AN ASSESSMENT OF TWO CHIROPRACTIC MANAGEMENT PROGRAMMES WITH RESPECT TO FREQUENCY OF TREATMENT OF MIGRAINE HEADACHES

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BY

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"I, Mark Spencer Whittle, declare that this dissertation represents my own work, both in conception and execution."

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ABSTRACT

The purpose of this investigation was to determine the most beneficial chiropractic management programme with respect to frequency of treatments for migraine headaches. It was hypothesised that an intensive treatment programme would be most effective.

Thirty four (34) patients were accepted into this single blind, randomised trial, however only thirty (30) patients were compliant. Only patients diagnosed with migraine were accepted. The sample group was drawn from a population of migraine sufferers from the greater Durban area, and they were randomly divided into the two treatment groups.

Both treatment groups received chiropractic adjustments to fixations found in the cervical and thoracic spines, as well as soft tissue massage to the above-mentioned areas. The only difference was the treatment periods, where the intensive treatment group was treated nine times in three weeks and the conservative treatment group was treated nine times in nine weeks. Both groups were re-evaluated after a six week follow-up period.

Only subjective measurements were taken, all tabulated weekly by each patient in the headache diaries provided.
Weekly averages were obtained for frequency, severity and duration of migraine headaches and total pill consumption per week was also statistically analysed. Demographic data was also obtained for migraine sufferers from a headache questionnaire.

Statistical analysis of the collected data was performed using the Wilcoxon Signed Rank test and the Mann-Whitney U test, both used at 95% level of significance. Results were displayed graphically and the key results were also tabulated.

The Mann-Whitney U test was used to determine significant difference between the two treatment groups at pre and post-treatment stages as well as after the follow-up period. No significant difference was noted between the two groups, therefore the null hypothesis was rejected.

The Wilcoxon Signed Rank test used to determine any significant change within each group, made use of the same figures as before. For the intensive treatment group, significant changes were noted with respect to frequency of migraine headaches and pill consumption during the treatment period. Continued significance was noted through to the end of the follow-up as well.
For the conservative group, significant difference is noted for duration and frequency, only during the treatment period.

The results indicate that both treatment groups have positive effects on migraine headaches however, neither treatment group fared better than the other. There is evidence to support the cervicogenic model for migraine, but further research is required to identify those patients in which this aspect is a primary factor.
UITTREKSEL

Die doel van hierdie navorsing was om die mees voordelige chiropraktiese hanteringsprogram ten opsigte van gereeldheid van behandeling vir migraine hoofpyne te bepaal. Die veronderstelling het daarop gedui dat 'n intensiewe behandelingsprogram die mees doeltreffend sou wees.

Vier-en-dertig (34) pasiënte is aanvaar vir hierdie enkel, ewekansige toets, waarvan slegs dertig (30) voldoen het aan die voorvereistes. Slegs pasiente wat gediagnoseer is as migrainelyers is aanvaar. Die toetsgroep is verkry van die groter Durban gebied en is lukraak in twee behandelingsgroepes verdeel.

Albei groepe is chiroprakties behandel vir fiksasies in die servikale en thorakale werwels. Sagte weefsel massering van bogenoemde areas is ook gedoen. Die enigste verskil was die tydperk van behandeling, waar die intensiewe behandelingsgroep nege behandeling binne drie weke ontvang het en die konserwatiewe behandelingsgroep nege behandeling binne nege weke. Beide groepe is na 'n tydperk van ses weke vir 'n opvolg behandeling geher-evalueer.

Slegs subjektiewe waarnemings is deur elke pasiënt self weekliks gemeet met behulp van tabelle op die voorsiene
hoofpyn dagboek.

Weeklikse gemiddeldes is verkry ten opsigte van gereeldheid, graad van pyn en duur van die migraine hoofpyne. Die totale pil/medikasie verbruik per week is ook geanaliseer met behulp van statistieke. Demografiese inligting van elke migraine lyer is ook verkry deur middel van 'n hoofpyn vraelys.

Die Wilcoxon Signed Rank toets en die Mann-Whitney U toets, is gebruik vir statistieke analisie van die ingevorderde data met 'n graad van 95% gewig betekenis-inhoud. Die resultate word grafies en per tabel vertoon.

Die Mann-Whitney U toets is gebruik om die betekenis-inhouds verskil tussen die twee groepe tydens die pre-en-post behandelingstadia te bepaal, asook vir die opvolg tydperk. Geen betekenis-inhouds verskil is gevind tussen die twee groepe nie, en die nul veronderstelling is dus verwerp.

Die Wilcoxon Signed Rank toets is gebruik om die betekenis-inhouds verandering van die twee groepe te toets. Die selfde syfers as vir die Mann-Whitney U toets is gebruik. 'n Betekenisvolle verandering met betrekking tot gereeldheid van migraine hoofpyne en pilverbruik tydens die behandelings tydperk is gemerk gevind in die intensiewe behandelingsgroep.
'n Volgehoue betekenisvolle verandering is gevind tot aan die einde van die opvolg behandelings tydperk.

Met die konserwatiewe behandelingsgroep was die betekenisvolle veranderings met betrekking tot gereeldheid en duur van die hoofpyne slegs sigbaar tydens die behandelings tydperk.

Die uitslag dui daarop dat behandeling vir beide die groepe 'n positiewe uitwerking op migraine hoofpyn gehad het, maar dat nie een van die groepe beter gevaar het as die ander een nie. Daar is bewyse ter ondersteuning van die "cervicogenic" model vir migraine, maar verdere navorsing word benodig om die pasiënte vir wie die aspek 'n belangrike faktor is, te identifiseer.
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LIST OF ABBREVIATIONS.

C = conservative group;
I = intensive group;
DUR = duration of migraine headaches;
FRE = frequency of migraine headaches;
PILL = pill consumption; and
SEV = severity of migraine headaches.
INTRODUCTION

According to recent studies, it has been shown that about 8% of the population suffer from migraine headaches (Blau, 1984: 65). It has been noted by numerous practitioners (McCullem et al., 1994), that chiropractic care is beneficial for the treatment of migraine headaches, but the frequency of treatment has never been considered when designing a management plan.

Due to the rising costs of medical treatment, the most beneficial management plan would not only aid the rate of recovery of the patient, but would also be saving him money. Migraine accounts for a loss of at least 3 million working days in Britain each year, and in the United States more than $500 million is spent annually by people seeking relief from headache (Wilkinson, 1982: 13). If the hypotheses are correct, then a further saving of money on analgesics and a reduction of the side effects following their use is to be expected.

In showing the advantage of an initial intensive series of treatments, over a more conservative treatment plan, and by using a long term follow up evaluation it will be possible to demonstrate the benefits of increased frequency in the treatment of migraine.
By determining the most beneficial treatment plan, and the publication of the relevant findings, chiropractors will be able to treat migraine sufferers with greater confidence and the patient will thus receive the most beneficial treatment their chiropractor can offer.
CHAPTER ONE

THE PROBLEM

AND ITS SETTINGS
1.1 PROBLEM STATEMENT.

The purpose of this investigation is to assess the effect of frequently as opposed to infrequently applied chiropractic treatment in the management of migraine headaches, in terms of the patient's perception, in order to determine the more effective of the two approaches.
1.2 STATEMENT OF THE SUBPROBLEMS.

1.2.1 The First Subproblem.
The first subproblem is to evaluate the effect of frequently applied chiropractic intervention in the treatment of migraine headaches in terms of the frequency, severity and duration of attacks, and the response of the subject's to analgesics, in order to determine the subject's perception of the efficacy of the treatment.

1.2.2 The Second Subproblem.
The second subproblem is to evaluate the effect of infrequently applied chiropractic intervention in the treatment of migraine headaches in terms of the frequency, severity and duration of attacks, and the response of the subject's to analgesics, in order to determine the subject's perception of the efficacy of the treatment.

1.2.3 The Third Subproblem.
The third subproblem is to integrate the data from subproblems one and two, in order to determine the most beneficial management plan.
1.3 THE HYPOTHESES.

1.3.1 Hypothesis One.

It is hypothesized that more frequently applied chiropractic treatment will result in a marked decrease in the subject's perception of pain, in terms of frequency, severity and duration of migraine headache, and there will also be a decrease in the requirement for analgesics.

1.3.2 Hypothesis Two.

It is hypothesized that less frequently applied chiropractic treatment will result in a slight decrease in the patient's perception of pain, in terms of frequency, severity and duration of migraine headache, and there will also be a slight decrease in the requirement for analgesics.

1.3.3 Hypothesis Three.

It is hypothesized that subject's who receive a greater frequency of treatments will have statistically significantly greater benefit than those receiving less frequent treatment.
1.4 THE DELIMITATIONS.

1. This study will only consider those patients who are positively diagnosed with migraine.

2. Patients must have had migraine headaches for at least one year, and suffer from at least one attack per month.

3. This study will only consider the effect of varying the frequency of chiropractic treatment of migraine headaches.

4. Patients must be at least 14 years of age in order to complete the necessary documentation.

5. This study will not consider the influence of allopathic treatment of migraine headaches, only the effect that chiropractic treatment has on the amount of medication needed to manage the migraine headaches.
1.5 THE ASSUMPTIONS.

1. It is assumed that a large enough sample size will be obtained (viz., 30 patients).

2. That chiropractic treatment is beneficial to migraine sufferers.

3. That the patient's general lifestyle remains unchanged for the duration of the study as alteration of diet and exercise may influence the migraine attacks.

4. That patients will only continue to use allopathic medicine for the treatment of their migraine headaches, and that the dose, type and frequency of use will be indicated on the headache diaries.

5. It is assumed that patients will log their migraine headaches as required.
1.6 **DEFINITIONS.**

**Frequency Of Chiropractic Treatment:**
This refers to the number of treatments received by patients per week depending on which group they belong. The conservative group will be treated once a week for nine weeks, while the intensive group will be treated three times per week for three weeks.

**Chiropractic Treatment:**
For the purpose of this study, chiropractic treatment refers to soft tissue therapy as well as cervical and thoracic adjustments.

**Soft Tissue Therapy:**
For the purpose of this study, soft tissue therapy refers to soft tissue massage, effluerage in order to loosen the muscular tissue in the neck and upper thoracic area.

**Adjustment:**
The chiropractic adjustment is a specific form of direct articular manipulation using either long or short leverage techniques with specific contacts and is characterized by a dynamic thrust of controlled velocity, amplitude and direction (Gatterman, 1990: 405).
Migraine Headache:
Episodic, typically unilateral or bitemporal head pain with or without focal visual or neurological disturbances (Wilkinson, 1982: 13).

Severity Of Migraine Headache:
The intensity of the migraine headache, when at its worst, as noted by each patient on a numerical rating scale, after each attack in their headache diary.

Duration Of Migraine Headache:
The length of time that the patient has the migraine headache as noted in the pain diary, in hours.

Frequency Of Migraine Headache:
How often the migraine headache occurs, again as noted in each patient's pain diary, on the relevant day.

Subluxation:
Subluxation is an aberrant relationship between two adjacent articular structures that may have functional or pathological sequelae, causing an alteration in the biomechanical and/or neurophysiological reflections of these articular structures, their proximal structures, and/or body systems that may be directly or indirectly affected by them (ACA, 1987, as cited in Gatterman, 1990: 415).
CHAPTER TWO

REVIEW OF THE

RELATED LITERATURE
2.0 INTRODUCTION.

Migraine is one of over twenty types of headache and accounts for 6% of all headaches (Blau, 1987: 3). About 8% of the population are migraine sufferers (Blau, 1984: 65). Berkow (1987: 1355) defines migraine as a paroxysmal disorder characterized by recurrent attacks of headache, with or without associated visual and gastro-intestinal disturbances.

2.1 ETIOLOGY AND INCIDENCE.

The exact cause is not known, but two main theories exist. There is evidence to suggest a genetically transmitted functional disturbance of cranial circulation (Moskowitz, 1989: 181). The prodromal symptoms, to be discussed later, occur following intracerebral vasoconstriction while during the headache phase there is dilation of the extra-cranial arteries (Moskowitz, 1989: 181). A family history is obtained in more than 50% of cases (Berkow, 1987: 1355).

Headaches are among the most prevalent yet poorly understood problems in clinical neurology (Edwards and Bouchier, 1991: 849). Headaches may develop in association with hypertension, seizures, stroke or without a recognizable pathophysiology such as with migraine and cluster headaches.
Cranial blood vessels are implicated as the most important source for all vascular headaches and are innervated by sensory fibres which arise from ganglia innervating the forehead, scalp and neck. Sensory fibres contain vasoactive neuropeptides which become released from peripheral and central terminations to mediate vasodilation and pain, respectively. The presence of vascular headache implies activation of this final common pain pathway which has been termed the trigeminovascular system. The existence of such a system: a) clarifies certain pain patterns which develop following stimulation of cephalic blood vessels, b) suggests a mechanism to explain the referral of pain to the forehead and c) provides a mechanism to explain the action of certain anti-migraine drugs (Moskowitz, 1989: 181-193).

There is increasing support for the neural theory of migraine, initially submitted some 100 years ago by Gower (cited in Blau, 1984: 64). According to the theory, the symptoms that occur the day before the headache implicate the hypothalamus. Secondly, the visual aura is not encountered in cerebrovascular disease and during the headache phase prodromal symptoms may still exist. Sleep often resolves attacks and the hangover symptoms often last as long as the preceding headache.
These symptoms resemble the prodrome and are not accompanied by a headache. The argument is that the vascular manifestations are secondary to neural stimulation since the meningeal and extra-cranial vessels are innervated by autonomic fibres (Blau, 1984: 64).

Pupilometric studies carried out on migraineurs, show an increased adrenergic response due to a receptor hypersensitivity. Since sympathetic fibers of pupil dilator and those forming the external and internal carotid plexuses are the same post-ganglionic fibers from the superior cervical ganglion, the finding of a sympathetic reflex hypofunction may be ascribed to post-ganglionic denervation with hypersensitivity of alpha and beta vascular receptors (Boiardi et al., 1988: 420).

The relationship between migraine and intracranial vascular malformations (IVM) was researched by a correlative study of the headache characteristics with the type and location of the IVM. A high prevalence (47%) of migraine type headaches and a strong positive correlation (88.8%) between the site of IVM and the side of the pain was found. Although highly suggestive this is not conclusive evidence that a pathophysiologic relationship exists between these two entities (Monteiro et al., 1993: 563-5).
Vernon, (1985) proposed the concept of vertebrogenic migraine which is a conglomeration of three previously accepted models; 1) autonomic/vascular; 2) neurogenic, and 3) somatic

The vertebrogenic migraine model came about through the apparent short-comings of the existing theories, thus by taking the strong points of each model and explaining them through the involvement of the cervical and thoracic spine, one would be able to appreciate Vernon's model.

The Autonomic/vascular model was a two stage model which involved firstly vasoconstriction and then vasodilation of the intracerebral and extracerebral vasculature, respectively. This was later expanded to a three stage model with firstly vasoconstriction, then cerebral ischemia and lastly vasodilation.

Oleson et al., (1981) was responsible for showing certain problems with the model using intracarotid injection of xenon. They included:

1) A diminution of cerebral blood flow in the occipito-parietal region which spread forward, with the possibility of affecting the entire hemisphere.

2) That oligemia was the only blood flow abnormality found
during the headache phase.

3) That the oligemia was not sufficient to cause ischemic symptoms, which therefore could not be due to vasospasm.

Vernon (1985:21) isolated some other problems with the vascular model which had previously not been explained. Firstly he noted that temporal artery dilatation can occur without headache, as seen in fevers and that the opposite temporal artery is often dilated without pain or headache on that side. He then questioned the reason for primarily unilateral symptoms. Furthermore, he noted that common migraine appears without aura, and therefore, possibly without vasoconstriction.

The Neurogenic model was given greater validity following the work of Oleson et al., (1981), with respect to the proposal of the migraine cascade, in particular the timing of the spreading oligemia.

It was concluded through research done by Lance, et al., (1983, 258-265) that following low frequency stimulation of the locus ceruleus, of cats and monkeys, vasoconstriction of the internal cerebral vasculature resulted. Increasing the frequencies of stimulation, produced vasodilatation of the external carotid artery and its branches, and these changes were predominately ipsilateral. Therefore since the locus
ceruleus is a central analogue of the sympathetic ganglia, any of the accepted migraine triggers could influence locus ceruleus activity and thus initiate the migraine cascade.

Lastly the Somatic model (as cited in Vernon, 1985: 21) focuses attention on the cranio-cervical musculoskeletal structures and their interaction with spinal segmental and cranial neuromeres. The symptoms involved are caused initially by cervical joint dysfunction which leads to a reflex disturbance of autonomic efferent activity resulting in an imbalanced vaso-control. These symptoms closely resemble those of the migraine prodrome.

The somatic dysfunctions that pertain to migraine involve somato-somato reflex disturbances which create local paraspinal muscular hypertonicity, tenderness, pain and restricted joint motion.

The vertebrogenic model of migraine etiology would operate as follows according to Vernon, (1985). Vertebral lesions of the cranio-cervico-thoracic spine results in pain and altered afferent impulses, which creates alteration of local segmental reflexes which allows for central facilitation and hypersensitivity of autonomic efferent pools. This creates the environment for vasoconstriction of the cerebral circulation which causes raised levels of circulating
catecholamines which causes activation of the trigeminal pain transmission pathway. This ultimately results in stimulation and triggering of the locus ceruleus.

Two operational descriptions are offered to explain the above model. The first relies on involvement of dysfunction in the upper cervical spine, (C0-C1) which produces local pain and fixations. This causes the upper cervical neuromeres to become facilitated and thus there is a reduction in the inhibitory effect of the descending pain pathways (at least one of which arises in the locus ceruleus). This then increases the facilitation of the second order neurons in the spinal tract of the trigeminus which leads to transient stimulation of the locus ceruleus, which, upon reaching threshold levels, results in focal and spreading vasoconstriction in the intracerebral circulation. In time this then spreads to a secondary vasodilation of the extra-carotid circulation accompanied by cranial pain being mediated by the ipsilateral trigeminal nerve.

The second description involves somatic dysfunction of the cervico-thoracic junction and upper thoracic spine, (C7-T4) causing joint pain and fixation. This leads to local segmental facilitation of the lateral horn cells of the spinal cord resulting in a central excitatory state. This will consequently lead to sustained neural discharges from
the sympathetic chain, which will result in transient cerebral ischemia. Under potentiated conditions, a threshold level might be reached, sufficient to create cerebral ischemia which would then activate the migraine cascade.

Increased catecholamine levels would result from the sub-threshold central excitation and would have direct effects on platelet membranes (Vernon, 1985: 22).

Support of these operational descriptions, and therefore for the vertebrogenic model of migraine comes from many researchers, including Sachse et al., (1982), who showed that the most frequent movement restrictions in migraineurs are CO-Cl, the cervico-thoracic junction and the first rib. Others like Parker et al., (1978) have shown that spinal manipulation for the treatment of migraine headaches is of some benefit.

After proposing the vertebrogenic model for migraine, Vernon (1985: 23) went on to describe three categories of migraineurs. The first are those who would fit into the vertebrogenic model, and it was proposed that this group would derive primary benefit from spinal manipulative therapy (SMT). The second category would be those in whom the vertebrogenic component was secondary, but synergistic and it was proposed that they could benefit from SMT. The third
category involves those in whom the vertebrogenic component was not active, and for whom manipulation would not provide any benefit.

Other causative factors, or factors that result in an increased predisposition include chocolate, cheese, alcohol, citrus fruit, Chinese food (excessive sodium intake), or patients taking oral contraceptives, especially higher dose oestrogen pills.

Hormonal contraception is particularly contra-indicated for patients with classical migraine. The headaches develop during the adaptational period of oral contraception and the migrainous attacks occur in the premenstrual period or at the beginning of menstruation which refer to an oestrogen withdrawal character. It has been suggested that vasoconstriction, which has existed during the use of the pills, changes over to relative vasodilation in this period and that the extent of the changes is dependent on the oestrogen content of the tablets (Karsay, 1990: 185).

Stress and anxiety may initiate or lead to perpetuation of attacks, thus an attack of migraine should be seen as the product of a patient's abnormal susceptibility and his/her reaction to a variety of internal and external stimuli (Sacks, 1970).
Migraine may occur at any age although it is most commonly diagnosed between the ages of 10 and 30, with the first attack occurring before the age of 40 in 90% of patients (Silberstein and Lipton, 1993: 179). Migraine occurs more often in women than in men, the generally accepted ratio being 3:1. The latest figures available according to Sheftell, (1993: 874) report that 17.6% of females and 5.7% of males are sufferers. Remission after 50 years of age is not uncommon (Berkow, 1987: 1355).

2.2 CLASSIFICATION.

The variants of migraine included in most text contain the four principal forms of migraine.

Classical: Visual or sensory symptoms precede or accompany the migraine headache.

Common: No visual or sensory features only headaches, nausea, vomiting and photophobia.

Hemiplegic: Prolonged headache lasting hours or days, followed by hemiparesis which recovers slowly over days.
Basilar: Occipital headache preceded by vertigo, diplopia, dysarthria with or without visual or sensory symptoms (Edwards and Bouchier, 1991: 851).

According to the latest classification of the International Headache Society (IHS), (1988) migraine is now simplified into two types, those with aura (classic) and those without aura (common) (Silberstein and Lipton, 1993: 179).

2.3 CLINICAL FEATURES.

Clinically the features can be divided into signs and symptoms and these differ in all 5 phases as described by Blau (1987: 3-17). Not all symptoms are experienced by each patient, and different patients also experience various combinations of the symptoms mentioned below.

Phase 1) Prodrome: Symptoms are usually subtle and they evolve slowly, and only experienced in about 60% of patients. This phase presents as either excitatory or inhibitory, and an average of 6 features per patient are observed beginning on average 3 hours before the aura or headache.
- mental state: irritable or withdrawn
- altered behaviour: hyperactive or clumsy
- altered appearance: flushed or pale
- neurological signs: photophobia or dysphasia
  phonophobia or slow thinking
- muscular signs: stiff or weak
- alimentary signs: craving food or anorexia
  diarrhoea or constipation
- altered fluid balance: thirst or fluid retention

Phase 2) Aura: This phase is characteristic of classic migraine. Visual disturbances are most commonly reported with scintillating scotoma and fortification. Dysphasia and nausea have been noted in this phase which usually lasts between 10 and 30 minutes.

Phase 3) Headache: This phase starts with an awareness in the head and becomes a discomfort, then develops into a headache which increases in severity. The pain is described by most to be throbbing or pressing. Associated symptoms include those found in the prodrome with possible neck pain included. Headache duration ranges from 2-72 hours.

Phase 4) Resolution: This usually occurs with sleep, but if the severity is not too bad, gradual fading of symptoms under the control of analgesics can occur.
Phase 5) Postdrome (recovery): Patients feel "washed out" or "drained", with accompanying lowered mood, impaired concentration, increased physical tiredness and an intolerance to food. Symptoms last for up to 24 hours after the headache has gone.

Evidence for an autonomic dysfunction has been researched, since significant autonomic manifestations are known to accompany the onset of pain. In a study to compare the cardiovascular reflex responses of common migraine and cluster headaches, it was observed that the emphasis of impairment was placed on the adrenergic sympathetic system (Boiardi et al., 1988: 422). Involvement of vestibulocochlear functions as revealed in a study on 94 migraine sufferers in the pain free interval, showed only 4 pathological readings on electronystagmographic investigations. It is concluded that vestibular dysfunctions, in particular those of central origin, are not common in migraine with the possible exception of basilar artery migraine (Schlake et al., 1989: 275).

Kudrow and Sutkus, (1979 as cited in Melzack and Wall 1984: 285) attempted to find psychological test patterns which were characteristic of headache patients. Patients studied in headache clinics typically have elevated scores on neurotic scales, however it is unclear whether these abnormalities
precede the headaches or, as is more likely, are the consequence of them. There are rarely significant differences among types of headache when other variables such as age, sex, frequency, duration and severity of pain are controlled. Three patterns, or groups of patients with similar test profiles have been identified:

A) Males and females with migraine and cluster headaches, essentially have normal profiles,

B) muscle contraction headaches and mixed migraine-muscle contraction headaches, both males and females showing borderline depression and

C) post-traumatic cephalalgia and conversion headaches, both males and females showing mild to moderate depression.

Group A also differs from the other groups in showing less elevation on the neurotic scales, this is contributed to the fact that although migraine is regarded as a chronic disposition, there may be periods of remission and there are more frequent and longer pain-free periods.

Mortimer et al., (1993: 429) found a significant association between atopic children and childhood migraine, with the strongest relationship being with those suffering from rhinitis.

Range of motion testing of the cervical spine in migraine
patients was recently researched testing neck rotation, lateral flexion and flexion and extension. Although there was no significant difference between treatment and control groups for each particular motion test, there was a significant difference when two or more abnormalities in combination was found. It was proposed that musculoskeletal dysfunction of the neck is a contributing factor to the etiology of migraine and tension headache (Kidd and Nelson, 1993: 567).

2.4 DIAGNOSIS OF MIGRAINE.

Not all severe headaches are migraine, despite a common lay misconception, and not all migraine are unilateral and throbbing (Day, 1990: 1797).

Helpful diagnostic features include the presence of an aura or prodrome; hemicranial distribution (typically one sided); the throbbing nature of the headache; associated systemic upset with nausea, vomiting or photophobia; and provocation by typical trigger factors (Day, 1990: 1797).

An accurate history and physical examination are necessary to diagnose migraine, but the differential diagnosis of benign headaches should only begin after ruling out more threatening conditions including infections, neoplasm and ocular
disorders. Barbuto, (1988), (as cited in Gatterman, 1990: 252); lists the following warning signs of organic disease:

1. Episodic fainting in relation to headache;
2. abrupt onset of a severe headache for the first time;
3. neurological abnormalities associated with headache;
4. elevation of body temperature associated with headache;
5. onset of headache after age 50;
6. headaches associated with an increase in pressure, eg: coughing;
7. history of recent blood pressure elevation;
8. personality change;
9. headache following head trauma;
10. disturbance of pulse rate or respiration;
11. constant sensory disturbances, and
12. onset of visual field defects.

The physical and neurological examinations are typically normal although transient neurological deficits may occur during an attack. Laboratory studies are not routinely done since there is no specific test for migraine. X-rays are also not usually taken in a typical case, however if other pathologies are suspected, skull X-ray or CT scans are necessary. Cervical X-ray studies offer little information in typical migraine, but are useful in cases of severe muscle spasm or neck trauma. Clinical discretion is vital in this regard.
Early studies of electroencephalography (EEG) suggested a significant incidence of dysrhythmia in migraine (Weil, 1952 as cited in Drake et al., 1988: 201). More recent studies have shown no specific patterns in vascular headache patients, although interictal and ictal slowing has been reported in vertebrobasilar migraine. Migraine and tension type headache patients don't differ significantly on an EEG spectral analysis, and because of this the EEG is of limited use in the evaluation of headache patients (Drake et al., 1988: 201).

2.5 DIFFERENTIAL DIAGNOSIS.

Because migraine is a multifactoral neurovascular response and there are no specific diagnostic tests to confirm a diagnosis the clinician must rely almost exclusively on the patient's history, which at the best of times can be misleading.

Included in the differentials are:

A) Functional Headaches:

1. Tension Type Headache: a non-specific, band-like sensation which is stress induced; most common of all headaches (Edwards and Bouchier, 1991: 851).


3. Cluster Headache: episodes of excruciating unilateral
facial pain, typically occurring in young men (Sjaastad, 1988: 667). Ipsilateral autonomic symptoms of nasal congestion, rhinorrhea, and lacrimation are commonly present (Mckenna, 1988: 173). This makes for an important differential diagnosis, however since they rarely occur in females a distinction can be made (Manzoni et al., 1988: 37).


7. Hangover: facial features normally evident, breath may be a give away (Gatterman, 1990: 251).

8. Eye strain: visual acuity may require attention, glasses may be necessary (Gatterman, 1990: 251).

9. Psychogenic: patient complains with pain which, "hurts all the time, is felt all over and is the worst pain ever", yet does not respond to treatment (Gatterman, 1990: 251).

B) Pathological Headaches:

1. Brain Tumor: rapidly progressive signs with signs and symptoms of raised intercranial pressure (Gatterman,
2. Brain Abscess: similar to the above, with neck rigidity (Smith, 1993).

3. Subdural Haematoma: following trauma characterised by a dull, diffuse pain (Smith, 1993).

4. Hypertension: elevated blood pressure 200/110 mmHg (Gatterman, 1990: 251).

5. Sentinel/Aneurismal: due to small bleeds, usually felt in vertex, worst headache ever (Gatterman, 1990: 251).

6. Giant Cell Arteritis: stroke may occur so signs and symptoms are vital (Gatterman, 1990: 251).

2.6 **TREATMENT.**

2.6.1 **Medical Treatment.**

Drugs used in the treatment of migraine includes analgesics and anti-nauseants for treating the symptoms, however there are more specific "antimigraine" drugs like dihydroergotamine and Sumatriptan. This is still considered to be the most effective treatment for an acute attack (Smith, 1994). Many drugs have been developed over the years, designed specifically for the prevention of migraine, but as yet no prophylactic treatment has shown to be significantly effective, although beta blockers are used with good results (Smith, 1994).
Hoffert, (1994: 633) reports that recent research refutes the vascular theory of migraine, and thus new insights into the phenomenon of migraine are leading to alternative treatments.

A study was performed to evaluate a long term course of non-invasively treated chronic headache. A total of 1015 adult patients with primary diagnosis of vascular migraine participated in the study, investigating symptom frequency and severity over a 36 month period after receiving treatment. Treatment consisted of either relaxation training, biofeedback, microelectrical therapy or multinodal treatment. 793 patients were finally accepted through admissibility questionnaire, and the patients were randomly assigned to treatment groups. They received either short-term intervention (less than 15 treatments), or long-term intervention of more than 15 treatments. Results indicate that all treatments significantly reduced frequency and intensity of cephalalgia. Repeated measure analysis indicated that grouping variables of biofeedback treatment and receiving more than 15 treatments best predicted successful intervention (Reich, 1989: 41).

An investigation of 130 chronic headache sufferers was done to assess the level of awareness and potential impact of dietary risk factors. It revealed that 75% were aware of possible food intake and headache connections and that less
than 50% were made aware of it by medical professionals. It was also noted that this awareness did not prompt significant changes in the subjects' diet (Guarnieri et al., 1990: 25).

A behavioral package was used to shape and maintain the adherence of subjects with vascular headache to a program of aerobic exercise training. Repeated measures of exercise behavior were examined through the use of a bi-directional changing criterion design. Repeated measures of headache activity were also collected. Results demonstrated a functional relationship between the behavioral package and exercise adherence. The results also indicated clinically significant collateral reductions in vascular headache activity (Fitterling et al., 1988: 19).

2.6.2 Chiropractic Treatment.

The role of chiropractic manipulative therapy in relieving headaches has long been proclaimed by practitioners McCullum et al., 1994). Many case reports and review papers have documented such therapeutic successes. Wight, (1978) cited in Vernon 1982: 109) reported 74.7% overall improvement in a sample of 87 migraine subjects and Rose, (1973) cited in Vernon 1982: 109) reported 62.5% improvement in 17 patients.

Hopkins's (1988: 337) view of manipulations for the treatment
of migraine is less supportive. He notes that no adequate trials exist which confirm the claims of manipulative therapists and that he further suggests that the successes obtained by manipulations are related to muscle stretching rather than realignment of joints. He concluded that the neck is rarely implicated in the production of migrainous headaches. Other researchers conclude that cervical manipulations have no more than a placebo effect on migraine (Blau, 1987: 200).

In a retrospective and prospective study performed by Vernon, (1982) it was demonstrated that all values of severity, duration and frequency decreased significantly to the 99% confidence limit. No significant correlation existed between the length of time, as a function of the number of treatments, and the extent of relief as measured by any of the variables.

The efficacy of cervical manipulation for migraine has been evaluated by only a few researchers (Parker et al., 1978). In a 6 month trial, volunteers suffering from migraine were randomly allocated to 3 treatment groups. One group received cervical manipulation performed by a physiotherapist, the other by a chiropractor, while the control group received mobilization. For the entire group, migraine symptoms were significantly reduced. No difference in outcome was found
between the three groups with respect to reducing frequency, duration or induced disability of migraine attacks, but chiropractic patients did report a greater reduction in severity of pain associated with their attacks (Parker et al., 1978).

Parker et al., (1980) asked the question, "why does migraine improve during a clinical trial?" They answered the question tentatively. It was suggested that a "trial effect", due to all the attention received, might have played a role. The 28% overall improvement during the trial was attributed to two possible causes:

1. That some form of manipulation or mobilisation of the cervical spine is an effective treatment for migraine. It was noted that it was unlikely that it could be an effective treatment for all patients since the reduction in migraine frequency during the trial was slight.

2. That the subjects underwent a natural and progressive remission from the time of starting treatment. It has been observed that the natural history of migraine may include periods of time when it would appear that the patient has been "cured", although unless the patient is over the age of 50 spontaneous remission is rare.
The idea of migraine management rather than treatment appears to be the best approach and because of the multifactoral aspect of migraine, it should be multifaceted including:
1. Explanation and reassurance;
2. avoidance of trigger factors and lifestyle modifications;
3. treatment of acute attacks, and

2.7 CONCLUSION.

Upon review of the literature vast gaps exist in our knowledge, especially regarding the role of chiropractic in the management of migraine headache.
CHAPTER THREE

MATERIALS AND

METHODS
3.0 INTRODUCTION.

This chapter deals with the data, how it was obtained and treated, it also looks in great detail at the materials used during the research, the methodology and the way the research was run.

3.1 THE DATA.

3.1.1. The Primary Data.

This was obtained directly from the patients in the form of:
- Case history.
- Physical exam and cervical regional.
- Patient's headache diary.
- Admissibility Questionaire to be completed by patients on initial consultation.

3.1.2. The Secondary Data.

This data was required to substantiate and explain certain problems and was obtained from the following:
- books
- journal articles
- periodicals
- pamphlets
Other secondary data needed in order complete sub-problem 3, will be obtained from the results and conclusions of sub-problems 1 and 2.

3.2 CRITERIA FOR ADMISSIBILITY.

Only fully completed headache diaries of the subjects were utilised in order to satisfy all the pre-set criteria for admissibility of the data. If the data, at any time, did not satisfy these criteria, the subject, along with the results, were dismissed from the study.

3.3 RESEARCH METHODOLOGY.

The following steps were followed in the execution of this study:
- Subjects were recruited either by advertising in local newspapers or by word of mouth.
- Each subject was screened using an admissibility questionnaire (appendix A), case history (appendix B) as well as having a full physical exam (appendix C) and cervical regional (appendix D) in order to positively diagnose each subject.
- All subjects had a cervical spine X-ray series taken so that it was possible to ascertain whether each subject had any contra-indications to adjustive therapy.
- Each subject, on being accepted into the sample, were asked to sign an informed consent form (appendix E), after they had been informed as to what was required from them.
- Next the subject was randomly placed into either the conservative or intensively treated groups.
- This was achieved by using a dice to divide the sample into two groups of 15 subjects each, one group receiving the intensive treatment program (I), the other, the more conservative management plan (C). Six variations of groups of four are possible so as to obtain equal numbers of each group in the final sample. The variations were as follows:
  1. IICC
  2. CCII
  3. ICIC
  4. CICI
  5. ICCI
  6. CIIC
- By throwing the die eight times a total of 32 places were available. The results were: 5, 1, 6, 2, 2, 5, 3, 6. This lead to the allocation of: ICCI IICC CIIC CCII CCII ICCI ICIC CIIC.
- As a subject was accepted into the sample he/she was placed into a group according to the allocation.
Subluxations located in the cervical or thoracic regions were recorded for both groups as determined by motion palpation, as described by Schafer and Faye (1989: 101-9) and taught at Technikon Natal Chiropractic College.

These subluxations were appropriately adjusted, depending on the type of fixation. (Typical techniques used were the cervical rotary and lateral break, thoracic cross-bilateral and thoracic anterior adjustment, as described in Szaraz, (1990) and taught at Technikon Natal Chiropractic College).

The intensive group received three treatments per week, for three weeks, while the conservative group received one treatment per week, for nine weeks.

Both groups then had a follow-up period of six weeks.

Both groups were required to document their migraine headaches for the duration of the study on the headache diaries provided (Appendix F).

During the follow-up period subjects were phoned weekly in order to remind them to continue to fill in their headache diaries as instructed.

Data collected and secured from the subjects headache diaries was statistically analysed using the Wilcoxon Signed Rank Test in order to establish if significant difference exists over time within each treatment group.

The same data was also analysed using The Mann-Whitney U Test to test for a significant difference between the two treatment groups at critical time intervals during the
treatment and follow-up period.
- All data was tested to the 5% level of significance, i.e.: $P = 0.05$, or to 95% confidence levels.
- The data was analysed using a computer software programme; STATGRAPHICS PLUS VERSION 6, supplied by Manugistics Inc.

3.4 SPECIFIC TREATMENT OF SUBPROBLEM 1.

The first sub-problem was to evaluate the effect of frequently applied chiropractic intervention in the treatment of migraine headaches in terms of the frequency, severity and duration of attacks, and the response of the subjects to analgesics, in order to determine the subject's perception of the efficacy of the treatment.

3.4.1 Data Needed.
The data needed for testing the hypothesis of sub problem one was the subjective response of the subjects in the group as noted in the headache diary (Appendix F).

3.4.2 How The Data Was Secured.
The data was collected from the subjects treated by the author at Technikon Natal Chiropractic Day Clinic, after the follow-up period.
3.5 SPECIFIC TREATMENT OF SUBPROBLEM 2.

The second sub-problem was to evaluate the effect of infrequently applied chiropractic intervention in the treatment of migraine headaches in terms of the frequency, severity and duration of attacks, and the response of the subject's to analgesics, in order to determine the subject's perception of the efficacy of the treatment.

3.5.1 Data Needed.
The data needed for testing the hypothesis of sub problem two was the subjective response of the subjects in the group as noted in the headache diary (Appendix F).

3.5.2 How The Data Was Secured.
The data was collected from the subjects treated by the author at Technikon Natal Chiropractic Day Clinic, after the follow-up period.

3.6 SPECIFIC TREATMENT OF SUBPROBLEM 3.

The third sub-problem was to integrate the data from sub-problems one and two, in order to determine the most beneficial management plan.
3.6.1 Data Needed.
This data is of a numerical form as well as the statistical interpretations and conclusions drawn from sub-problems 1 and 2.

3.6.2 How The Data Was Secured.
The data was obtained by referring to the interpretations made and conclusions drawn from sub-problems 1 and 2.

3.7 GENERAL REMARKS.
In total 34 subjects were assessed and accepted into the sample and by the end of the final follow-up period there were 4 subjects who, for various reasons, were non-compliant. All the data was collected by the author and treated with the usual confidentiality.
CHAPTER FOUR

THE RESULT
4.0 INTRODUCTION.

This chapter covers the results obtained following the statistical analysis of the figures secured from the headache diaries, ie:
- duration of migraine headaches
- frequency of migraine headaches
- pill consumption
- severity of migraine headaches

The results are tabulated and/or graphically displayed, and significant differences are also indicated. Demographic data is also tabulated at the end of the chapter.

The first figure indicated in each table represents the mean for that group for the specific week of the study, which is indicated by the figure in brackets. For example; the mean for the duration of migraine headaches for the intensive group at the end of the treatment period is indicated as follows 3.90 (4), since the treatment period ended on week four and for the conservative group it was week number ten, this also applies for the results of the statistical tests.
4.1 DURATION OF MIGRAINE HEADACHE.

TABLE 1. COMPARISON BETWEEN THE TWO TREATMENT GROUPS WITH RESPECT TO DURATION (IN HOURS) OF MIGRAINE HEADACHES AT SPECIFIC TIME INTERVALS.

<table>
<thead>
<tr>
<th></th>
<th>PRE-TREATMENT</th>
<th>POST-TREATMENT</th>
<th>FOLLOW-UP</th>
</tr>
</thead>
<tbody>
<tr>
<td>INTENSIVE TREATMENT</td>
<td>5.73 (1)</td>
<td>3.90 (4)</td>
<td>4.67 (10)</td>
</tr>
<tr>
<td>CONSERVATIVE TREATMENT</td>
<td>4.43 (1)</td>
<td>2.40 (10)</td>
<td>5.10 (16)</td>
</tr>
</tbody>
</table>

MANN-WHITNEY U TEST (at 5% level of significance)

DUR I (1) * DUR C (1) Non significant
DUR I (4) * DUR C (10) Non significant
DUR I (10) * DUR C (16) Non significant

WILCOXON SIGNED RANK TEST (at 5% level of significance)

INTENSIVE TREATMENT GROUP:
DUR (1) * DUR (4) Non significant (0.1)
DUR (1) * DUR (10) Non significant
DUR (4) * DUR (10) Non significant

CONSERVATIVE TREATMENT GROUP:
DUR (1) * DUR (10) Significant (0.05)
DUR (1) * DUR (16) Non significant
DUR (10) * DUR (16) Non significant (0.12)
FIGURE 1. COMPARISON OF DURATION (IN HOURS) OF MIGRAINE HEADACHES AT KEY TIME INTERVALS.

FIGURE 2. COMPARISON OF THE TWO TREATMENT GROUPS WITH RESPECT TO DURATION OF MIGRAINE HEADACHES.
4.2 FREQUENCY OF MIGRAINE HEADACHES.

TABLE 2. COMPARISON BETWEEN THE TWO TREATMENT GROUPS WITH RESPECT TO FREQUENCY OF MIGRAINE HEADACHES PER WEEK AT SPECIFIC TIME INTERVALS.

<table>
<thead>
<tr>
<th></th>
<th>PRE-TREATMENT</th>
<th>POST-TREATMENT</th>
<th>FOLLOW-UP</th>
</tr>
</thead>
<tbody>
<tr>
<td>INTENSIVE TREATMENT</td>
<td>2.60 (1)</td>
<td>0.87 (4)</td>
<td>1.00 (10)</td>
</tr>
<tr>
<td>CONSERVATIVE TREATMENT</td>
<td>2.60 (1)</td>
<td>1.47 (10)</td>
<td>1.67 (16)</td>
</tr>
</tbody>
</table>

MANN-WHITNEY U TEST (at 5% level of significance)

FREQ I (1) * FREQ C (1) Non significant
FREQ I (4) * FREQ C (10) Non significant
FREQ I (10) * FREQ C (16) Non significant

WILCOXON SIGNED RANK TEST (at 5% level of significance)

INTENSIVE TREATMENT GROUP:
FREQ (1) * FREQ (4) Very significant (0.002)
FREQ (1) * FREQ (10) Significant (0.007)
FREQ (4) * FREQ (10) Non significant

CONSERVATIVE TREATMENT GROUP:
FREQ (1) * FREQ (10) Significant (0.05)
FREQ (1) * FREQ (16) Non significant (0.06)
FREQ (10) * FREQ (16) Non significant
FIGURE 3. COMPARISON OF FREQUENCY (PER WEEK) OF MIGRAINE HEADACHES AT KEY TIME INTERVALS.

FIGURE 4. COMPARISON OF THE TWO TREATMENT GROUPS WITH RESPECT TO FREQUENCY OF MIGRAINE HEADACHES.
4.3 PILL CONSUMPTION.

TABLE 3. COMPARISON BETWEEN THE TWO TREATMENT GROUPS WITH RESPECT TO PILL CONSUMPTION PER WEEK AT SPECIFIC TIME INTERVALS.

<table>
<thead>
<tr>
<th></th>
<th>PRE-TREATMENT</th>
<th>POST-TREATMENT</th>
<th>FOLLOW-UP</th>
</tr>
</thead>
<tbody>
<tr>
<td>INTENSIVE TREATMENT</td>
<td>5.53 (1)</td>
<td>0.77 (4)</td>
<td>1.60 (10)</td>
</tr>
<tr>
<td>CONSERVATIVE TREATMENT</td>
<td>2.13 (1)</td>
<td>2.77 (10)</td>
<td>3.40 (16)</td>
</tr>
</tbody>
</table>

MANN-WHITNEY U TEST (at 5% level of significance)

PILL I (1) * PILL C (1) Significant (0.05)
PILL I (4) * PILL C (10) Non significant (0.16)
PILL I (10) * PILL C (16) Non significant

WILCOXON SIGNED RANK TEST (at 5% level of significance)

INTENSIVE TREATMENT GROUP:
PILL (1) * PILL (4) Very significant (0.004)
PILL (1) * PILL (10) Significant (0.05)
PILL (4) * PILL (10) Non significant (0.14)

CONSERVATIVE TREATMENT GROUP:
PILL (1) * PILL (10) Non significant
PILL (1) * PILL (16) Non significant
PILL (10) * PILL (16) Non significant
FIGURE 5. COMPARISON OF PILL CONSUMPTION (PER WEEK) FOR MIGRAINE HEADACHES AT KEY TIME INTERVALS.

FIGURE 6. COMPARISON OF THE TWO TREATMENT GROUPS WITH RESPECT TO PILL CONSUMPTION FOR MIGRAINE HEADACHES.
4.4 SEVERITY OF MIGRAINE HEADACHES.

TABLE 4. COMPARISON BETWEEN THE TWO TREATMENT GROUPS WITH RESPECT TO SEVERITY (OUT OF 10) OF MIGRAINE HEADACHES AT SPECIFIC TIME INTERVALS.

<table>
<thead>
<tr>
<th></th>
<th>PRE-TREATMENT</th>
<th>POST-TREATMENT</th>
<th>FOLLOW-UP</th>
</tr>
</thead>
<tbody>
<tr>
<td>INTENSIVE TREATMENT</td>
<td>3.67 (1)</td>
<td>2.07 (4)</td>
<td>2.13 (10)</td>
</tr>
<tr>
<td>CONSERVATIVE TREATMENT</td>
<td>3.13 (1)</td>
<td>2.77 (10)</td>
<td>3.40 (16)</td>
</tr>
</tbody>
</table>

MANN-WHITNEY U TEST (at 5% level of significance)

SEV I (1) * SEV C (1) Non significant
SEV I (4) * SEV C (10) Non significant
SEV I (10) * SEV C (16) Non significant (0.16)

WILCOXON SIGNED RANK TEST (at 5% level of significance)

INTENSIVE TREATMENT GROUP:

SEV (1) * SEV (4) Significant (0.05)
SEV (1) * SEV (10) Non significant (0.09)
SEV (4) * SEV (10) Non significant

CONSERVATIVE TREATMENT GROUP:

SEV (1) * SEV (10) Non significant
SEV (1) * SEV (16) Non significant
SEV (10) * SEV (16) Non significant
**FIGURE 7.** COMPARISON OF THE SEVERITY (OUT OF 10) OF MIGRAINE HEADACHES AT KEY TIME INTERVALS.

**FIGURE 8.** COMPARISON OF THE TWO TREATMENT GROUPS WITH RESPECT TO THE SEVERITY OF MIGRAINE HEADACHES.
4.5 DEMOGRAPHIC DATA.

TABLE 5.

<table>
<thead>
<tr>
<th></th>
<th>INTENSIVE TX. GROUP</th>
<th>CONSERVATIVE TX. GROUP</th>
<th>COMBINED SAMPLE</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAMPLE SIZE</td>
<td>15</td>
<td>15</td>
<td>30</td>
</tr>
<tr>
<td>SEX RATIO (M:F)</td>
<td>8:7</td>
<td>1:14</td>
<td>9:21</td>
</tr>
<tr>
<td>MEAN AGE (YEARS)</td>
<td>35.40</td>
<td>37.87</td>
<td>36.63</td>
</tr>
<tr>
<td>DURATION OF MIGRAINE</td>
<td>15.8</td>
<td>13.5</td>
<td>14.65</td>
</tr>
<tr>
<td>(YEARS)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FAMILIAL TENDANCY</td>
<td>66.6%</td>
<td>73.3%</td>
<td>70%</td>
</tr>
<tr>
<td>NO. WITH COMMON MIGRAINE</td>
<td>10 (66.7%)</td>
<td>11 (73.3%)</td>
<td>21 (70%)</td>
</tr>
<tr>
<td>DEGENERATIVE CHANGES OF CERVICAL SPINE</td>
<td>46.6%</td>
<td>53.3%</td>
<td>50%</td>
</tr>
<tr>
<td>LOSS OF CERVICAL LORDOSIS</td>
<td>53.3%</td>
<td>40%</td>
<td>46.7%</td>
</tr>
</tbody>
</table>
CHAPTER FIVE

DISCUSSION
5.0 **INTRODUCTION.**

This chapter deals firstly with the discussion of the results, looking how they relate to each subproblem, giving possible explanations for them and with the aid of references will attempt to give possible reasons for the changes that took place. Secondly, the demographic data is discussed with reference to previous studies. Then some of the problems encountered are discussed and some possible changes are provided for future studies. Lastly, conclusions and recommendations are made with respect to this project and for further research in this field.

5.1 **DISCUSSION OF RESULTS.**

5.1.1 **Subproblem 1.**

It was hypothesized that frequently applied chiropractic treatment (intensive group) would result in a marked decrease in the subject's perception of pain, in terms of frequency, severity and duration of migraine headache, and that there would also be a reduction in the need for analgesics for treatment of migraine headache.

For this hypothesis the Wilcoxon Signed Rank test was used to determine significant difference at the 95% confidence level.
The results in this regard show no significant difference with respect to duration of migraine headache.

For severity, significant change was noted following treatment, however there was no significant difference between pre-treatment and post follow-up levels. This indicates that there was a significant decrease in pain intensity following treatment, but after six weeks follow-up the change that was effected no longer existed. A possible explanation for this trend is that while under treatment a "trial effect" as described by Parker et al. (1980) might have been at work, since considerable attention was paid to subjects during the treatment period, but during the follow-up period the only contact was telephonic communication once a week.

Frequency of headache, and pill consumption for this group followed a different trend with similar patterns. There was a very significant difference between pre and post-treatment figures, well above the 95% confidence. When considering the change over the entire period, pre-treatment to end of follow-up, there was still a significant difference noted, however no change was seen from the end of the last treatment to the end of the follow-up. This indicates that most change occurs during the treatment period and that, although there is no further improvement, the change remains as such for at
least six weeks following treatment.

The close resemblance of these two trends can be related by the fact that as the frequency of headaches decreases, so will the medication required to treat the migraine headaches, because there are less migraines to treat.

5.1.2 Subproblem 2.

The second hypothesis was that infrequently applied chiropractic treatment (conservative group) would result in a slight decrease in the subject's perception of pain, in terms of frequency, severity and duration of migraine headache, and that there would also be a slight reduction in the need for analgesics for treatment of migraines.

The Wilcoxon Signed Rank test was again used to determine significant difference at the 95% confidence level. Two different trends were set in this group. Firstly, with respect to severity of headaches and pill consumption, no significant difference was noted throughout the study.

Secondly, there was a significant change following treatment for duration and frequency of headaches. No other significant differences were noted, indicating that some change had
occured, but that it did not last through the follow-up period of six weeks. The relevance of the results obtained shows that there was significant change effected by the treatment, but that this change was not long lasting, in fact not even for six weeks following the last treatment. In order to explain this, it could be the "trial effect" as elaborated by Parker et al., (1980).

5.1.3 Subproblem 3.

The third hypothesis states that a greater frequency of treatment is most beneficial in the treatment of migraine headaches.

In order to evaluate this hypothesis the Mann-Whitney U test was used at the 95% level of confidence. To be able to accept or reject the hypothesis, the aspects that make up the two treatments must first be dealt with.

Firstly, with respect to the frequency of migraine headaches as compared between the two groups, no significant difference was noted for pre and post treatment figures as well as the follow-up values. It is desirable that the pretreatment figures are similar, this shows even distribution between the two treatment groups.
The post treatment and follow-up readings should ideally show a significant difference between the two groups, with the intensive treatment group showing greater improvement, however at the current level of significance this is not the case for any of the test parameters.

The one significant difference was that of pill consumption at the pre-treatment stage. This however is not helpful since both groups should be the same before the treatment begins. In an attempt to explain this irregularity, it can only said that when designing this aspect of the research, the researcher was faced with the problem of having to combine all medication used in the treatment/management of migraine headaches into one category, "pill consumption". This included analgesics, anti nausiant and specific migraine medication, which often involved a cocktail of tablets. There was also the fact that in the one group, two particular patients would take in excess of twelve tablets in a day for their headaches, which often lasted two to three days. These individuals made for unusually high readings for the conservative group. The other periods of the research were not significantly different.

To relate all this information back to the third hypothesis, it is evident that no significant difference exists between the two groups and therefore the hypothesis is rejected.
An explanation for the changes that occurred can best be illustrated using the vertebrogenic migraine model (Vernon, 1985). To look at individual patients' results, it would be apparent that there were certain patients, in both groups but to a greater extent the intensive group, who showed total remission of any headaches for the duration of the study, including the six-week follow-up period. This would indicate that certain patients (23%), who had been suffering up to five headaches per week, were now following treatment, not having a headache for six to eight weeks. It would have been interesting to be able to follow these patients up after a longer time period, however, time constraints limited the follow-up period.

These patients would fit perfectly into Vernon's first group, those in which the vertebrogenic component of migraine is a primary role player. Spontaneous remission is not considered since none of these patients were older than fifty years old.

The vast majority of patients (60%), could be considered to fit into the second group where there was a degree of the vertebrogenic component, but it does not play a primary role rather a part of the multifactorial aspect of migraine etiology. These patients showed definite benefit, specifically with respect to frequency of headaches, again better results were noted in the intensive group, but no
significant difference, was made.

The remaining patients (17%) showed little or no improvement during and after treatment, this group according to Vernon would have triggers that were not of vertebrogenic origin.

Other findings in this research which would strengthen the model proposed by Vernon, (1985) include the presence of motion restrictions found almost exclusively in the upper cervical spine (C0-C2) and at the cervicothoracic junction, which would fit into his two descriptions offered for the etiology of vertebrogenic migraine.

5.2 DISCUSSION OF DEMOGRAPHIC DATA.

In considering the demographic data, it was clear that this study demonstrated similar findings to those noted by Sheftell, (1993: 874), with respect to sex ratio, and it also compliments research by Parker et al., (1978: 591) with respect to other data concerning mean age, average duration of migraine and percentage of patients with common migraine. The figures for percentage breakdown of those suffering with migraine with an aura, was noted to be considerably higher than previous research done by Blau, (1984: 64) who states that classical symptoms are only noted in 10% of migraine patients, as apposed to 30% in this research.
The figures obtained for familial tendency of migraine were significantly higher than those noted in Berkow (1987: 1355). It is the opinion of the researcher that this study was representative of the greater population when considering migraine headaches.

An interesting finding, not previously discussed in the literature, was that of the number of patients with a marked loss of cervical lordosis, as seen on the lateral X-ray view. It was remarkable to the researcher that so many of the patients screened for migraine had such reduced cervical lordosises. Since the average age of subjects in the combined sample was slightly over 36 years, bony degeneration could not be seen to be a major factor, and since the changes noted were primarily of soft tissue origin and little evidence of bony degeneration was seen, it can only be suggested that altered cervical muscle tone was responsible.

This finding was merely noted and not investigated further, since follow-up X-rays would have been required to be able to ascertain whether the loss of lordosis was in any way corrected after treatment, and the necessary funds were not available. The researcher does however feel that further investigation is indicated in this regard.
5.3 PROBLEMS ENCOUNTERED.

Certain problems that arose in carrying out the research included non-compliance. This aspect of the study was anticipated and allowances were made when the sample size was selected. The main problem in this regard was the relatively long time period involved especially with the conservatively treated group, where a diary was to be kept for sixteen weeks.

The other main problem was that patients had problems with scheduled appointments, once again the conservative group was mostly to blame. It was specified in the proposal that this group would receive one treatment per week for nine weeks, in hope that they would be approximately one week apart, however this was not entirely the case. If a patient was treated on a Monday one week, and on Friday the following week, although he has been treated once a week, the treatments are in fact nearly two weeks apart. This could have increased the chance of a significant difference, but none were present.

The last problem that existed was that of patients not listing their headaches accurately in the diaries. This was corrected by checking through all diaries with the patient involved at the end of the follow-up period. Data was also checked for legibility, which made data capture far easier.
5.4 CONCLUSIONS AND RECOMMENDATIONS.

In conclusion, the results obtained strengthen Vernon's vertebrogenic model and the researcher notes that although no significant difference exists between the two treatment groups, the intensive treatment group did receive a greater benefit. It was noticed that the increased frequency of treatment did reduce the number of migraine attacks experienced by the patients, and that if a migraine did start, because of the cascade of events in causing the headache, the treatment had no effect on the duration and severity of the headache.

Since the patients fit so well into the three categories of Vernon's vertebrogenic migraine model, the researcher believes that further research is indicated. The diagnosis and isolation of those patients, in whom the vertebrogenic aspect of migraine etiology is primary ie; group one, would almost be like finding the "cure" for a large portion of the population who suffer from migraine headaches, through chiropractic manipulative therapy.
   In Vernon, H. Upper cervical Syndrome Chiropractic 
   diagnosis and treatment. Baltimore, Williams and 
   Wilkins. 253p.

Berkow, R.(Ed.) 1987. The Merck Manual of Diagnosis and 
   Therapy. 15th Edition. USA, Merck Sharp and Dohme 
   Research Laboratories:1351-1356.

   (5):64-65.

   and Research Aspects. London, Chapman and Hall:3-17, 
   200.

Boiardi, A., Munari, L., Milanesi, I., Paggetta, C., 
   Lamperti, E. and Bussone, G. 1988. Impaired 
   cardiovascular reflexes in Cluster headache and Migraine 
   patients: Evidence for an autonomic dysfunction. 
   Headache. 28(6):417-422.

   overview of diagnosis and management. Australian Family 


HEADACHE QUESTIONNAIRE
(Derived from Canadian Memorial Chiropractic College Migraine Trial Headache History)

Patient name

1) Age
2) Male / Female
3) Occupation
4) Onset Total duration
   Hour Weeks
5) Initial precipitating event NO YES

TYPICAL HEADACHE
6) Frequency of headache Daily / Weekly / Monthly

7) Duration of headache
   Time of onset:
   Duration:
   Hours Days

8) Severity: Rate on the following scale the Severity of the headache:
   1 2 3 4 5 6 7 8 9 10 (none) (worst)
9) Location NO YES

Date / /

5. Orbital NO YES
6. Vertex
7. Mandibular

10) 1. Neck pain / 2. Upper back pain

11) 1. Unilateral Right / Left / 2. Bilateral

12) 1. Focal / 2. Diffuse / 3. Radiating

13) Character NO YES

14) Associated Symptoms NO YES
15) Diurnal
   1. Awakes at night
   2. Morning
   3. Afternoon
   4. Evening
   5. All day
   6. Variable

16) Precipitation
   1. Hunger
   2. Tension/Stress
   3. Fatigue
   4. Weather change
   5. Movement of head/neck
   6. Certain foods

17) Aggravation
   1. Menstruation
   2. Sneezing/coughing
   3. Exertion
   4. Head/neck movements
   5. Motion
   6. Noise
   7. Light

18) Medications
   1. None
   2. Analgesic
   3. Aspirin
   4. Tylenol
   5. Flurinol
   6. Ibuprofen
   7. Abortion
   8. Ergot
   9. Cafergot
   10. Other

19) Self-Help
   1. Lying down
   2. Massage
   3. Heat
   4. Cold
   5. Food
   6. Other

20) Past Treatment

21) Previous diagnosis?

22) Past diagnosis?

23) Do you have any separate pain in:

24) Are you taking any medication?
25) Have you had any recent illnesses or operations?
   NO / YES

26) Do you have
   1. High blood pressure /
   2. Vascular disease /
   3. Neurological disease /
   4. Other diseases /

Lifestyle

27) 1. Are you married?
    /

28) Are you a smoker
    / How much?

29) Family history of headache
    1. Mother /
    2. Father /
    3. Grandparents /
    4. Sibling /
    5. Children /

30) How would you rate your job stress
    1. Low /
    2. Medium /
    3. High /
TECHNIKON NATAL CHIROPRACTIC DAY CLINIC

CASE HISTORY

Patient: ___________________________ Date #: ___________________________

File #: ____________________________

X-ray #: ____________________________

Age: _______ Sex: _______ Occupation: ____________________________

Intern: ____________________________ Signature: ____________________________

FOR CLINICIAN’S USE ONLY

Initial visit clinician: ____________________________ Signature: ____________________________

Case History:

Examination:
- Previous: TN Other
- Current: TN Other

X-ray Studies:
- Previous: TN Other
- Current: TN Other

Clinical path. lab.:
- Previous: TN Other
- Current: TN Other

Case status:
- PTT: Conditional: Signed off: Final sign out:

Recommendations:
Intern's case history

1. Source of history:

2. Chief complaint: (patient's own words)

3. Present illness:

   Location

   Onset

   Duration

   Frequency

   Pain (character)

   Progression

   Aggravating factors

   Relieving factors

   Associated S & S

   Previous occurrences

   Past treatment and outcome
4. Other complaints:

5. Past history:

   General health status

   Childhood illnesses

   Adult illnesses

   Psychiatric illnesses

   Accidents/injuries

   Surgery

   Hospitalizations
6. Current health status and life-style:
   - Allergies
   - Immunizations
   - Screening tests
   - Environmental hazards
     (home, school, work)
   - Safety measures
     (seat belts, condoms)
   - Exercise and leisure
   - Sleep patterns
   - Diet
   - Current medication
   - Tobacco
   - Alcohol
   - Social drugs

7. Family history:
   - Immediate family:
     - Age
     - Health
     - Cause of death
     - DM
     - Heart disease
     - TB
     - HBP
     - Stroke
     - Kidney disease
     - CA
     - Arthritis
     - Anaemia
     - Headaches
     - Thyroid disease
     - Epilepsy
     - Mental illness
     - Alcoholism
     - Drug addiction
     - Other
8. Psychosocial history:
   Home situation
   Daily life
   Important experiences
   Religious beliefs

9. Review of systems:
   General
   Skin
   Head
   Eyes
   Ears
   Nose/sinuses
   Mouth/throat
   Neck
   Breasts
   Respiratory
   Cardiac
   Gastro-intestinal
   Urinary
Genital
Vascular
Musculoskeletal
Neurologic
Haematologic
Endocrine
Psychiatric.
PHYSICAL EXAMINATION

Underline abnormal findings in RED and elaborate on back of relevant page, if necessary. Mark "NAD" if normal.

Patient: ___________________ File #_____

Last name  First name

Clinician: _______________ Signature: _______________

Intern: _______________ Signature: _______________

Date: _______________

Height: _______  Weight: _______  Temp: _______

Rates: Heart: _____  Pulse: _____  Respiration: _______

Blood pressure: Arms:  L / R / L

Legs:  L / R / L

General appearance:
STANDING EXAMINATION.

Minor's sign
Skin changes
Posture
erect
Adam's

Ranges of motion:

T/L spine: Flexion: 90 Fingers to floor
Extension: 50
R.lat.flex.: 30 Fingers down leg
L.lat.flex.: 30 Fingers down leg
Rot.to R.: 35
Rot.to L.: 35

Flex.

L.Rot. R.Rot.

L.lat. flex. R.lat. flex.

Ext.

/ = pain-free limitation; // = painful limitation.

Romberg's sign.
Pronator drift.
T:endelenburg's sign.
Gait.
  rhythm
  balance
  pendulousness
  on toes
  on heels
  tandem
Half squat.
Scapular winging.
Muscle tone.
Spasticity/Rigidity.
Shoulder:
skin
symmetry
ROM - glenohumeral
scapulo-thoracic
acromioclavicular
elevator
wrist
Chest measurement
inspiration
expiration
Visual acuity

Breast examination:
Inspection:
skin
size
contour
nipples
arms overhead
hands against hips
leaning forward.
Palpation:
axillary lymph nodes.

SEATED EXAMINATION.

Spinal posture
Head
scalp
skull
face
skin
Eyes
conjunctiva
sclera
eyebrows
eyelids
lacrimal gland
nasolacrimal duct
alignment
corneal reflex
ocular movement

visual fields
accommodation
iris
pupils
red reflex
optic disc

L

III IV VI

R

III IV VI
vessels
general background
macula
vitreous
lens
Ears:
auricle
ear canal
drum
auditory acuity
Weber test
Rinne test

Nose:
external
internal
septum
turbinates
olfaction
Sinuses (frontal & maxillary):
tenderness
transillumination
Mouth and pharynx:
lips
buccal mucosa
gums and teeth
roof
tongue
inspection
movement
taste
palpation
pharynx
inspection
CN X
Neck:
posture
size
swelling
scars
discoloration
hair line
**ROM:**

| Flexion:       | 45 chin to larynx  |
|               | chin to sternum    |
| Extension:    | 55 forehead parallel to floor |
| L.lat.flex:   | 40                 |
| R.lat.flex:   | 40                 |
| L.rot.:       | 70                 |
| R.rot.:       | 70                 |

**L.Rot.**

| L.lat. flex. | R.lat. flex. |

**R.Rot.**

**Ext.**

lymph nodes
trachea
thyroid
carotid arteries (thrills, bruit)

**CN V**
**CN VII**
**CN VIII (nystagmus)**
**CN IX**
**CN XI**

**THJ**

Inspection
ROM
development
Palpation
crepitus
tenderness
Cardiovascular:
  auscultation (aortic murmurs)
  Allen's test

SUPINE EXAMINATION

JVP
PHI
  auscultation heart (L.lat.recumbent)
  respiratory excursion
  percussion chest (anterior)
  breast palpation

The abdomen:
  Inspection:
    skin
    umbilicus
    contour
    peristalsis
    pulsations
    hernias (umbilical/incisional)
  Auscultation:
    bowel sounds
    bruit
  Percussion:
    general
    liver
    spleen
  Palpation:
    superficial reflexes
    cough
    light
    rebound tenderness
    deep
    liver
    spleen
    kidneys
    aorta
    intra-/retro-abdominal wall mass
    shifting dullness
    fluid wave

Acute abdomen:
  where pain began and now
  cough
  tenderness
  guarding/rigidity
  rebound tenderness
  Rovsing's sign
  psoas sign
  obturator sign
  cutaneous hyperaesthesia
  rectal exam
  Murphy's sign.
Neurological:
Dermatomes
C5
C6
C7
C8
T1
Tendon reflexes
biceps
triceps
brachioradialis
Muscle strength
C5
C6
C7
C8
T1
Coordination:
point-to-point
dysdiadochokinesia
Thorax:
Chest:
Inspection:
skin
shape
respiratory distress
rhythm (respiratory)
deepth
"effort"
intercostal/supracleavicular retraction
Palpation:
tenderness
masses
respiratory expansion
tactile fremitus
Percussion:
lungs (posterior)
diaphragmatic excursion
kidney punch
Auscultation:
breath sounds
vesicular
bronchial
adventitious sounds
crackles (rales)
wheezes (rhonchi)
voice sounds
bronchophony
whispered pectoriloquy
egophony
Male genitals and hernias.

Inspection:
- skin
- prepuce
- glans
- meatus
- nits/lice
- scrotum
- inguinal/femoral bulges

Palpation:
- penis (tenderness/induration)
- testes
- epididymis
- inguinal canal
- femoral canal
- cremasteric reflex

Auscultation:
- scrotal mass.

Peripheral vasculature:

Inspection:
- skin
- nail beds
- pigmentation
- hair loss

Palpation:
- pulses - radial, brachial, femoral, popliteal, post.tibial, dorsalis pedis
- lymph nodes - epitrochlear, femoral (horizontal & vertical)
- temperature (feet & legs)
- Manual compression test
- Retrograde filling (Trendelenburg) test
- Arterial insufficiency test

Musculoskeletal:

ROM

- hip
  - flex. 90/120
  - ext. 15
  - abd. 45
  - add. 30
  - int rot 40
  - ext rot 45

- knee
  - flex. 130
  - ext. 0/15

- ankle
  - plantar flex 45
  - dorsiflex 20
  - inversion 30
  - eversion 20

- leg length
Mental status

Appearance and behaviour:
level of consciousness
posture and motor behaviour
dress, grooming, personal hygiene
facial expression
affect

Speech and language:
quantity
rate
volume
fluency
aphasia (prn)

Mood

Thought processes (logical, relevant, organized)
Memory and attention:
orientation (time, place, person)
remote memory
recent memory
new learning ability

Higher cognitive functions:
information and vocabulary (general & specialised knowledge)
abstract thinking.

Neurological:
dermatomes
L1
L2
L3
L4
L5
B1

muscle strength
hip flexion
knee extension
ankle dorsiflexion
plantar flexion
tendon reflexes
patellar
Achilles
plantar reflex

Rectal examination:
Inspection
sacroccygeal & perianal areas
Palpation
sphincter tone
tenderness
induration
nodules
prostate
seminal vesicles
TECHNIKON NATAL CHIROPRACTIC DAY CLINIC.

REGIONAL EXAMINATION -- CERVICAL SPINE.

PATIENT: ____________________________

FILE #: ______________________________ DATE: ______________________________

INTERN/RESIDENT: ____________________________

SUPERVISING CLINICIAN: ____________________________

OBSERVATION:

Posture
Swellings
Scars
Discoloration
Hair Line
Bony and soft tissue contours

Shoulder position:
Left =
Right =

Muscle spasm
Facial expression

RANGE OF MOTION:

Flexion = 45 degrees.
Extension = 70 degrees.
L/R Rotation = 70 degrees.
L/R Lateral flexion = 45 degrees.

KEY:
/
PAINLESS LIMITATION.
// PAINFUL LIMITATION.

PALPATION:
lymph nodes.
trachea.
thyroid gland.
**ORTHOPAEDIC EXAMINATION:**

Tenderness
Active MP Trigger Points:
- SCM.
- Trapezius.
- Scaleni.
- Levator Scapulae.
- Posterior Cervical musculature.

- Doorbell Sign
- Kemp's Test
- Cervical Distraction
- Halstead's Test
- Hyperabduction Test (Wright's)
- Shoulder abduction Test
- Pizziness rotation Test
- Cervical Plexus Tension

**Remarks:**

**THROTOLOGICAL EXAMINATION:**

**RHTOMES:** Left | Right. **MYOTOMES:** Left | Right. **REFLEXES:** Left | Right.

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<th>C2</th>
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VASCULAR:

BLOOD PRESSURE
CAROTIDS
SUBCLAVIAN ARTERIES
WALLENBERG'S TEST

COMMENTS:

MOTION PALPATION:

<table>
<thead>
<tr>
<th>play</th>
<th>Left</th>
<th>Right</th>
<th>Jt.play</th>
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<tbody>
<tr>
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</table>
PATIENT CONSENT FORM

TENT: ........................................

te: ...........

the undersigned, ........................................, give my
formed consent to be examined, treated and/or x-rayed at the
Chnikon Natal Chiropractic Day Clinic, and will comply with the
structions as stipulated by the intern with regards to his/her
search project.

nature: ..................
# HEADACHE DIARY

**Patient name:**

<table>
<thead>
<tr>
<th>DAY:</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>6</th>
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</tbody>
</table>

1) Did you have a headache today?

2) If yes, how long did it last? (hours)

3) How severe was it? (0-10)
   - None ........... 0
   - Mild ........... 2
   - Moderate ....... 4
   - Heavy ........... 6
   - Severe .......... 8
   - Intolerable ... 10

4) Medication taken:
   - a) How much?
   - b) How often?
   - c) What type?