holloszy, 1984). Inactivity for three weeks may lead to a loss of up to 17% of cardiovascular conditioning (Coyle et al., 1985; Coyle et al., 1984; Taylor, Henschel, Brosek, & Keys, 1949), while a loss of 50% of initial improvement has been shown after four to twelve weeks of detraining (Fringer & Stull, 1974; Kendrick, Pollock, Hickman, & Miller, 1971).

Benefits of Aerobic Exercise

Aerobic exercise has been associated with an overall sense of both physical and psychological well-being. In particular, aerobic fitness has been associated with stress reduction, feelings of control and mastery, mood elevation, enhanced interpersonal relationships, and increased vigor.

Exercise and stress. Aerobically fit people appear to develop a physiological tolerance for stress (Bahrke & Morgan, 1978; D. R. Brown, 1986; Gal & Lazarus,

1973; Heaps, 1978; Hollander & Seraganian, 1984; Keller & Seraganian, 1984; Lightman & Poser, 1983; Morgan, 1979; Morgan & Pollack, 1976; Sinyor, Schwartz, Peronnet, Brisson, & Seraganian, 1983). This statement follows from the observation that, physiologically, exercise is a form of stress, and thus may enable the individual's biochemical system to adapt to increases in monoamines and endorphins, and ultimately exhibit less reactivity to stress (Heaps, 1978; Ledwidge, 1980).

Exercise has been found to effect activity in serotonergic neurons (Barchas & Freedman, 1963; B. S. Brown, Payne, Kim, Moore, Krebs, & Martin, 1979). For example, Barchas and Freedman (1963) discovered that rats who ran on a treadmill for three hours had a 10% increase in brain serotonin. Similar results were found in studies using rats who ran on a treadmill for a more realistic thirty minutes (B. S. Brown et al., 1979).

Several experiments have demonstrated that exercise may also enhance synaptic transmission of catecholamines (Barchas & Freedman, 1963; Bowers, Heninger, & Gerbode, 1969; B. S. Brown et al., 1979; R. S. Brown, Ramirez, & Taub, 1978; Ebert, Post, & Goodwin, 1972; Fotherby, Ashcroft, Affleck, & Forrest, 1963; Gordon, Spector, Sjoerdsma, & Udenfriend, 1966). For example, Gordon et al., (1966) injected rats with amine synthesis inhibitors and then exercised them in a large motor driven rotation drum for one hour. The result was a marked depletion of norepinephrine. These experimenters then injected rats with tyrosine-C, a catecholamine precursor. Those rats who exercised demonstrated an increased conversion of tyrosine-C to brain norepinephrine. Thus, the experimenters concluded that exercise enhances the synaptic release of brain norepinephrine.

In addition, plasma levels of B-endorphins have universally been reported to increase with aerobic exercise (Colt, Wardlaw, & Frantz, 1981; Greist, Klein, Eischens, Faris, Goffman, & Morgan, 1978). Moreover, beta-endorphin changes appear to vary according to the intensity of the exercise (Farrell, 1985).

Since aerobic exercise appears to increase sympathetic nervous system functioning, it would follow, then, that exercise may result in aura and prodrome symptoms. However, such is not necessarily the case. The more fit an individual is, the lower will be his/her physiological response to the stress, including exercise.

Aerobically fit individuals have lower resting heart rates, as well as reduced heart rates at any given work load (Frick, 1968; Frick, Elobvainio, & Somer, 1967). This decrease in heart rate during submaximal exercise is similar to what is experienced when using beta-blockers (Masironi & Denolin, 1985). This suggests that physical training can exert its influence by increasing one's threshold for sympathetic arousing stimuli, such that, at any given work load, the circulation of the monoamines in the blood of trained individuals is significantly less than that in nontrained people (Masironi & Denolin, 1985). In fact, compared to nontrained individuals, trained individuals seem to have a preponderance of parasympathetic activity during rest, and their bodies secrete lower levels of monoamines (Hartley, 1975; Leblanc, Boulay, Dulac, Jobin, LaBrie, & Rousseau-Mogneron, 1977; Masironi & Denolin, 1985). Their physiological reactions to moderate exercise are also less intense, demonstrating lower values of released monoamines, and less sympathetic reactivity (Hartley, 1975; Leblanc et al., 1977; Masironi & Denolin, 1985). Under intense exercises, fit and non fit individuals have equal levels of sympathetic

reactions (Hartley, 1975; Leblanc et al., 1977; Masironi & Denolin, 1985). This would suggest that moderate levels of exercise are optimal for improving cardiovascular fitness aimed at enhancing one's level of tolerance to stress. Finally, fit people experience a quicker return to normalization following arousal (Hartley, 1975; Leblanc et al., 1977; Masironi & Denolin, 1985). Such autonomic nervous system stability has been found to generalize beyond exercise to different types of stress, e.g. noise, light, cold (Simkin & Korobkou, 1967).

Compared to controls, aerobically-fit individuals respond to stress with lower heart rate increases, lower subjective levels of anxiety (Sinyor et al., 1983), and more rapid returns to baseline following stress (Clausen, 1977; Keller & Seraganian, 1984). For example, Keller and Seraganian (1984) discovered that subjects who were put through a fitness program not only improved their fitness level, but also demonstrated faster autonomic recovery, e.g. heart rate quickly returned to baseline levels, after a series of stressors (e.g. the Bolt Head Maze, the Stroop Color-word test, a digit span test, and others) were terminated.

Exercise and control/mastery. Many have concluded that improvements in fitness may lead to a personal sense of control and mastery, which may transfer over to other sphere's of one's life (Gal & Lazarus, 1975; Hughes, 1984; Simon, McGowan, Eapstein, Kuper, & Robertson, 1985). Control and mastery have been offered as potential cognitive explanations for improvements in affect often noted with exercise.

Exercise and affect. Exercise has consistently been linked with improvements in mood. Indeed, aerobic activity has been found to be effective in reducing

depression in both normal individuals (D. R. Brown, 1986; Ledwidge, 1980; Morgan, 1981; Morgan & Pollack, 1986; Trugillo, 1983), and clinical populations (Greist, Klein, Eichens, Faris, & German, 1979). For example, Greist et al. (1979) found running to be at least as effective as psychotherapy in the treatment of individuals with moderate depression.

Both psychological and physiological factors have been offered as explanations for the therapeutic benefit of exercise on depression. Psychologically, running may not only enhance feelings of control and mastery, but has also been associated with improved self-concept (Layman, 1974). Some physiologically oriented theorists have focused on the roles of norepinephrine and serotonin, which have been found to be lacking in depressed individuals and are released during exercise (Barchas & Freedman, 1963; Bowers et al., 1969; R. S. Brown et al., 1978; Ebert et al., 1972; Fotherby et al., 1963; Gordon et al., 1966), while others have implicated the release of endorphins to euphoric moods (Hughes, 1984; Morgan, 1985).

Exercise and interpersonal relationships. Aerobic fitness has also been associated with sexual well being and improvements in intimate relationships (Harris & Gurin, 1985; Harris & Kagan, 1985). Regular participation in exercise results in marked physical changes, including firmer muscles and reduced body fat. Such changes not only generate improvements in self-concept and self-esteem, but they may also alter the perception of others (Greenberg & Pargman, 1989). In fact, according to White (as told in Greenberg & Pargman, 1989), aerobically fit individuals are sexier, experience more sexual desire, arousal, and have more satisfying interpersonal relationships.

Exercise and functional activities. Finally, exercise has been associated with improved work performance (Donoghue, 1977), decreased fatigue (Astrand, 1977; Baekeland, 1970; Baekeland & Lasky, 1966), and overall feelings of vigor (Blumenthal, Williams, Terri, Needels, & Wallace, 1982). Such benefits have been attributed to improvements in sleep, in particular to an increase in slow wave sleep after exercise (Shapiro, Bortz, & Mitchell, 1981). Slow wave sleep has been linked to the release of rejuvenating growth hormones (Baekeland & Lasky, 1966; Horne, 1981; Horne & Staff, 1983; Pawel, Sassin, & Weitzman, 1972), which may be associated with increases in energy and productivity.

In summary, aerobic exercise may increase one's tolerance for stimuli which trigger the sympathetic nervous system. In addition, aerobic activity may allow one to recover more quickly from such arousing stimuli. Psychologically, exercise has been found to restore feelings of control, to reduce depression, and improve intimate relationships. Finally, aerobic exercise has been associated with increased vigor, such that regular training should afford one added energy to engage more productively in work, home, and play.

Exercise and Migraines

Some studies have concluded that strenuous exercise may trigger a migraine episode (Dalessio, 1980; Rooke, 1968; Sacks, 1970; Thompson, 1987). However, these studies have lumped aerobic activity with other strenuous acts, such as lifting (Lambert & Brunet, 1985). Physiological changes associated with such exertion are initiated by a primary increase in oxygen consumption and an abrupt increase in oxygen demand (Braun & Klawans, 1986). Indeed, Lambert and Brunet (1985) have

found that a proper warm up prior to exercise may prevent exercise-related migraines. In addition, the pre-experimental data provided by Atkinson (1977), and Ekblom and Lindahl (1970) suggest a therapeutic relationship between aerobic exercise and migraines.

Given the known physiological and cognitive benefits of cardiovascular exercise and theories relating the autonomic nervous system to classical migraine, further investigation of the relationship between exercise and classical migraine is warranted.

The purpose of this study was to explore the effects of maintained aerobic fitness on classical migraines. Several hypotheses are proposed to this end.

First, since aerobic exercise is an acceptable form of cardiovascular conditioning, it may be hypothesized that those partaking in aerobic exercise will improve their cardiovascular fitness levels over the course of the study.

Second, classical migraines have been associated with sympathetic overreactivity to stimuli which initiate the stress response. In addition, migraineurs appear to be slower to recover, physiologically, from stress. Aerobic fitness has been associated with increased stress tolerance and allows for quicker recovery from the stress reaction. It may therefore be hypothesized that improvements in aerobic fitness may lead to fewer, less painful, and shorter migraines.

Finally, migraines have been found to impact on physical, psychological, interpersonal, and functional spheres of life, and aerobic fitness has been associated with various physical, psychological, interpersonal, and functional benefits. It may therefore be hypothesized that migraineurs enrolled in an aerobic exercise program

will experience a drop in perceived physical and psychological suffering, along with decreased affective distress, increased feelings of control, more satisfying interpersonal and social relationships, and an increase in activity level.

Method

Subjects

Subjects were recruited from the Ottawa community. The exercise program was advertised in local newspapers, hospitals, clinics, stores, and universities, as an aerobic exercise program for women with classical migraine headaches, to be administered and taught by a certified fitness instructor, and offered free of cost. Women were used exclusively in order to enhance the homogeneity of subject variables.

Thirty people responded initially to the advertisements. Of these, twenty six women met the criteria for classical migraine. Consistent with past research (Adams, et al., 1980; Duckro, Tait, & Margolis, 1989), these women were predominantly Caucasian, middle class, with some college education. Most of these women had tried various forms of medication over the years, and, while some had given up on, and no longer used any medications, most were currently using some form of analgesic, e.g. Fiorinol, Ergomar, Tylenol.¹

Material

Each subject completed a headache questionnaire (Appendix A), the Physical Activity Readiness Questionnaire (PAR-Q) (Appendix B) (Canadian Association of Sports Sciences (CASS) & Fitness Canada, 1986), a consent and release form for the fitness appraisal (Appendix C), a consent and release for the fitness classes (Appendix D), the Canadian Aerobic Fitness Test (CAFT) (Government of Canada,

¹Participants were requested to continue using those medications which they had been using prior to the study, and, if possible, to refrain from beginning a new medication program until the study concluded.

Fitness and Amateur Sport, 1976) (Appendix E), the West Haven-Yale Multidimensional Pain Inventory (MPI) (Appendix F) (Kerns, Turk, & Rudy, 1985), and a headache diary (Appendix G).

Headache Questionnaire. A modified version of the Questionnaire for One Headache (Campbell, 1988) (Appendix A) was administered to confirm the diagnosis of classical migraine. The criteria for classical migraine, which were presented in a checklist format, were based on a synthesis of the literature (e.g. Adams et al., 1980; Atkinson & Appenzeller, 1982; Blau, 1980; Campbell, 1988; Fisher, 1980; Kroner, 1983; Zhao, Tsay, Cheng, Wong, Li, Yao, Chang, & Schoenberg, 1988), and the recommended criteria as suggested by the Headache Classification Committee of the International Headache Society (1988).

In order to be included in the study, subjects were required to experience at least two migraines a month. This frequency requirement was necessary to allow for adequate representation of migraine episodes for the headache diary. In addition, during their migraines, subjects were required to experience one or more fully reversible aura symptoms, accompanied, usually, by a headache. A checklist of typical sensory and cognitive disturbances was provided for them, and included, among others, visual disturbances, hearing disturbances, gastrointestinal disturbances, mood disturbances, and thought disturbances.

In addition, the questionnaire allowed for the screening of those individuals for whom known physical traumas (e.g. head trauma) or illnesses (e.g. non-cephalic infection, disorders of cranium) account for their migraines.

The headache questionnaire also included a section on headache history. Questions were aimed at determining how old each participant was when she first experienced a migraine, and allowed the subject to express, in her own words, her migraine experience. In addition, the headache history asked the participant to list all medications, for migraines and other conditions, currently being used.

Physical Activity Readiness Questionnaire. The PAR-Q form (Appendix B) is a standardized form developed by the Canadian Association of Sports Sciences (CASS) and Fitness Canada (1986), designed to identify those individuals for whom certain physical activities might be inappropriate, i.e those with heart conditions, high blood pressure.

Consent Forms. The consent and release forms were designed to inform the participants of their responsibilities in the fitness appraisal and fitness classes. One of the consent and release forms was aimed at informing the subjects of their right to stop the fitness appraisal (Appendix C). The second consent and release form was devised to inform participants of their right to stop exercising during a fitness class, at any time, should they feel the need to do so (Appendix D).

Fitness Appraisal. The fitness appraisal was conducted by the experimenter, a certified Standardized Test of Fitness Appraiser. The appraisal consisted of the Canadian Aerobic Fitness Test (CAFT) (formerly called the Canadian Home Fitness Test -- CHFT) (Appendix E) (CASS & Fitness Canada, 1986). This test is designed to measure the overall functional capacity of the cardiovascular system, by predicting the greatest rate at which oxygen can be consumed during exercise (maximum oxygen uptake) from a submaximal test (CASS & Fitness Canada, 1986).

The CAFT procedure for predicting maximum oxygen uptake appears to provide comparable results to the volitional tests derived from a continuous multi-stage treadmill test with a direct measurement of oxygen consumption (Jette, 1979). For example, Jette (1979) found no significant differences between the mean CHFT predicted (34.8) and observed (34.6) maximum oxygen uptake values, and found these two tests to be significantly correlated ($r = .71, p < .001$). Shephard (1976) found the correlation between the CAFT and a direct test of oxygen consumption to be slightly higher ($r = .88, p < .001$).

The CAFT consists of a series of stepping sequences performed on double 20.3 cm steps (see Appendix E-1 for step dimensions) to a six-count musical rhythm, set by a CAFT cassette tape, with progressive increases in tempo. The CAFT is structured such that a participant begins by performing a three minute warm-up exercise at a cadence intensity of 65 to 70 per cent of the average aerobic power expected of a person ten years older. Instructions and time signals are given by the cassette tape as to when to start and stop exercising.

If a predetermined ceiling post-exercise heart rate (see Appendix E-2 for chart) is not attained or exceeded, the participant performs a further three minutes of exercise at 65-70 per cent of the average aerobic power expected for her age group. Heart rate is again measured at the end of the three minutes. If the participant does not attain or exceed the ceiling heart rate, a further three minutes of stepping is performed at an intensity equivalent to 65 to 70 per cent of the average aerobic power for a person ten years younger.

A standard sphygmomanometer (blood pressure cuff) and stethoscope were used to monitor blood pressure. Blood pressure was taken with the participant sitting in a chair with the left arm supported at heart level, feet resting flat on the floor. The blood pressure cuff was wrapped firmly and smoothly around the participants' left arms. The stethoscope was positioned below the cuff. The cuff was rapidly inflated to a level 20 to 30 mmHg above the radial palpatory pressure. The systolic pressure was determined by the first perception of the Korotkoff sound, and the diastolic pressure was determined when the tapping ceased. Once these pressures were attained, the cuff pressure was released at a rate of approximately two mm-per-second.

As part of the CAFT, the participant's weight, in kilograms, was recorded using a computerized scale. Weight is required to calculate maximum oxygen uptake. Instructions on calculating maximum oxygen uptake scores are presented in Appendix E-3, and the table for converting these scores to percentiles is presented in Appendix E-4.

West Haven-Yale Multidimensional Pain Inventory. The impact of the migraines on the participants' well being was assessed using the Pain Severity, Life Control, Affective Distress, Support, and General Activities scales of the West Haven-Yale Multidimensional Pain Inventory (MPI) (Appendix F) (Kerns, Turk, & Rudy, 1985). The MPI is a comprehensive pain assessment inventory that places an emphasis not only on the assessment of subjective distress, but also on the impact of the pain problem on various aspects of the individuals' lives.

The MPI subscales have been found elsewhere (Kerns et al., 1985) to have convergent, as well as discriminant validity. Convergent validity was assessed by correlating all items with hypothesized factors. Factor 1 was related to general affective distress, factor 2 represented support from significant others, factor 3 suggested a pain severity and interference dimension, while factor 4 represented an activity level dimension. Factor loadings were all significant, ranging from 0.43 to 0.96 (Kerns et al., 1985). The intercorrelations among the MPI scales range in magnitude from 0.00 to 0.58, all of which are lower than the reliability coefficients for the scales, thus indicating discriminant validity (Kerns et al., 1985).

The reliability of the MPI also appears to be satisfactory, with internal consistency scores ranging from 0.70 to 0.90 (Kerns et al., 1985). The stability of the test is also adequate, with test-retest coefficients ranging from 0.62 to 0.91 (Kerns et al., 1985).

In its final form, the MPI is a 52-item inventory, divided into three parts, each containing several scales or subscales. Five scales from the MPI were utilized in the present study.

The Pain Severity scale (PS) reflects subjects' reports of pain severity and suffering. It addresses such questions as "How much suffering do you experience because of your pain?". It was chosen as a global measure of the extent to which migraineurs experience both physical and psychological distress as a result of their migraines.

The Affective Distress scale (AD) measures emotional distress, including ratings of depressed moods, irritability, and tension. A sample question from this

scale would be "Rate your overall mood during the past week". It was chosen as an indicator of overall psychological functioning.

The Life Control scale (LC) incorporates perceived ability to solve problems and feelings of personal mastery and competence. It asks, among other things, "During the past week, how much do you feel that you've been able to deal with your problems?". It was chosen as an indicator of one's ability to deal with daily events and crises.

The Support scale (SUP) measures the amount of support and concern one receives from spouse, family, and significant others. It asks, for example, "How worried is your spouse (significant other) about you in relation to your pain?". It was selected as a measure of interpersonal support and satisfaction.

Finally, the General Activity (GA) scale was used to assess the subjects' report of their participation in four categories of common daily activities, including scales of household chores, outdoor work, activities away from home, and social activities. For example, it queries, "How often do you visit your friends?". It measures the extent to which migraines may interfere with daily activities.

The scoring of the MPI is accomplished by calculating a mean score for each of the scales, and deriving, from these, the MPI scale scores (PS, LC, AD, SUP, GA).

Headache Diary. Headache activity (Frequency, Intensity, and Duration) was monitored using a headache diary (Appendix G). The headache diary has been shown to be sensitive to changes in pain intensity (Kerns, Finn, & Haythornthwaite, 1988), and a valid indicator of treatment improvement, when compared to ratings

obtained from significant others of treated headache patients (Blanchard, Andrasik, Neff, Jurish, and O'Keefe, 1981). Others have found one form of diary monitoring, the Self-Monitored Pain Intensity (SMPI) (Turk, Meichenbaum, & Genest, 1983), to be correlated with both the Present Pain Intensity Index ($r = .51, p < .001$) and the Pain Rating Index ($r = 0.27, p < .01$) from the McGill Pain Questionnaire (Melzack, 1975) (Kerns et al., 1985; Kerns et al., 1988).

Self-monitoring diary procedures are also reliable measures of pain. Some have found these procedures to be stable over a one week period (Follick, Ahern, & Laser-Wolston, 1984) while others have demonstrated stability over a two week time frame (Kerns et al., 1988). For example, Kerns et al. (1988) found that the Pearson product-moment correlations for pain intensity, as measured by the SMPI, were significant across two weeks ($r = .93, p < .0001$).

The headache diary, in this study, consisted of a record (on standard record sheets provided by the researcher) of migraine activity, which included the presence or absence of migraine on any given day, the intensity of the pain (on a six point scale), and the duration of the pain (in hours and minutes). The pain intensity measure was presented on a 5-point scale similar to the scale developed by Melzack (1975) (1 = mild; 2 = discomforting; 3 = distressing; 4 = horrible; 5 = excruciating). In a previous study, Melzack and Torgerson (1971) had demonstrated that the word descriptors assigned to each of the numbers of the scale were approximately equally far apart, so that they represent equal scale intervals and thereby provide anchors for the specification of overall pain intensity.

Subjects were requested to complete the headache diary every day, for twelve weeks. The daily entries were then divided into four time frames. An initial four week period served as the baseline. A subsequent three week baseline served as a mid-treatment measure. The following three week period served as the posttest. Finally, another two week period was monitored as a follow-up measure.

The Frequency measure of the diary was scored by adding the total number of migraines experienced within a given time frame and dividing by the number of weeks in that time frame. Pain Intensity was assessed by adding up the intensity ratings, for a given time frame, and dividing by the number of migraines experienced during that time frame. The total number of hours and minutes an individual experienced migraines, for a given time frame, were summed and divided by the number of migraines experienced for that time frame to yield the Duration variable.

Procedure

Prospective classical migraine subjects were initially screened by telephone. Screening was intended to achieve a preliminary confirmation of classical migraine and health status. Those women who claimed to suffer from classic migraines and who stated that an exercise program would pose no apparent health risk, were invited to meet with the researcher. Subjects were asked to refrain from eating any foods and from drinking caffeine and smoking for two hours prior to the meeting. In addition, they were asked to refrain from alcohol and exercise for six hours prior to the meeting. Finally, prospective subjects were requested to bring along work-out attire.

At the initial meeting, prospective subjects began by completing the headache questionnaire, followed by the PAR-Q (CASS & Fitness Canada, 1986). Anyone answering "Yes" to one or more items on the PAR-Q (except where an answer of "Yes" to feeling faint was associated with migraines only) were thanked for their participation thus far and told that their engaging in a fitness program, at this time, may be a health risk. As no one presented with major health problems, they all proceeded to the next phase of the study, the fitness appraisal.

The researcher began by explaining the purpose of the fitness appraisal. Participants were told that the appraisal would focus on cardiovascular conditioning. As such, they would be requested to partake in a series of up to three stepping sequences to the cadence of the CAFT cassette, and that their heart rate would be taken prior to, and after, each three minute stepping sequence. In addition, their blood pressure would be taken prior to, and at the end of the fitness appraisal, to ensure that their health would not be jeopardized by the stepping test. Age and weight, they were told, were necessary to calculate their aerobic fitness level. Participants were then asked to sign a consent form, indicating that they understood the fitness appraisal process, and would take responsibility to stop the testing at any time, should the need arise.

The researcher began by measuring the participant's resting heart rate and blood pressure. After ensuring that heart rate and blood pressure were within the normal range, participants were weighed. A demonstration of the CAFT stepping sequence was then provided by the researcher, and all participants were encouraged to practice this sequence, first without the music, and then with the music. Upon

completion of the fitness test, post-exercise heart rate were taken, and participants were instructed to walk for thirty seconds. At the end of thirty seconds, their blood pressure was taken. Subjects were then instructed to relax for a couple of minutes. At the end of two minutes, their blood pressure was again taken, followed by their heart rate. These final measures of blood pressure, and heart rate, they were told, were to ensure that the cardiovascular system is indeed in a recovery mode.

Following the fitness appraisal, subjects were asked to complete the MPI. They were then introduced to the headache diary, which they were asked to monitor every night at bedtime, beginning the next day, for the next twelve weeks. These diaries were to be submitted to the researcher in four weeks, seven weeks, 10 weeks, and 12 weeks from the initial meeting. In addition, participants were asked to meet with the researcher in seven weeks, 10, and 12 weeks from the initial meeting, so that they might complete, once again, the MPI and undergo a fitness appraisal. Subjects were asked not to engage in any new activities until instructed. Before closing the initial meeting, participants were told that they would be contacted by telephone to be informed of the commencement date for their aerobic classes.

Following the initial meeting, and after ensuring that all subjects met the criteria for participation, subjects were randomly assigned, using a random number table, to an experimental, aerobic treatment group or a waiting-list control group. Subjects were contacted by telephone and told that they would either begin their aerobic classes immediately (the experimental group) or that, due to space limitations, they would begin their classes in 12 weeks from the initial meeting (waiting-list control group). It was further requested of the control group that they

not take up exercise before the classes begin, in order that all participants might start together.

Participants were required, on the first day of the aerobic classes, to complete a consent and release form to ensure that they understood their rights to stop exercising at any time, should they feel the need. Participants were reminded of the necessity to be consistent in their attendance, in order to experience improvements in fitness. However, they were encouraged not to work out while experiencing a migraine.²

The aerobic classes were administered and taught by a certified fitness instructor. In accordance with the recommendations of CASS and Fitness Canada (1986), fitness classes were held three times a week for 45 minutes each, with each subject instructed to work within her target zone of 70-85% of maximal aerobic capacity (see Appendix H for target heart rate chart). Classes focused on the development of aerobic capacity through low impact aerobic dancing and calisthenics. Included in each class was a 10 minute warm-up period, followed by a 25 minute aerobic work-out, and a ten minute cool down period. The aerobic program ran for six weeks, which is consistent with the length of time required to note significant changes in cardiovascular and autonomic functioning (Fried & Shephard, 1969; Hickson, 1981).

Three weeks into the aerobic program (seven weeks after the initial meeting), all subjects were asked to meet with the researcher, to submit their diaries, undergo

²Approximately 25 percent of the participants were unable to attend classes on a regular basis. Video-cassettes were therefore provided to them so that they could exercise at home.

a second fitness evaluation, using the CAFT, and to complete the MPI.

Following the six week treatment program, appraisals of fitness levels, for both the experimental and control groups, were again conducted, using the CAFT, headache diaries were collected and analyzed, and subjects were once again asked to complete the MPI.

Finally, aerobic fitness and migraine impact were reassessed two weeks after the conclusion of the exercise program, and final diaries were collected.

Following the final testing session, participants were thoroughly debriefed. The purpose of the study was explained to them, and they were told that the results would be provided to each of them as soon as they become available. They were each thanked for their participation, and arrangements were then made to provide aerobic fitness classes to the waiting-list control group.

Results

Descriptive Statistics

Twenty-five women participated in the study. Of these, four women, two in each of the treatment and control group, were excluded from the data analysis because they had not experienced any migraines during the baseline period, thereby precluding any comparison prior to, and after treatment. Also excluded from the analysis was one woman, in the control group, who experienced migraines every day, making her not only an outlier, but also placing into question her status as a classical migraine sufferer. According to Wilkinson (1987), "Classical migraine never occurs daily although a headache may last up to 48 hours" (p. 211). This participant most likely suffered from "status migrainosus", a term used to denote migraines when the headache does not completely disappear but continues unabated or in varying intensity for longer than three days (Couch and Diamond, 1983).

In the final analysis, then, were 20 women, 11 in the treatment group and 9 in the control group. Demographic characteristics pertinent to these women are presented in Table 1. A series of T-tests revealed no significant differences between groups on age, duration of pain, and fitness level. The age of the participants ranged from 19 to 50, with a mean age of 32.5 for the treatment group, and 32.2 for the control group. Overall, participants in the treatment group suffered their migraines for an average of 18.3 years while the control group suffered a slightly longer 18.9 years. Finally, both groups presented initially with average fitness levels. The treatment group's average fitness level fell at the 44.8 percentile in relation to the average female Canadian population. Control subjects were slightly less fit,

TABLE 1

Demographic Characteristics of Sample

Variable	Treatment Group	Control Group
Age	32.5	32.2
Years with Migraines	18.3	18.9
Initial Fitness Level	44.8	46.3

presenting initially with an average fitness level falling at the 46.3 percentile. The raw scores for each participant, on each dependent variable, Fitness, Frequency, Intensity, Duration, Pain Severity, Life Control, Affective Distress, Support, and General Activity, across each testing -- baseline (Time A), mid-treatment (Time B), post-treatment (Time C), and follow-up (Time D) -- are presented in Appendix I. Intercorrelations between variables are presented, for each testing period, in Appendix J. Means, standard deviations, and skewness and kurtosis coefficients are presented for each variable, per group and testing, in Appendix K.

Statistical Analysis

Repeated-measure analyses of variance (ANOVA) with a between factor were used to compare fitness levels, migraine activity, and migraine impact variables for subjects in the exercise and control group. Where the assumption of homogeneity of covariance was not met (Fitness and Support variables), the more conservative multivariate analysis of variance (MANOVA), using each repetition of the dependent variable as separate dependent variables, was used.

The use of multiple ANOVAs and MANOVAs are an acceptable alternative when dealing with small sample sizes that preclude the use of a doubly multivariate analysis of variance (Hand & Taylor, 1987; J. Tombaugh, personal communication, Aug. 13, 1990). An alpha level of .01 was deemed to be sufficiently conservative to control for the effects of multiple tests, while being liberal enough to detect differences given the small sample size (J. Tombaugh, personal communication, Aug. 13, 1990). Several measures were taken to ensure that the assumptions inherent in

repeated measures ANOVA were satisfied. These are presented in Appendix L.

Cardiovascular fitness. A MANOVA, using each repetition of the dependent variable as separate dependent variables, indicated a significant interaction effect of cardiovascular fitness (Pillais = .628, $p < .001$) (Table 2).

Turning to Figure 2, one can see that the two groups were relatively equivalent in terms of their fitness level, at Time A. Group differences, however, became apparent after just three weeks of fitness classes. As may be seen in Figure 2, the treatment group improved their cardiovascular fitness level by an average of 10.4 percentiles from time A to time B (from 44.8 to 55.2), with an additional increment of 4.7 percent at Time C (59.9), and slight drop at time D (57.9). The control group, on the other hand, remained relatively stable in their fitness levels across the four time periods (46.3, 46.0, 46.2, 45.8, respectively).

Further analysis, using the Roy-Bargman Stepdown F Test, revealed that most of the change in fitness level accounting for group differences occurred from Time A to B, $F(1,18) = 28.36$, $p < .000$. Such an improvement resulted in a significant T test for the treatment group, $t(10) = -4.66$, $p < .001$, while the change in the control group from Time A to B was not significant, $t(8) = .44$, $p = .674$. We may therefore conclude that the significant interaction effect between group and time for Fitness is due to significant improvements experienced by the treatment group in cardiovascular fitness, from Time A to Time B. Having established that the treatment was effective in improving fitness, we turn to an analysis of the effects of cardiovascular fitness on migraine activity and impact.

TABLE 2

Multivariate Analysis of Variance for Fitness Level

Source	SS	DF	MS	F
Between Ss				
Group	1387.53	1	1387.53	0.54
Error	46588.91	18	2588.27	
	Pillais	DF	Error DF	Approx. F
Within Ss				
Time	0.62092	3	16	8.74*
Group x Time	0.62823	3	16	9.01*
<u>Roy-Bargman Stepdown F Test</u>				
Time	Hyp MS	DF	Error DF	Stepdown
A vs B	460.91	1	18	28.36**
AB vs C	0.09	1	17	0.01
ABC vs D	2.81	1	16	0.7

* $p < .001$ ** $p < .000$ Note. Box's M = 30.9F (10, 1390) = 2.32, $p = .01$

Mauchly Sphericity Test = .28

Chi Square Approx. = 21.09, $p = .001$

Figure 1. Comparison of mean fitness level across group and time

Migraine activity variables. Three separate repeated ANOVAS with a between factor for each of the migraine activity variables, Frequency, Intensity, and Duration, while showing some improvements for the treatment group, failed to produce statistically significant effects.

The results of the variable Frequency are displayed in Table 3. As can be seen, no main effects for either group or time, and no interaction effects were found. Yet, as is presented in Figure 3, the treatment group did experience a steady decline in the frequency of their migraines over the course of the study. Thus, while they experienced an average of 1.3 migraines per week at Time A, this figure dropped to 1.0, 0.9, and 0.8 upon subsequent testings. The frequency of migraines experienced by the control group did not show such systematic improvements (1.7, 1.0, 1.3, 1.4 respectively).

The variable Intensity displayed neither significant main nor interaction effects (Table 4), although some interesting patterns in the data did emerge. The treatment group experienced a steady decline in Intensity (as measured on a scale of 1 to 5) throughout the study (2.5, 1.8, 1.6, 1.3, respectively), while the control group experienced relatively no change (2.6, 2.5, 2.6, 2.4, respectively) (Figure 4).

Finally, although Duration of migraine, as displayed in Table 5, did not produce significant main or interaction effects, it did show some promising trends for the treatment group. According to Figure 5, the duration of migraine, for the treatment group, fell from a baseline average of 6.4, to 5.2, 4.5, and 4.0 hours respectively, while the control group experienced increases in the duration of their migraines (6.2, 6.7, 6.9, 6.7 hours respectively).

TABLE 3

Analysis of Variance for Frequency

Source	SS	DF	MS	F
Between Ss				
Group	2.75	1	2.75	1.61
Error	30.77	18	1.71	
Within Ss				
Time	2.75	3	0.92	2.07
Group x Time	1.09	3	0.36	0.83
Error	23.87	54	0.44	

Note.

Box's M = 14.63

F (10, 1390) = 1.10, p = .361

Mauchly Sphericity Test = .60

Chi Square Approx. = 8.50, p = .131

Figure 2. Comparison of Frequency of migraine across group and time

TABLE 4

Analysis of Variance for Intensity

Source	SS	DF	MS	F
Between Ss				
Group	10.78	1	10.78	5.33
Error	36.42	18	2.02	
Within Ss				
Time	4.42	3	1.47	1.40
Group x Time	3.15	3	1.05	1.00
Error	56.80	54	1.05	

Note.

Box's M = 24.36

F (10, 1390) = 1.82, p = .052

Mauchly Sphericity Test = .47

Chi Square Approx. = 12.77, p = .026

Figure 3. Comparison of Intensity of migraine across group and time

TABLE 5

Analysis of Variance for Duration

Source	SS	DF	MS	F
Between Ss				
Group	52.81	1	52.81	0.67
Error	1429.27	18	79.40	
Within Ss				
Time	10.79	3	3.63	0.41
Group x Time	24.95	3	8.32	0.94
Error	478.18	54	8.86	

Note.

Box's M = 24.36

F (10, 1390) = 1.82, p = .052

Mauchly Sphericity Test = .47

Chi Square Approx. = 12.77, p = .026

Figure 4. Comparison of Duration of migraine across group and time

Migraine impact variables. The five migraine impact measures, taken from the MPI (Pain Severity, Life Control, Affective Distress, Support, and General Activity), were measured on seven point scales (ranging from 0 to 6). Separate statistical analyses of these variables revealed a significant main effect of time for Pain Severity. All other impact variables failed to reach significance.

As can be seen in Table 6, a repeated measure ANOVA with a between factor on Pain Severity revealed no significant interaction effects and no significant main effect of group. However, a significant main effect for time was found, $F(3,54) = 6.79, p < .001$.

Turning to Figure 7, we can see that while the treatment group had a mean Pain Severity score of 2.3 at Time A, their scores fell to 1.3 at Time B, and 1.2 at Time C, and rose slightly to 1.6 at Time D. Pain Severity scores for the control group went down from 2.6 at Time A, 1.9 at Time B, 1.8 at Time C, and rose slightly to 2.0 at Time D.

Further analysis, using the Roy-Bargman Stepdown F test, indicated that the main effect for time was most likely due to a significant difference between Time A and Time B, $F(1,18) = 6.85, p = .017$. Paired T tests for each group at Time A to B revealed that the drop in Pain Severity experienced by the treatment group was significant, $t(10) = 3.20, p = .009$, while the difference from time A to Time B for the control group was not significant, $t(8) = 1.93, p = .09$. The significant main effect for time is thus apparently due to a significant decrease in Pain Severity in the treatment group, from Time A to Time B.

A repeated measures ANOVA with a between factor on Life Control failed

TABLE 6

Analysis of Variance for Pain Severity

Source	SS	DF	MS	F
Between Ss				
Group	4.59	1	4.59	0.052
Error	19.12	18	1.06	
Within Ss				
Time	10.73	3	3.58	6.79**
Group x Time	0.34	3	0.11	0.22
Error	28.43	54	0.53	

Roy-Bargman Stepdown F Test

Time	Hyp MS	Hyp DF	Stepdown	Error DF
A vs B	0.41	1	6.85*	18
AB vs C	1.88	1	4.19	17
ABC vs D	2.41	1	4.34	16

* $p < .017$ ** $p < .001$ Note. Box's M = 9.15F (10, 1390) = .686, $p = .735$

Mauchly Sphericity Test = .92

Chi Square Approx. = 1.32, $p = .933$

Figure 5. Comparison of Pain Severity
across group and time

TABLE 7

Analysis of Variance for Life Control

Source	SS	DF	MS	F
Between Ss				
Group	0.22	1	0.22	0.05
Error	73.32	18	4.07	
Within Ss				
Time	7.25	3	2.42	1.92
Group x Time	1.15	3	0.38	0.30
Error	68.08	54	1.26	

Note.

Box's M = 25.38

F (10, 1390) = 1.90, p = .041

Mauchly Sphericity Test = .79

Chi Square Approx. = 4.00, p = .549

Figure 6. Comparison of Life Control
across group and time

TABLE 8

Analysis of Variance for Affective Distress

Source	SS	DF	MS	F
Between Ss				
Group	1.52	1	1.52	0.44
Error	62.57	18	3.48	
Within Ss				
Time	2.46	3	0.82	0.52
Group x Time	0.66	3	0.22	0.14
Error	84.45	54	1.56	

Note.

Box's M = 9.24

F (10, 1390) = .69, p = .732

Mauchly Sphericity Test = .70

Chi Square Approx. = 5.98, p = .308

Figure 7. Comparison of Affective Distress across group and time

TABLE 9

Multivariate Analysis of Variance for Support

Source	SS	DF	MS	F
Between Ss				
Group	1.55	1	1.55	0.23
Error	121.42	18	5.75	
Within Ss				
	Pillais	DF	Error DF	Approx. F
Time	0.25289	3	16	1.81
Group x Time	0.00472	3	16	0.03

Note.

Box's M = 29.00

F (10, 1390) = 2.18, p = .017

Mauchly Sphericity Test = .08

Chi Square Approx. = 42.3, p = .000

Figure 8. Comparison of Support
across group and time

TABLE 10

Analysis of Variance for General Activity

Source	SS	DF	MS	F
Between Ss				
Group	0.68	1	0.68	0.66
Error	18.47	18	1.03	
Within Ss				
Time	0.95	3	0.32	2.97
Group x Time	0.15	3	0.05	0.47
Error	5.78	54	0.11	

Note.

Box's M = 10.08

F (10, 1390) = .756, p = .672

Mauchly Sphericity Test = .61

Chi Square Approx. = 8.19, p = .146

Figure 9. Comparison of General Activity across time by group

Time A

Time B

Time C

Time D

to reveal significant main effects for either group or time, nor did it reveal any interaction effects (Table 7). Indeed, turning to Figure 8, it is apparent that little change in Life Control was experienced by either group. While the treatment group presented with Life Control scores of 4.3, 4.1, 4.4, 3.6 respectively for the four time periods, the control group presented with scores of 4.2, 4.3, 3.9, 3.5, respectively.

A repeated measures ANOVA with a between factor on the variable Affective Distress (Table 8) also failed to produce significant interaction effects as well as significant main effects for either group, or time. However, as can be seen in Figure 9, the treatment group did experience some improvement in Affective Distress from the baseline period through to the end of the aerobic classes (2.9, 2.6, 2.3, respectively), while the control group remained relatively stable (2.9, 3.0, 2.7). Both the treatment and control group experienced slight increases in affective distress at the follow-up (2.7 and 3.0 respectively).

The results of a MANOVA for the variable Support, using the repeated testings of the dependent variable as dependent measures, are displayed in Table 9. Here too we see no significant main effects for either group or time nor were any significant interaction effects found. Turning to Figure 10, one will notice that Support scores for the treatment group (4.1, 4.3, 4.3, 4.1) and control group (3.8, 4.0, 4.0, 3.9) increased slightly at Time B, stabilized at Time C, then decreased upon the final testing.

A repeated measures ANOVA with a between factor on the final impact variable, General Activity, similarly did not produce any significant main or interaction effects (Table 10). In fact, as may be observed in Figure 11, the

treatment group experienced a slight decrease in General Activity over the course of the aerobic program (3.0, 2.9, 2.7, respectively), and a slight increase in activity level upon termination of the program (2.8). The control group, on the other hand, remained relatively stable (3.1, 3.1, 3.0, 2.9 respectively) throughout the study.

Discussion

This study addressed the relationship between cardiovascular fitness and several measures of migraine impact and migraine activity. Aerobic fitness classes were found to be effective in improving cardiovascular fitness. Improvements in cardiovascular fitness had a positive effect on Pain Severity, as perceived by the subject. The effects on Frequency, Intensity, Duration, Life Control, Affective Distress, Support, and General Activity were not statistically significant, although some positive trends did emerge.

Among the leading triggers to migraines is stress. Stress may be perceived as any psychological, physical, or environmental stimuli which activates the sympathetic nervous system. Indeed, classical migraines may be due, in part, to sympathetic overactivity when faced with stimuli which initiate the stress response (Ellerston & Hammerborg, 1982; Johnson, 1978; Sicuteri, 1967). Physiological responses to stress include elevations in heart rate and blood pressure (Pittner & Houston, 1980), increases in monoamines and cortisol levels (Frankenhauser, 1980; Glass et al., 1980; Mason, 1968), and increases in endorphin levels (Grossman, 1983; 1984; Grossman et al., 1982). To the migraineur, this is accompanied by vasoconstriction of the arteries, and reductions in blood flow and is experienced as the aura (Ellerston & Hammerborg, 1982; Johnson, 1978; Sicuteri, 1967). Eventually, in what may be the organism's attempt to re-establish homeostasis, a reversal occurs where parasympathetic functionings take over, creating dilation of the blood vessels and an increase in cranial blood flow (Havanka-Kanninen, 1986; Johnson, 1978). This increase in blood flow may in turn lead to an increase in

pulsation of cranial arteries and be experienced as the headache (Rall & Schleifer, 1980).

In addition to having a low tolerance to stress, migraineurs also appear to require longer periods of time to recover from stressors (C. D. Anderson et al., 1982; Anthony, 1981; Drummond, 1982). Fit people not only can better tolerate stress, but also seem to have the ability to recover faster from different stressors (Clausen, 1977; Keller & Seraganian, 1984; Masironi & Denolin, 1985; Sinkin & Koroblou, 1967; Sinyor et al., 1983). Thus, it was hypothesized that those engaged in aerobic fitness classes would experience fewer, less painful, and shorter migraines.

Migraine activity was assessed using a migraine diary. The migraine diary included a measure of the Frequency, Intensity, and Duration of migraines. Over the course of the study, the treatment group experienced systematic, though nonsignificant, improvements on all three diary measures. No such patterns were detected for the control group. Thus, we may tentatively conclude that, indeed, aerobic exercise may increase one's tolerance for those stimuli which trigger the stress response, which may in turn lessen the frequency of migraines. In the event that migraines do ensue, fitness may moderate the stress reaction, such that the usual and dramatic parasympathetic counterreaction, e.g. dilation of blood vessels and increase in blood flow, will also be moderated, resulting in less painful headaches. Finally, being fit may enable one to recover more quickly from not only the stress which triggered the migraine, but also from the migraine itself.

Migraine sufferers tend to experience more overall distress than controls (Avidrasik et al., 1982; Alvarez, 1984; Beckman, 1989; Bond, 1980; Craig, 1980;

Merikangas et al., 1988; Philips & Jahanshashi, 1985; Wolff, 1937). Studies on cardiovascular fitness have consistently associated fitness with overall physical and psychological health (D.R. Brown, 1986; Ledwidge, 1980; Morgan, 1981; Morgan & Pollack, 1986; Trugillo, 1983). Thus, it was hypothesized that those engaged in aerobic fitness classes would experience a reduction in perceived Pain Severity.

The Pain Severity scale of the MPI requires a retrospective, subjective assessment of the extent to which one suffers as a result of one's pain. As such, it may be used as a global measure of the extent to which one is distressed by one's pain, both physically and psychologically. While both the treatment and control groups experienced reductions in Pain Severity, only that which was experienced by the treatment group proved to be significant. Thus, using a rather global measure of migraine impact, such as the Pain Severity scale of the MPI, we may conclude that aerobic exercise and cardiovascular fitness may lessen both perceived physical, as well as psychological, suffering which often accompagnies migraines. Suffering with an illness, such as migraines, may force one to face one's vulnerablility to the uncertainty of life and lead to a loss of sense of control (Cohen & Lazarus, 1979). Studies have concluded that exercise may lead to increases in feelings of control and mastery (Gal & Lazarus, 1975). It was therefore hypothesized that those engaged in the aerobic fitness classes would experience enhancements in the extent to which they felt control in their lives.

No significant improvements in Life Control were found in the present study. In fact, no systematic pattern of Life Control could be found for either group. Thus, the present study's findings did not concur with past literature on exercise and

control. Such inconsistency may be due to, among other factors, the clinical nature of the sample used in this study. Most studies on exercise and control have used clinically depressed subjects or normal, healthy subjects. None have looked at the effects that exercise may exert on mastery and control with clinical pain populations, such as migraineurs. It may be that improvements in fitness, coupled with only slight improvements in headache activity, are not sufficient to improve feelings of life control when one suffers with an unpredictable illness, such as migraines.

Studies have found migraineurs to be more emotionally distressed and to experience more depression than controls (Avidraski et al., 1982; Alvarez, 1974; Friedman et al., 1954; Merikangan et al., 1988; Wolff, 1937). Past research has consistently related aerobic activity with declines in depression and affective distress (D. R. Brown; Greist et al., 1979; Lederman, 1980; Morgan, 1981; Trugillo, 1983). Such benefits have been associated with both cognitive (Layman, 1974), and physiological (e.g. Barchas & Freedman, 1963; Hughes, 1984; Morgan, 1985) factors. Thus, it was hypothesized that the exercising group would experience a reduction of Affective Distress.

The Affective Distress scale of the MPI assesses the extent to which persons are subjectively distressed by their pain. The treatment group did experience a steady reduction in affective distress over the course of the aerobic program. Their Affective Distress scores fell steadily from the baseline period through to the end of the aerobic classes. This improvement in emotional functioning, however, failed to reach significance. The control group showed no such improvement in their affective experience. Thus, while a trend toward reduced affective distress was found for the

treatment group, it failed to reach significance in our small sample, over the course of a six week exercise program.

Research has demonstrated that persons with migraines tend to be somewhat more interpersonally distressed than controls (Beckstead, 1989; Craig, 1980; Philips & Jahanshahi, 1985). Exercise has been associated with sexual well-being and improvements in intimate relationships (Greenberg & Pargman, 1989; Harris & Guin, 1985; Harris & Kagan, 1985). Such improvements may be due, in part, to improved physical appearance, improved self-confidence, and improvements in mood, so often associated with exercise (Allen, 1972; Hart, 1971; Morgan, 1981; Morgan & Pollack, 1976; Trugillo, 1983). It was therefore hypothesized that those in the aerobics treatment would experience improved interpersonal satisfaction, as reflected in the Support scale of the MPI.

The Support scale of the MPI measures the extent to which one feels care and support from spouse, family, and significant others, and may reflect interpersonal distress or satisfaction. Both the treatment and control groups experienced, over the course of the study, some, albeit statistically nonsignificant, improvement in the extent to which they felt supported by their significant other. Moreover, since improvements were experienced by both groups, we must conclude that something other than exercise may have contributed to improvements in feelings of support, e.g. being involved in a study.

Migraines have been found to limit one's ability to enjoy free time or function properly at work (Bond, 1980; Levor et al., 1986; Zenz, 1983). Exercise increases one's level of energy, mental alertness, and productivity (Astrand, 1977; Debats,

1981; Falkenberg, 1987; Gutin & Digenmare, 1966; Howard & Mikilachi, 1979; Lightman & Poser, 1973; Powel, 1975). Indeed, the better one feels both physically and psychologically, the more one may be expected to engage in various activities. Thus, it was hypothesized that those engaged in aerobic exercise would experience an increase in overall activity level.

The General Activity scale of the MPI measures the degree to which one engages in various activities, including household chores, outdoor work, activities away from home, and social activities. No significant changes in activity level were found for either the treatment or control group. In fact, those in the treatment condition actually decreased (though not significantly) their level of activity over the course of the aerobic classes, while the control group remained relatively stable over the study. It may be that the treatment group's activity level fell as a function of their participating in the aerobic classes, which curtailed the amount of time left over for other activities.

A plausible explanation for the observed improvements in several variables by the treatment group may be that they had greater expectations for improvements. However, a two-week follow up ensured that fitness levels for the treatment group remained elevated, and no significant regressions in any of the activity or impact variables took place. In addition, the pattern of improvement for the treatment group was not uniform, as would be expected by a generalized expectancy effect. For example, the exercise group did not experience improved feelings of control, support, or increases in general activities. Thus, expectancy effects may be ruled out in this study.

In addition, some may conclude that such improvements in support may account for those improvements found in migraine activity and impact, certain factors would negate such a conclusion. First, participants did not form collective friendships during the course of the study. They came to the aerobic classes, completed the classes, and left promptly. Rarely did they interact socially with one another. In addition, since both the treatment and control group experienced improvements in support, yet only the treatment group experienced some improvement in migraine activity and impact, we may conclude that social support was not likely acting as a buffer to migraines.

Overall, then, aerobic exercise has been confirmed to be an effective activity for improving cardiovascular fitness. Improvements in cardiovascular fitness have been found to significantly reduce the amount of suffering experienced as a result of having migraines. In other words, those involved in aerobic classes did not experience significantly fewer or shorter migraines over the course of the study, but those migraines that they did experience were considered, retrospectively, to be less severe and less distressing both physically and psychologically.

Certain cautions must be exercised in generalizing the results of this study to the classical migraine population. To begin, this study focused exclusively on women, and most were white middle class with university degrees, who were required to meet a strict inclusion criteria, e.g. experience at least two migraines a month. As a result, the sample was quite small. Thus, one cannot conclude that the same pattern of results would be found with males, or with people of different socioeconomical status and educational levels. Future research should be sensitive

to these needs and incorporate large samples of migraineurs from both sexes, at all levels of the SES scale, with varying degrees of migraine experience. In addition, future research should incorporate longer exercise programs, so that the trends toward reduced negative affect, frequency, intensity, and duration of migraines for those who exercised may be explored in more detail. Also, prospective studies should follow the course of migraines more closely, to explore just which phase of the episode (prodrome, aura, headache, or postdrome) is most affected by exercise.

An additional sampling problem in this study is the potential tendency, of those women who initially presented themselves to the study, to engage in fitness. Although the women were sampled from all levels of the fitness scale, it may be that their motivation to exercise and to alleviate their migraines created for a special set of people. Future research should be sensitive to this potential.

A further issue which must be addressed, and one which plagues all pain research, is self-medication. It is virtually impossible to follow the course of a migraine when participants self-medicate. Of course, one may not ethically request that subjects stop medicating, but one should be aware of the limitations imposed in the interpretation of research as a result of self-medication.

Finally, the monumental question remains...What is pain??. There are no physical, objective measures of head pain. It is necessarily a subjective experience. Therefore, one must rely on the rather unscientific, subjective interpretation of the pain sufferer. While it is impossible to objectively assess the pain experienced by migraineurs, future research with other pain groups should make an attempt to incorporate both objective and subjective measures in the determination of pain

intensity. Such an approach would allow for some enlightening comparisons of actual and perceived physical distress in other pain conditions.

In conclusion, the results of this study provide a promising rationale for persons with migraines to exercise. While only the retrospective perception of physical and psychological distress associated with migraines reached significance, many other measures provided promising trends, including the potential that exercise may reduce the negative affect which so often accompanies migraines, as well as enhance interpersonal and social relationships, and decrease the frequency, intensity, and duration of migraines.

Perhaps the most important finding to come out of this study is the result that aerobic exercise does not hurt. Migraines did not increase in number as a result of exercise, nor did they last longer, a significant finding in its own right. Thus, at the very least, exercise may enable migraineurs to get fit, feel good about themselves, and gain self-respect. At the very most, exercise may make migraines more bearable and possibly do away with them altogether.

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Appendix A

Headache Questionnaire

Informed Consent -- Headache Questionnaire

The information to be provided in this questionnaire is freely volunteered by me with the expressed understanding that such information is to be used for research purposes only and that I will not be identified in any use to which the information may be put.

Signature

Date

Questionnaire Con't...

Below is a list of symptoms which are sometimes associated with migraines. Please mark a check in the space beside each symptom if it occurs during your typical migraine. Feel free to add any other symptoms which you have experienced.

___ visual difficulties, and/or odd visual sensations,
i.e. patterns, flashing lights, lines, spots, double
images, strange color perceptions or sensations,
blurred vision, partial blindness, and sensitivity
to light increases, strange object or size
perception

___ auditory sensations, i.e. buzzing, ringing, or
roaring in my ears, and/or increased sensitivity
to sound

___ hypersensitivity to touch

___ feelings of pins and needles or numbness

___ chills or cold

___ hot, feverish flashes

___ dizziness

___ hypersensitivity to odours or strange odor
sensations

___ trouble communicating with other, i.e. I have
trouble speaking, understanding what others are
saying to me.

___ forgetfulness and/or confusion

___ depression

___ elation

___ fatigued, sleepy or groggy

___ changes in appetite, cravings

___ constipation, diarrhea, nausea and/or vomiting

Questionnaire Con't...

The following questions pertain to the symptom (s) which you checked off in the previous page. Please answer in reference to your typical migraine.

- A. In an average month, how many migraines do you have?

- B. Approximately how many headaches which were accompanied with some of the above associated symptoms have you experienced? Please specify time frame, i.e. two in the past month.

- C. Do these associated symptoms develop gradually, or onset very rapidly? Please indicate the period of time over which these symptoms usually develop (i.e. over 10 minutes).

- D. During your average migraine episode, how long do the above associated symptoms which you listed last?

- E. How soon after the above associated symptoms stop does your typical headache begin (please specify if in minutes, hours).

Questionnaire Con't...

Headache History

The following is a headache history form. Please read it carefully and answer, to the best of your recollection.

Name:

Date of Birth:

Occupation:

- a) What is the highest level of education you have completed? Check that which approximates your last level completed.
- ☐ Some high school
 - ☐ Graduated high school
 - ☐ Some college
 - ☐ Graduated college
 - ☐ Some graduate studies
 - ☐ Completed graduate studies
- b) When did you first experience migraines similar to those you have now?
- c) Describe any major changes in your life which you feel were associated with the onset of your migraines.

- d) The following is a list of some common causes of head pain. Please mark a check in the space beside any of the following which your physician has suggested may be the cause of your head pain:

- ☐ Head Trauma, i.e. car accident, fall
- ☐ Stroke
- ☐ Hematoma
- ☐ Hypertension
- ☐ Tumor
- ☐ Alcohol
- ☐ Narcotics
- ☐ Carbon monoxide
- ☐ Viral infection
- ☐ Bacterial infections
- ☐ Disorders associated with: neck, ears, eyes, teeth, nose, or other cranial or facial structures

- e) If the frequency, intensity, and/or duration of your migraines have changed significantly in the past years, describe the changes and indicate any life events which occurred at the same time as these changes.

- f) Please list all medications you are currently using, whether these are to control your migraines or any other condition.

Appendix B

Physical Activity Readiness Questionnaire (PAR-Q)

Physical Activity Readiness
Questionnaire (PAR-Q)*

NAME OF PARTICIPANT _____

DATE _____

PAR Q & YOU

PAR-Q is designed to help you help yourself. Many health benefits are associated with regular exercise, and the completion of PAR-Q is a sensible first step to take if you are planning to increase the amount of physical activity in your life.

For most people physical activity should not pose any problem or hazard. PAR-Q has been designed to identify the small number of adults for whom physical activity might be inappropriate or those who should have medical advice concerning the type of activity most suitable for them.

Common sense is your best guide in answering these few questions. Please read them carefully and check (✓) the ☐ YES or ☐ NO opposite the question if it applies to you.

YES NO

- ☐ ☐ 1. Has your doctor ever said you have heart trouble?
- ☐ ☐ 2. Do you frequently have pains in your heart and chest?
- ☐ ☐ 3. Do you often feel faint or have spells of severe dizziness?
- ☐ ☐ 4. Has a doctor ever said your blood pressure was too high?
- ☐ ☐ 5. Has your doctor ever told you that you have a bone or joint problem such as arthritis that has been aggravated by exercise, or might be made worse with exercise?
- ☐ ☐ 6. Is there a good physical reason not mentioned here why you should not follow an activity program even if you wanted to?
- ☐ ☐ 7. Are you over age 65 and not accustomed to vigorous exercise?

If
You
Answered

YES to one or more questions

NO to all questions

If you have not recently done so, consult with your personal physician by telephone or in person BEFORE increasing your physical activity and/or taking a fitness appraisal. Tell your physician what questions you answered YES to on PAR-Q or present your PAR-Q copy.

programs

After medical evaluation, seek advice from your physician as to your suitability for:

- unrestricted physical activity starting off easily and progressing gradually
- restricted or supervised activity to meet your specific needs, a restriction on initial tasks, or seek in your community for special programs or services.

If you answered PAR-Q accurately you have reasonable assurance of your present suitability for:

- A GRADUATED EXERCISE PROGRAM - a gradual increase in proper exercise promotes good fitness development while minimizing or eliminating discomfort
- A FITNESS APPRAISAL - the Canadian Standardized Test of Fitness (CSTF)

postpone

If you have a temporary minor illness, such as a common cold.

Appendix C

Consent and Release Form for the
Canadian Standardized Test of Fitness

I, the undersigned, do hereby acknowledge:

a) my consent to perform a fitness test, conducted by a certified Standardized Test of Fitness Appraiser who has been trained to administer the Canadian Standardized Test of Fitness, consisting of stepping on double 20 cm steps at speeds appropriate for my age and gender, measurements of standing height, and weight, the results of which will assist in determining my level of cardiovascular fitness;

b) my understanding that my heart rate and blood pressure will be measured prior to and at the completion of the test;

c) my understanding that there are potential risks, i.e. episodes of transient light-headedness, fainting, leg cramps, and nausea, and that I assume willfully those risks;

d) my obligation to immediately inform the appraiser of any pain, discomfort, fatigue, or any other symptoms that I may suffer during and immediately after the testing;

e) my understanding that I may stop or delay further testing, or withdraw from the study, if I so desire and that the testing may be terminated by the appraiser upon observation of any symptoms of distress or abnormal response;

f) that I have read, understood, and completed the Physical Activity Readiness Questionnaire, and that my answers to all the questions were negative (except where question 3 relates to my migraine episodes only);

g) that I hereby release Donna Lockett and Carleton University from any liability with respect to any damage or injury that I may suffer during the administration of the Canadian Standardized Test of Fitness, except where the damage or injury is caused by the negligence of the appraiser.

Signature

Date

Appendix D

Consent and Release Form for Fitness Classes

I, the undersigned, do hereby acknowledge:

My consent to partake in aerobic fitness classes, taught by a certified fitness instructor, suitable for my age, gender, and level of fitness. To the best of my knowledge, I am in good health and have no physical disabilities or conditions which might be exacerbated by fitness classes. I understand that I may stop exercising at any time during a class, and that I am free to withdraw from the study at any time. I also understand that I may be requested to stop exercising by the instructor upon observation of any symptoms of distress. Further, I understand that it is my obligation to immediately inform the instructor of any pain, discomfort, fatigue, or any other symptoms that I may suffer during and immediately after classes.

I hereby release Donna Lockett and Carleton University from any liability with respect to any damage or injury that I may suffer during the fitness classes.

Signature

Date

Appendix E

Canadian Aerobic Fitness Test

Appendix E-1

Construction Plans for the Ergometer Steps

Ergometer Steps

It is advised that steps be constructed in 1.2 meter (4 foot) lengths in order to store and transport easily.

Details:

1. Double 203 cm steps, cut to desired length.
2. Use 19 cm. (¾ inch) plywood
3. Supporting panels (F) every 0.9 to 1.2 m

4. Step Dimensions:

A 184 cm.	} by 1.2 m
B 25 cm.	
C 203 cm.	
D 45 cm.	
E 26.8 cm.	
F 70 cm.	

5. Handrail Dimensions:

- G Approx. 100 cm
H Approx. 137.5 cm

Source: CASS and Fitness Canada, 1986.

Appendix E-2

Ceiling Target Heart Rate Chart

Age Group	Starting Stage		Heart Rate (10 seconds) after 1st session		Heart Rate (10 seconds) after 2nd session		
	Male	Female					
60's	1	1	STOP if 24 or more Calculate average heart rate	GO only if 22 or less	Everyone STOP Calculate average heart rate		Note: Everyone aged 60 or over stop stepping after the 2nd stage if handgrips are not available or the ergometer stops the appraiser should stop with older participants as a precaution against falls
50's	2	1	STOP if 23 or more Calculate average heart rate	GO only if 20 or less	STOP if 23 or more Calculate average heart rate	GO only if 22 or less	
40's	3	2	STOP if 26 or more Calculate average heart rate	GO only if 25 or less	STOP if 26 or more Calculate average heart rate	GO only if 22 or less	
30's	4	3	STOP if 28 or more Calculate average heart rate	GO only if 27 or less	STOP if 28 or more Calculate average heart rate	GO only if 24 or less	
20's	5	3	STOP if 29 or more Calculate average heart rate	GO only if 29 or less	STOP if 29 or more Calculate average heart rate	GO only if 25 or less	
15-19	5	4	STOP if 30 or more Calculate average heart rate	GO only if 29 or less	STOP if 30 or more Calculate average heart rate	GO only if 26 or less	

This chart summarizes the administration of the CAFT. Determine the heart rate with the stethoscope placed either on the sternum or over the second intercostal space on the left side. Start counting the pulse at the termination of the command word "COUNT" and continue counting until the first sound of the command word "STOP". This is a 10 second timing sequence and the first beat is counted as one.

Calculate the aerobic fitness level utilizing:
a) Aerobic Calculator: Norms and Percentiles for Predicted $\dot{V}O_2$ max
b) Norms and Percentiles for Mean Rate Final Score

Source: CASS and Fitness Canada, 1986

Appendix E-3

Calculating Aerobic Fitness

- 1 Factor 1
Select factor 1 from LAST STEPPING STAGE COMPLETED (see below)

Stage	1	2	3	4	5	6	7
Male	61.4	64.9	69.5	72.8	75.8	81.4	88.4
Female	58.1	59.8	64.4	67.3	69.5	72.2	

Factor 1 _____

- 2 Factor 2
Determine factor 2 from participant's NEAREST BODY WEIGHT AND AGE from table below

Factor 2 _____

- 3 Factor 3
SUBTRACT factor 2 from factor 1

Factor 3 _____

- 4 Factor 4
Select factor 4 from POST-EXERCISE HEART RATE for last stepping stage completed (see below)

Factor 4 _____

Post-Ex HR	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35
Factor 4	122	129	137	144	151	158	166	173	180	187	194	202	209	216	223	230	236	243	252

- 5 SUBTRACT factor 4 from factor 3 to get PREDICTED VO_2 MAX ($\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$)

 VO_2 MAX _____

- 6 Determine ranking from NORMS AND PERCENTILES FOR PREDICTED VO_2 MAX (see Operations Manual, pg 34)

Percentile _____

Aerobic fitness may also be determined directly from NORMS AND PERCENTILES FOR HEART RATE FINAL SCORES (see Operations Manual, pg 35)

Percentile _____

Factor 2

Age	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42	43	44	45	46	47	48	49	50	51	52	53	54	55	56	57	58	59	60	61	62	63	64	65	66	67	68	69	70	71	72	73	74	75	76	77	78	79	80	81	82	83	84	85	86	87	88	89	90	91	92	93	94	95	96	97	98	99	100																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																														
 

Appendix E-4

Norms and Percentiles by Age Group and Gender
for Predicted Maximum Volume of Oxygen Consumption

Norms

Age (yrs.)	15-19		20-29		30-39		40-49		50-59		60-69	
Gender	M	F	M	F	M	F	M	F	M	F	M	F
Excellent	≥ 60	≥ 43	≥ 57	≥ 40	≥ 48	≥ 37	≥ 42	≥ 36	≥ 38	≥ 30	≥ 30	≥ 25
Above Average	58-59	40-42	52-56	37-39	46-47	34-37	40-42	32-34	36-38	27-29	29-30	24-25
Average	54-57	37-39	43-51	33-37	42-45	31-33	37-39	29-31	34-35	25-27	27-28	22-23
Below Average	44-53	35-37	40-42	32-34	38-41	29-31	34-37	24-25	31-33	22-25	26-27	20-22
Poor	≤ 43	≤ 34	≤ 40	≤ 31	≤ 37	≤ 29	≤ 33	≤ 23	≤ 30	≤ 21	≤ 26	≤ 19

Percentiles

Age (yrs)	15-19		20-29		30-39		40-49		50-59		60-69	
Gender	M	F	M	F	M	F	M	F	M	F	M	F
Percentiles												
95	82	45	59	43	51	39	44	36	40	31	32	26
90	61	43	58	42	50	38	43	35	39	30	31	26
85	60	43	57	40	48	37	42	35	38	30	30	25
80	58	42	56	39	47	37	42	34	38	29	30	25
75	58	41	55	39	47	36	41	33	37	28	29	24
70	56	40	54	38	46	35	40	33	36	28	29	24
65	58	40	52	37	46	34	40	32	36	27	29	24
60	57	39	48	37	45	33	39	31	35	27	28	23
55	57	38	47	36	44	32	38	30	35	26	28	23
50	56	38	43	35	43	32	38	29	34	26	28	22
45	54	37	43	35	42	31	37	28	34	25	27	22
40	52	37	42	34	41	31	37	26	33	25	27	22
35	47	36	42	34	40	30	36	25	33	24	27	21
30	46	35	41	33	39	30	35	24	32	23	27	21
25	44	35	40	32	38	29	34	24	31	22	26	20
20	43	34	40	31	37	29	32	23	29	21	26	19
15	42	34	39	31	36	28	31	22	28	20	25	19
10	41	33	38	30	34	28	30	22	25	19	24	18
5	40	32	37	29	33	27	29	21	24	18	23	17

*Based on data from the Canada Fitness Survey, 1961

Source: CASS and Fitness Canada, 1986

Appendix F

West Haven-Yale Multidimensional Pain Inventory

Informed Consent

West Haven-Yale Multidimensional Pain Inventory

The information to be provided in this questionnaire is freely volunteered by me with the expressed understanding that such information is to be used for research purposes only and that I will not be identified in any use to which the information may be put.

Signature

Date

SCORING PROCEDURES

SECTION I - MPI Version 1

Scale 1: Pain Severity	= $(Q1+Q7+Q12) / 3^a$
Scale 2: Interference	= $(Q2+Q3+Q4+Q8+Q9+Q13+Q14+Q17+Q19) / 9$
Scale 3: Life Control	= $(Q11+Q16) / 2$
Scale 4: Affective Distress	= $((6-Q6)+Q18+Q20) / 3$
Scale 5: Support	= $(Q5+Q10+Q15) / 3$

SECTION II

Scale 6: Punishing Responses	= $(Q1+Q4+Q7+Q10) / 4$
Scale 7: Solicitious Responses	= $(Q2+Q5+Q8+Q11+Q13+Q14) / 6$
Scale 8: Distracting Responses	= $(Q3+Q6+Q9+Q12) / 4$

SECTION III

Scale 9: Household Chores	= $(Q1+Q5+Q9+Q13+Q17) / 5$
Scale 10: Outdoor Work	= $(Q2+Q6+Q10+Q14+Q18) / 5$
Scale 11: Activities Away From Home	= $(Q3+Q7+Q11+Q15) / 4$
Scale 12: Social Activities	= $(Q4+Q8+Q12+Q16) / 4$
Scale 13: General Activity Level	= $(Scale9+Scale10+Scale11+Scale12) / 4$

^a This scoring procedure calculates a mean score for each scale. The denominator, which reflects the number of items in that scale, will need to be adjusted if there are missing values for the summed items in a particular scale (i.e., the numerator). For example, if a patient indicated that question 1 in Section I was not applicable or left this question blank, then the denominator of scale 1 would be 2 rather than 3 and only 2 items would be summed to form the numerator. This type of adjustment should be made for each scale that contains missing values so that a patient's score can be compared to scale norms as well as compared back to the original unit of measurement, the item ratings, on which scale scores are based.

0	1	2	3	4	5	6
No change						Extreme change

0	1	2	3	4	5	6
Not at all
supportive						Extremely
supportive

0	1	2	3	4	5	6
Extremely
low mood						Extremely
high mood

0	1	2	3	4	5	6
Not at all
severe						Extremely
severe

0	1	2	3	4	5	6
No change						Extreme change

0	1	2	3	4	5	6
No change						Extreme change

0	1	2	3	4	5	6
Not at all						Extremely
worried						worried

0	1	2	3	4	5	6
Not at all						Extremely
in control						in control

0	1	2	3	4	5	6
No suffering						Extreme suffering

0	1	2	3	4	5	6
No change						Extreme change

0	1	2	3	4	5	6
No change						Extreme change

0	1	2	3	4	5	6
Not at all attentive						Extremely attentive

0	1	2	3	4	5	6
Not at all						Extremely well

0	1	2	3	4	5	6
No change						Extreme change

0	1	2	3	4	5	6
Not at all						Extremely
irritable						irritable

0	1	2	3	4	5	6
No change						Extreme change

20. During the past week how tense or anxious have you been?

0	1	2	3	4	5	6
Not at all						Extremely
tense or anxious						tense and anxious

Section 2

In this section, we are interested in knowing how your spouse (or significant other) responds to you when he or she knows that you are in pain. On the scale listed below each question, circle a number to indicate how often your spouse (or significant other) responds to you in that particular way when you are in pain. Please answer all of the 14 questions.

1. Ignores me.

0	1	2	3	4	5	6
Never						Very often

2. Asks me what he/she can do to help.

0	1	2	3	4	5	6
Never						Very often

3. Reads to me.

0	1	2	3	4	5	6
Never						Very often

4. Expresses irritation at me.

0	1	2	3	4	5	6
Never						Very often

5. Takes over my jobs or duties.

0	1	2	3	4	5	6
Never						Very often

6. Talks to me about something else to take my mind off the pain.

0	1	2	3	4	5	6
Never						Very often

7. Expresses frustration at me.

0	1	2	3	4	5	6
Never						Very often

8. Tries to get me to rest.

0 1 2 3 4 5 6
Never Very often

9. Tries to involve me in some activity.

0 1 2 3 4 5 6
Never Very often

10. Expresses anger at me.

0 1 2 3 4 5 6
Never Very often

11. Gets me some pain medications.

0 1 2 3 4 5 6
Never Very often

12. Encourages me to work on a hobby.

0 1 2 3 4 5 6
Never Very often

13. Gets me something to eat or drink.

0 1 2 3 4 5 6
Never Very often

14. Turns on the T.V. to take my mind off my pain.

0 1 2 3 4 5 6
Never Very often

Section 3

Listed below are 18 common daily activities. Please indicate how often you do each of these activities by circling a number on the scale listed below each activity. Please complete all 18 questions.

1. Wash dishes.

0 1 2 3 4 5 6
Never Very often

2. Mow the lawn.

0 1 2 3 4 5 6
Never Very often

3. Go out to eat.

0	1	2	3	4	5	6
Never						Very often

4. Play cards or other games.

0	1	2	3	4	5	6
Never						Very often

5. Go grocery shopping.

0	1	2	3	4	5	6
Never						Very often

6. Work in the garden.

0	1	2	3	4	5	6
Never						Very often

7. Go to a movie.

0	1	2	3	4	5	6
Never						Very often

8. Visit friends.

0	1	2	3	4	5	6
Never						Very often

9. Help with the house cleaning.

0	1	2	3	4	5	6
Never						Very often

10. Work on the car.

0	1	2	3	4	5	6
Never						Very often

11. Take a ride in a car.

0	1	2	3	4	5	6
Never						Very often

12. Visit relatives.

0	1	2	3	4	5	6
Never						Very often

13. Prepare a meal.

0	1	2	3	4	5	6
Never						Very often

14. Wash the car.

0	1	2	3	4	5	6
Never						Very often

15. Take a trip.

0	1	2	3	4	5	6
Never						Very often

16. Go to a park or beach.

0	1	2	3	4	5	6
Never						Very often

17. Do a load of laundry.

0	1	2	3	4	5	6
Never						Very often

18. Work on a needed house repair.

0	1	2	3	4	5	6
Never						Very often

Appendix G

Headache Diary

DIARY INFORMED CONSENT

The information to be provided in this diary is freely volunteered by me with the expressed understanding that such information is to be used for research purposes only and that I will not be identified in any use to which the information may be put.

Signature

Date

Headache Diary Con't...

Headache Diary

Please indicate, on the sheets provided, the occurrence (or non occurrence) of a migraine for each day specified, as well as the severity of the migraine pain, and the duration of your migraine (in hours and minutes). The following instructions should help you determine what you should fill out:

- a) In the first column, entitled "Pain", please indicate whether you had a migraine today. If you did experience a migraine, indicate "Yes" and complete the subsequent columns. If you did not have a migraine today, indicate "No" and disregard the subsequent questions.
- b) In the second column, entitled "Pain Intensity", please indicate, on a scale of 1 to 5 the intensity of the pain associated with your migraine for that day.
 - 1 = mild pain
 - 2 = discomforting pain
 - 3 = distressing pain
 - 4 = horrible pain
 - 5 = excruciating pain
- c) In the third column, below the title "Duration", please indicate the duration of your migraine, in hours and minutes.

The diary should be filled out every night, at bedtime. Please feel free to make any comments on the back of the sheets. Should you have any questions or concerns, you may contact me at anytime.

Thank You for your cooperation.

Donna Lockett

(731-7933)

Headache Diary

Name: _____

<u>Date</u>	<u>Pain</u>	<u>Pain Intensity</u>	<u>Duration</u>
	(Yes/No)	(0-5)	(Hours/Minutes)

May 1 _____

May 2 _____

May 3 _____

May 4 _____

May 5 _____

May 6 _____

May 7 _____

May 8 _____

May 9 _____

May 10 _____

May 11 _____

May 12 _____

May 13 _____

May 14 _____

May 15 _____

May 16 _____

May 17 _____

May 18 _____

May 19 _____

Headache Diary

Name: _____

<u>Date</u>	<u>Pain</u>	<u>Pain Intensity</u>	<u>Duration</u>
	(Yes/No)	(0-5)	(Hours/Minutes)
May 20	_____	_____	_____
May 21	_____	_____	_____
May 22	_____	_____	_____
May 23	_____	_____	_____
May 24	_____	_____	_____
May 25	_____	_____	_____
May 26	_____	_____	_____
May 27	_____	_____	_____
May 28	_____	_____	_____

Headache Diary

Name: _____

<u>Date</u>	<u>Pain</u>	<u>Pain Intensity</u>	<u>Duration</u>
	(Yes/No)	(0-5)	(Hours/Minutes)
May 29	_____	_____	_____
May 20	_____	_____	_____
May 31	_____	_____	_____
June 1	_____	_____	_____
June 2	_____	_____	_____
June 3	_____	_____	_____
June 4	_____	_____	_____
June 5	_____	_____	_____
June 6	_____	_____	_____
June 7	_____	_____	_____
June 8	_____	_____	_____
June 9	_____	_____	_____
June 10	_____	_____	_____
June 11	_____	_____	_____
June 12	_____	_____	_____
June 13	_____	_____	_____
June 14	_____	_____	_____
June 15	_____	_____	_____
June 16	_____	_____	_____

Headache Diary

Name: _____

Date Pain Pain Intensity Duration

(Yes/No)

(0-5)

(Hours/Minutes)

June 17 _____

June 18 _____

Headache Diary

Name: _____

<u>Date</u>	<u>Pain</u>	<u>Pain Intensity</u>	<u>Duration</u>
	(Yes/No)	(0-5)	(Hours/Minutes)
June 19	_____	_____	_____
June 20	_____	_____	_____
June 21	_____	_____	_____
June 22	_____	_____	_____
June 23	_____	_____	_____
June 24	_____	_____	_____
June 25	_____	_____	_____
June 26	_____	_____	_____
June 27	_____	_____	_____
June 28	_____	_____	_____
June 29	_____	_____	_____
June 30	_____	_____	_____
July 1	_____	_____	_____
July 2	_____	_____	_____
July 3	_____	_____	_____
July 4	_____	_____	_____
July 5	_____	_____	_____
July 6	_____	_____	_____
July 7	_____	_____	_____

Headache Diary

Name: _____

<u>Date</u>	<u>Pain</u>	<u>Pain Intensity</u>	<u>Duration</u>
	(Yes/No)	(0-5)	(Hours/Minutes)

July 8 _____

July 9 _____

Headache Diary

Name: _____

<u>Date</u>	<u>Pain</u>	<u>Pain Intensity</u>	<u>Duration</u>
	(Yes/No)	(0-5)	(Hours/Minutes)
July 10	_____	_____	_____
July 11	_____	_____	_____
July 12	_____	_____	_____
July 13	_____	_____	_____
July 14	_____	_____	_____
July 15	_____	_____	_____
July 16	_____	_____	_____
July 17	_____	_____	_____
July 18	_____	_____	_____
July 19	_____	_____	_____
July 20	_____	_____	_____
July 21	_____	_____	_____
July 22	_____	_____	_____
July 23	_____	_____	_____

Appendix H

Target Heart Rate Chart

In order to improve aerobic or energy fitness, it is necessary to get your heart beating faster at a steady and sustained rate for a minimum of 15 to 20 min (30 to 45 min as you become better conditioned). Activities such as walking, jogging, cycling, swimming, dancing, and cross-country skiing are the best kinds of aerobic activities because they are intense enough to increase your heart rate, and continuous, so that you can keep your heart rate at a constant level for the duration of the activity.

For maximum benefit, your heart rate should be within a certain range during exercise. This range is known as the *Target Zone* and it is determined by your age. The graph below gives the upper and lower limits of the target zone for people of different ages.

Heart Rate Target Zone

Your target zone: upper limit _____
lower limit _____

Note:

1. Working out below the lower limit of your target zone will not do much to improve your aerobic fitness.

2. Working out at a level of intensity that pushes your heart rate above the upper limit will not add anything to your fitness (it will only tire you out quickly, and, in some cases may be dangerous).

3. As you get fitter, your target zone does not change. Rather, you will have to work out more energetically to get your heart rate into the target zone.

4. When you're starting out on a fitness program, don't push your heart rate too far above the lower limit of your target zone.

Checking Your Heart Rate

You can check your heart rate by taking your pulse, either in your neck, at the soft spot just beside your Adam's apple; or on the inside of your wrist, a few centimetres below the base of your thumb. Pressing *lightly*, use the tips of your first two fingers to feel the pulse. Counting the first pulse as 0, count the total number of pulses for 15 s (seconds). Multiplying this number by 4 will give you your heart rate in beats per minute.

To determine your heart rate during activity, you should take your pulse as soon as you stop your exercise. If you wait (even 30 s), your heart rate will have begun to drop, and you will not get an accurate reading.

Using the chart below, which allows you to fill in your heart rate during exercise for 4 activity sessions, you will be able to see if you are working out in your target zone.

Date	Activity	Heart Rate (beats/min)
1		
2		
3		
4		

Source: Participaction, Metric Commission Canada,

and Fitness Canada, 1986

Appendix 1

RAW DATA

ID	FITNESS				PAIN SEVERITY				LIPID CONTROL				AFFECTIVE DISTRESS				SUPPORT				GENERAL ACTIVITY				REMOUNTAIN				INTENSITY				DURATION																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																				
	A	B	C	D	A	B	C	D	A	B	C	D	A	B	C	D	A	B	C	D	A	B	C	D	A	B	C	D	A	B	C	D																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																					
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64	57	59	59	1.5	0.7	1.3	1.7	4.5	5.5	5.0	6.0	2.7	3.7	2.7	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3

CONTROL GROUP

631	10	5	5	5	2.7	1.0	1.7	3.0	4.0	5.0	4.5	4.0	2.0	2.7	2.7	2.7	2.7	2.7	2.7	2.7	2.7	2.7	2.7	2.7	2.7	2.7	2.7	2.7	2.7	2.7	2.7	2.7
247	36	36	55	55	2.0	1.7	0.7	1.7	0.0	0.0	1.0	1.0	2.7	3.7	6.0	5.7	4.3	2.0	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7
323	40	40	43	43	2.7	2.0	1.0	2.0	3.5	4.0	3.5	4.0	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3
150	23	23	31	30	3.7	1.0	2.7	1.3	4.0	5.0	4.0	5.0	1.7	3.3	1.3	1.0	1.0	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7
544	70	72	70	60	1.7	2.0	0.3	0.3	4.5	2.5	2.0	1.5	2.3	2.0	2.3	2.0	2.0	2.0	2.0	2.0	2.0	2.0	2.0	2.0	2.0	2.0	2.0	2.0	2.0	2.0	2.0	2.0
90	24	31	33	31	2.3	2.0	2.3	1.7	5.5	4.0	2.5	1.0	2.7	0.0	4.7	3.7	3.7	3.7	3.7	3.7	3.7	3.7	3.7	3.7	3.7	3.7	3.7	3.7	3.7	3.7	3.7	3.7
767	70	95	97	97	2.7	1.7	1.7	1.7	5.0	5.0	5.0	5.0	3.7	1.0	2.0	1.0	3.0	0.7	4.7	4.3	1.9	3.0	2.5	2.4	2.0	2.0	2.0	2.0	2.0	2.0	2.0	2.0
944	45	45	47	46	3.0	3.0	1.3	3.0	1.0	5.5	6.0	6.0	1.7	0.0	0.7	1.7	6.0	6.0	6.0	6.0	6.0	6.0	6.0	6.0	6.0	6.0	6.0	6.0	6.0	6.0	6.0	6.0
945	47	47	46	40	2.7	2.3	2.0	1.3	4.5	4.0	4.5	1.0	1.7	3.3	2.0	1.0	6.0	0.7	6.0	6.0	1.9	3.4	3.5	3.5	1.3	1.7	0.3	3.0	3.0	3.0	3.0	3.0

Note.

A = Time A
B = Time B
C = Time C
D = Time D

APPENDIX J
CORRELATIONAL MATRIX

TIME A

	FITNESS	PAIN SEVERITY	LIFE CONTROL	AFFECTIVE DISTRESS	SUPPORT	GENERAL ACTIVITY	FREQUENCY	INTENSITY	DURATION
FITNESS	1.000								
PAIN SEVERITY	-0.400	1.000							
LIFE CONTROL	0.257	-0.245	1.000						
AFFECTIVE DISTRESS	-0.277	0.496	-0.768 *	1.000					
SUPPORT	-0.403	0.241	0.352	-0.209	1.000				
GENERAL ACTIVITY	0.049	-0.044	-0.008	-0.253	0.116	1.000			
FREQUENCY	-0.018	0.449	-0.310	0.308	0.117	-0.068	1.000		
INTENSITY	-0.355	0.173	-0.186	0.053	-0.258	0.003	0.236	1.000	
DURATION	0.195	0.033	0.468	-0.453	0.156	0.111	0.104	0.505	1.000

TIME B

	FITNESS	PAIN SEVERITY	LIFE CONTROL	AFFECTIVE DISTRESS	SUPPORT	GENERAL ACTIVITY	FREQUENCY	INTENSITY	DURATION
FITNESS	1.000								
PAIN SEVERITY	0.343	1.000							
LIFE CONTROL	-0.314	-0.427	1.000						
AFFECTIVE DISTRESS	0.136	0.469	-0.776 *	1.000					
SUPPORT	-0.227	-0.013	0.206	-0.219	1.000				
GENERAL ACTIVITY	0.256	0.447	-0.323	0.355	-0.018	1.000			
FREQUENCY	0.149	0.197	-0.052	-0.191	0.030	-0.073	1.000		
INTENSITY	0.028	0.011	0.349	-0.318	-0.112	0.108	0.495	1.000	
DURATION	-0.157	0.013	0.239	0.032	0.137	0.372	0.254	0.491	1.000

APPENDIX J CONTINUED

TIME C							
	FITNESS	PAIN SEVERITY	LIFE CONTROL	AFFECTIVE DISTRESS	SUPPORT	GENERAL ACTIVITY	FREQUENCY INTENSITY DURATION
FITNESS	1.000						
PAIN SEVERITY	-0.129	1.000					
LIFE CONTROL	0.170	-0.098	1.000				
AFFECTIVE DISTRESS	-0.160	0.206	-0.808 *	1.000			
SUPPORT	-0.145	0.263	0.565 *	-0.427	1.000		
GENERAL ACTIVITY	0.129	0.145	0.262	-0.136	0.036	1.000	
FREQUENCY	0.276	0.054	0.332	-0.410	0.385	-0.083	1.000
INTENSITY	-0.184	0.547	0.246	-0.182	0.611 *	0.292	0.557 * 1.000
DURATION	-0.330	0.395	0.465	-0.202	0.391	0.384	0.068 0.523 * 1.000
TIME D							
	FITNESS	PAIN SEVERITY	LIFE CONTROL	AFFECTIVE DISTRESS	SUPPORT	GENERAL ACTIVITY	FREQUENCY INTENSITY DURATION
FITNESS	1.000						
PAIN SEVERITY	-0.048	1.000					
LIFE CONTROL	0.034	0.367	1.000				
AFFECTIVE DISTRESS	-0.155	0.438	-0.745 *	1.000			
SUPPORT	-0.161	-0.108	0.402	-0.133	1.000		
GENERAL ACTIVITY	0.174	0.025	0.209	-0.154	0.060	1.000	
FREQUENCY	0.000	0.223	-0.158	0.180	0.317	-0.216	1.000
INTENSITY	-0.245	0.344	0.348	-0.104	0.362	-0.035	0.652 ** 1.000
DURATION	-0.311	0.281	0.108	-0.141	0.179	-0.034	0.538 * 0.763 * 1.000

* - 01 ** - 001

Appendix B
Descriptive Statistics For Raw Data

GROUP	MEAN	Time A			Time B			Time C			Time D		
		STD. DEV.	SKW.-NESS	RUR-TOSS	MEAN	STD. DEV.	SKW.-NESS	RUR-TOSS	MEAN	STD. DEV.	SKW.-NESS	RUR-TOSS	MEAN
Pain	1 44.8	25.62	0.10	3.46	35.20	26.97	-0.01	3.32	35.9	24.46	-0.18	3.71	37.9
	2 46.3	24.82	0.34	3.48	46.00	24.38	0.44	3.47	46.2	24.81	0.47	3.77	46.8
Pain Severity	1 2.50	0.67	-0.00	3.10	1.50	0.86	1.09	4.43	1.20	0.83	1.13	3.87	1.60
	2 2.60	0.56	0.20	3.00	1.90	0.88	0.03	2.37	1.80	0.81	0.54	2.97	1.80
Late	1 4.32	1.10	-1.63	7.76	6.16	1.25	-0.61	2.15	4.26	1.40	-1.30	6.60	2.55
Control	2 4.23	1.44	-1.30	6.00	6.23	1.15	-0.20	2.23	3.99	1.30	-0.59	2.77	3.50
Alternative Diet	1 2.40	1.25	0.29	3.93	2.60	1.25	0.22	1.79	2.30	1.42	1.00	4.62	2.70
	2 2.90	1.43	1.31	4.56	3.00	1.29	0.13	3.16	2.70	1.70	0.66	3.63	3.00
Support	1 4.10	1.17	0.24	1.19	4.30	1.39	-0.09	1.68	4.30	1.35	-0.13	1.73	4.10
	2 3.80	1.40	0.20	2.39	4.00	1.34	-0.49	2.49	4.00	1.90	-0.60	2.99	3.90
General Activity	1 3.00	0.66	-0.36	4.90	2.90	0.56	0.50	2.39	2.70	0.60	0.56	2.73	2.80
	2 3.10	0.55	-1.19	6.15	3.10	0.51	0.57	2.04	2.80	0.71	1.30	2.60	2.90
Frequency	1 1.30	0.79	0.16	1.20	1.00	0.70	0.06	2.16	0.90	0.74	0.44	2.73	0.80
	2 1.70	0.80	0.50	2.65	1.00	0.97	1.10	2.87	1.30	0.95	0.42	2.61	1.40
Intensity	1 2.40	0.65	0.09	2.04	1.81	0.97	-1.20	3.86	1.55	1.19	0.27	3.69	1.34
	2 2.60	0.76	-1.63	0.86	2.07	1.26	-0.96	2.43	2.63	1.35	-0.25	2.73	2.44
Duration	1 6.40	4.50	0.51	1.76	5.30	4.23	0.20	1.70	4.50	4.60	1.00	3.00	4.00
	2 6.20	4.70	1.34	0.90	6.70	5.31	0.50	1.80	6.90	5.90	0.79	3.34	6.70

Group 1 = Treatment group
Group 2 = Control group

Note 3.

Standard error for duration Group 1 = 0.66
Standard error for duration Group 2 = 0.72

Note 4.

Standard error for intensity Group 1 = 1.20
Standard error for intensity Group 2 = 1.40

Appendix L

Tests of Assumptions

Repeated measures analysis of variance requires that the data be normally distributed (Tabachnick & Fidell, 1989). While graphical tests provide a good visual cue to non-normality, with a small or moderate sample size, it is difficult to tell whether the non-normality is real or apparent, because of considerable sampling error (Stevens, 1986). Non-graphical tests were therefore used to assess normality.

A quick check for outliers can be found in Table L-1.

Stevens (1986) has suggested that a Z-score equal to, or greater than, 3, should be considered an outlier. As can be seen in Table L-1, none of the Z-scores for either group appears to reach the status of an outlier.

The statistical tests used to assess normality follow the recommendations of Wilk, Shapiro, and Chen (1968). When dealing with sample sizes ranging from 10 to 50, Wilk and his colleagues (1968) found the Shapiro-Wilk statistic, along with skewness and kurtosis coefficients, to be powerful in detecting departures from normality.

Shapiro-Wilk statistics are presented in Table L-2. As can be seen, Life Control for the treatment group at Time A is significantly non-normal (Shapiro-Wilk = .74, $p = .01$), as is Duration at Time D (Shapiro-Wilk = .78, $p = .01$). Table L-1 Intensity at time A is significantly non-normal for the control group (Shapiro-Wilk = .72, $p = .01$). Turning to Table L-3 one can examine in greater detail the nature of these deviations.

By dividing skewness and kurtosis coefficients by their respective standard

TABLE L-1

Z SCORES

GROUPS	ID	FITNESS	PAIN SEVERITY	LIFE CONTROL	NEGATIVE AFFECT	SUPPORT	GENERAL ACTIVITY	FREQUENCY	INTENSITY	DURATION
TIME A										
TREATMENT	64	-0.15	-1.24	0.17	-0.18	-0.72	-2.17	-0.38	0.97	1.02
	457	1.10	-0.94	1.52	-1.22	-0.98	1.78	-1.01	0.61	1.57
	648	0.59	0.56	0.16	0.04	-1.23	-0.80	-0.63	-0.33	-0.76
	752	-0.11	-0.04	-0.74	0.78	0.13	-0.80	1.26	-0.69	-0.42
	159	0.75	0.56	0.16	0.26	1.32	-0.50	1.26	0.73	1.46
	941	0.12	-1.98	0.16	-1.66	-0.12	0.26	-1.01	-1.75	-1.09
	269	-0.77	0.56	0.16	0.56	0.99	0.41	1.26	-0.69	0.02
	63	-1.56	1.45	-2.56	2.04	-0.97	0.57	0.25	-0.38	-1.09
	771	1.76	-0.40	0.16	-0.70	-0.72	0.72	-1.26	1.79	0.46
	644	-1.16	0.56	0.62	-0.18	1.33	-0.35	0.00	-0.21	-0.20
	262	-0.57	0.56	0.16	-0.26	0.99	0.11	-1.01	-0.80	-0.98
CONTROL	651	-1.46	0.15	-0.15	0.05	0.63	-0.39	-0.87	0.54	-0.16
	267	0.39	-1.06	-0.15	0.55	0.34	0.71	-1.17	-1.08	-0.68
	252	-0.25	0.15	-0.50	-0.44	0.13	0.16	-0.19	0.54	0.16
	158	-0.98	1.89	1.24	-0.86	0.84	-0.20	0.11	-0.27	2.15
	544	0.95	-1.58	0.19	-1.30	-1.30	0.53	0.11	0.68	0.89
	70	-0.50	-0.54	0.89	-0.16	-0.09	0.02	-1.17	-2.16	-1.10
	767	1.88	0.15	0.54	0.55	-0.59	-2.22	0.31	0.54	-0.57
	964	-0.05	0.67	-2.24	2.17	-1.52	-0.02	1.09	0.68	-0.74
	445	-0.03	0.15	0.19	-0.86	1.56	1.44	1.78	0.54	0.05

TABLE L-1 CONTINUED

Z SCORES

GROUPS	ID	FITNESS	PAIN SEVERITY	LIFE CONTROL	NEGATIVE AFFECT	SUPPORT	GENERAL ACTIVITY	FREQUENCY	INTENSITY	DURATION
TIME B										
TREATMENT	64	0.07	-0.75	1.04	-0.66	-0.43	-0.97	-0.01	1.22	1.14
	457	0.65	0.61	-0.49	1.27	-0.22	1.71	-0.44	0.19	1.61
	648	1.14	-0.75	0.28	0.30	-0.72	-0.98	-1.44	-1.86	-1.23
	752	-0.51	-1.22	0.28	-0.88	-0.22	-0.26	-0.01	-0.01	-0.52
	159	0.68	0.76	-2.02	1.56	1.23	-0.08	1.42	-0.01	0.07
	941	0.30	-0.75	0.66	-1.18	0.00	0.81	-0.44	0.20	-0.87
	269	-0.58	-0.05	1.43	-1.40	1.01	-0.26	0.42	1.02	1.13
	63	-1.66	-0.75	-0.10	-0.66	-1.44	-1.33	0.99	0.50	-0.73
	771	1.64	2.27	-0.87	1.04	-1.44	1.53	-0.44	0.20	0.43
	664	-1.16	0.61	0.66	0.30	1.01	-0.44	1.42	0.51	0.19
	262	-0.58	0.41	-0.87	0.08	1.23	0.28	-1.45	-1.86	-1.23
CONTROL	651	-1.54	-1.37	0.58	-0.26	0.51	1.77	-0.74	0.42	0.90
	267	0.38	-0.34	-0.29	0.52	-1.50	-0.88	-0.74	1.20	-0.99
	252	-0.23	0.10	-1.60	1.76	-0.01	0.29	-0.74	-1.15	-0.30
	158	-0.87	-1.37	1.45	-0.57	0.51	-0.88	-0.02	0.18	1.68
	544	0.98	1.12	-1.16	0.21	-1.50	-0.88	2.04	0.26	0.22
	70	-0.56	0.10	-0.29	0.75	-0.53	-0.88	-1.05	-1.93	-1.16
	767	1.84	-0.34	0.58	-1.58	0.51	-0.29	1.01	0.97	-0.47
	964	-0.04	1.56	1.01	-1.04	1.48	-0.88	-0.02	-0.37	-0.85
	445	0.04	0.54	-0.29	0.21	0.51	0.88	0.29	0.42	0.97

TABLE 1-1 CONTINUED

Z SCORES

GROUPS	ID	FITNESS	PAIN SEVERITY	LIFE CONTROL	NEGATIVE AFFECT	SUPPORT	GENERAL ACTIVITY	FREQUENCY	INTENSITY	DURATION
TIME D										
TREATMENT	64	0.05	0.11	0.33	0.27	-0.82	-1.39	1.50	1.93	1.51
	457	0.63	-0.52	0.69	0.27	0.16	1.87	-0.33	0.54	1.29
	648	1.13	-0.79	0.69	0.53	-0.29	0.58	-0.33	-0.27	0.42
	752	-0.33	-1.14	1.04	0.53	-0.29	0.40	0.28	-0.27	0.63
	159	0.50	0.11	-1.48	0.27	1.42	-0.09	2.11	0.34	-0.10
	941	0.25	-1.14	0.69	-1.43	-0.29	1.21	-0.95	-1.09	-0.85
	269	-0.83	1.89	-0.03	2.22	0.68	-0.03	0.28	1.36	1.72
	63	-1.70	-0.25	-1.84	-1.17	-1.49	0.40	-0.95	-1.90	-0.85
	771	1.70	1.26	-0.75	-0.83	-1.34	-0.58	-0.95	-1.90	-0.85
	664	-1.16	-0.52	1.05	-0.32	1.20	1.87	-0.33	-0.27	-0.42
	262	-0.25	1.00	-0.39	-0.32	1.05	-1.39	-0.33	-0.27	-0.42
CONTROL	651	-1.52	1.20	0.29	0.59	0.07	-0.09	-0.44	0.91	0.24
	267	0.34	-0.43	-1.44	1.57	-1.62	-0.36	-1.42	-2.11	-1.18
	252	-0.14	-0.03	0.29	-0.38	-1.11	-0.45	-0.93	-0.38	-0.29
	158	-0.96	-0.96	1.15	-1.13	0.74	-0.09	0.05	1.34	1.83
	544	0.83	1.97	-0.87	0.19	-0.95	0.99	1.04	0.31	0.45
	70	-0.55	-0.43	-1.44	0.42	0.43	-1.44	1.53	-0.47	1.12
	767	1.91	-0.43	0.87	-1.13	0.23	-1.44	0.55	0.31	-0.47
	964	0.01	-0.30	0.29	1.00	1.10	0.45	0.55	0.48	-0.95
	445	0.08	-0.96	0.87	-1.13	1.10	1.52	-0.93	-0.38	-0.74

Maha CV ($p=0.01$) = 23.0

Table L-2

Shapiro - Wilk Statistic for Univariate Normality

Variable	<u>Time A</u>		<u>Time B</u>		<u>Time C</u>		<u>Time D</u>	
	1	2	1	2	1	2	1	2
Fitness	0.98	0.98	0.98	0.98	0.98	0.97	0.99	0.97
Pain Severity	0.88	0.93	0.88	0.95	0.89	0.96	0.92	0.83
Life Control	0.74 *	0.88	0.96	0.95	0.86	0.93	0.89	0.86
Affective Distress	0.97	0.88	0.94	0.99	0.91	0.95	0.92	0.91
Support	0.88	0.97	0.90	0.89	0.90	0.90	0.94	0.91
General Activity	0.95	0.89	0.93	0.85	0.95	0.82	0.95	0.94
Frequency	0.88	0.93	0.93	0.88	0.93	0.96	0.83	0.96
Intensity	0.97	0.72 *	0.82	0.92	0.85	0.98	0.89	0.92
Duration	0.90	0.89	0.93	0.93	0.87	0.81	0.78 *	0.94

* P = 0.01

Note.

TABLE L-3

Skewness and Kurtosis Coefficients for non-normal variables

Variable	<u>Treatment Group</u>		<u>Control Group</u>	
	Skewness	Kurtosis	Skewness	Kurtosis
Life Control				
Time A	-1.63 *	7.76	-1.38 *	6.00
Intensity				
Time A	0.09	2.84	-1.63 *	4.86
Duration				
Time D	1.03	2.24	0.74	0.22

*p < 0.01

errors, one may determine appropriate Z-scores for the coefficients, and the probability that these Z-scores are significantly different from a standard mean of 0 (Tabachnick and Fidell, 1989). Table L-3 presents, for those deviating variables, skewness scores, along with their Z-score equivalents, and kurtosis coefficients, along with their Z-score equivalents.

As can be seen in Table L-3 Life Control at time A, for the treatment group, is significantly negatively skewed (Skewness = -1.63, $p = .005$), as well as significantly leptokurtic (Kurtosis = 7.76, $p = .0001$). Intensity at time A for the control group is also significantly negatively skewed (Skewness = -2.26, $p = .01$). Duration for the treatment group at time D was neither significantly skewed (Skewness = 1.03, $p = .03$) nor significantly abnormal on kurtosis (Kurtosis = 2.24, $p = .28$) at the .01 level of significance.

According to Stevens (1986), skewed distributions have very little effect on either the level of significance or the power of the F-test. In addition, while leptokurtosis may influence both alpha and power, its effects are negligible if only one variable, for one group, is leptokurtotic (Stevens, 1986). The decision was made, therefore, to retain the variables Life Control and Intensity, and to refrain from imposing transformations upon them.

Repeated measures analysis of variance also requires that the variance-covariance matrices within each cell be homogeneous, and that all covariances in the pooled matrix be equivalent (Tabachnick & Fidell, 1989). The former assumption was assured by conducting the Box's M test, for each analysis. Using a conservative significance level of .001, as recommended by Hakstain, Roed, & Lind (1979), we

conclude that the variance-covariance matrices within each cell in each design are sampled from the same population variance-covariance matrix. The latter assumption was assessed using Mauchly's Sphericity test. Two variables (Fitness and Support) failed to meet the sphericity assumption, and therefore these variables were analyzed using the less restrictive MANOVA, treating each repetition of the dependent variable in the analysis as separate dependent variables.

END

1 5 0 4 9 1

FIN