

THE ANALGESIC EFFECTIVENESS OF HYPNOSIS IN THE  
TREATMENT OF MIGRAINE AND AURA



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Dedicated to the memory of my mother

Maria von Breitkopf

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## STATEMENT OF THE PROBLEM

Migraine has a strong and costly impact on the personal well being and productivity of migraine sufferers. A full understanding of the nature of migraine, its aetiology, and its treatment, remains elusive despite its prevalence and long history. It is estimated that in excess of 20% of migraine patients also suffer the often debilitating, neurological symptoms of aura, which are part of the 'classic migraine' syndrome as opposed to 'common migraine.'

Aura manifests as a separate set of symptoms which are neurologically contrastive to, and usually precede the onset of migraine proper. Despite its sequesterian symptomatology however, the responsiveness of aura itself to forms of psychological treatment such as hypnosis, appears not to have been investigated prior to this study.

The greater volume of research into the treatment of migraine as a whole, has been focused on, and retained within the domains of physiology and pharmacotherapy. However, migraine is a psychophysiological disorder, as categorised by DSM-IV (1994) criteria. It follows, that any attempt to gain a more comprehensive understanding of migraine and aura, also needs to take relevant psychological antecedents into account.

The psychological aspects of the migraine syndrome suggest that this disorder is likely to respond positively to psychological treatment. To date, treatment most commonly available to migraine patients, is pharmacotherapy. This however, has proved to be only partially effective, and at a cost of \$100.- per injection, or up to \$42.- per tablet, it is beyond the financial means of most migraine patients.

Certain psychological therapies, including hypnotherapy, have been shown to be effective techniques in the treatment of headache pain. A number of clinical case studies have also demonstrated that hypnosis can be an effective treatment for migraine (Harding, 1967,78; Milne, 1983). While lacking the rigour of controlled studies, these clinical cases nevertheless suggest, that hypnosis may be a potentially effective alternative in the treatment of migraine and aura. This potential efficacy can be further enhanced by incorporating self-help treatment techniques such as self-hypnosis, which are of additional psychological value in terms of increasing self-reliability and decreasing anxiety (Mountier, 1994; Prendergast, 1992).

Most research which has tested the efficacy of certain techniques in the treatment of chronic pain, such as migraine, has measured effectiveness in terms of only the frequency of attacks and their severity. Yet the duration of any attack is a determining factor in the length of time spent in pain, the other factor being that of frequency. Duration is therefore an essential measure in determining the efficacy of any pain treatment.

Motivated by the real need for effective and affordable migraine and aura therapies, the objectives of this study were to test the effectiveness of hypnosis in the treatment of both migraine and aura, in terms of duration, frequency, and severity.

## ANNOTATED TABLE OF CONTENTS

The objective of this research was to test the effectiveness of hypnosis as a natural treatment for migraine and aura. The assumption that hypnosis is primarily psychological in nature whereas migraine and aura are psycho-neurological phenomena, determined the adoption of a psycho-neurological model for this research.

### Chapters 1 and 2

Any proposed treatment modality has to be aimed at the nature and manifestations of the complaint it seeks to treat. Therefore, an adequate understanding of the neurological manifestations of migraine and aura, their aetiology, their clinical manifestations, their psychological underpinnings, and previous treatment responses, is essential. These aspects are reviewed and examined in Chapters 1 and 2.

Links between the neurological aspects of migraine and aura, and relevant psychological factors are, within the context of these two chapters, considered in section 2.15. Conclusions arising from the literature reviewed so far, and which warrant particular consideration in the later formulation of the hypnotic treatment to be tested, are discussed and summarised in section 2.16.

### Chapter 3

Literature regarding the nature of hypnosis, its psychological aspects and therapeutic applications, with emphasis on its analgesic capacities in relation to headaches, is reviewed in chapter 3. The considerations and conclusions drawn from chapters 1 to 3, are summarised in section 3.61, and lead to the formulation of scientific methodology for testing the effectiveness of hypnosis in the treatment of migraine and aura, in section 3.62.

## Chapters 4 and 5

Chapters 4 and 5 set out the clinical experiment, the methodology adopted, and the results achieved, concluding with a general discussion of the research, its implications and recommendations. While an attempt has been made to avoid unnecessary medical jargon, the use of some medical and physiological terms for the purpose of correct descriptions, was obviously unavoidable. The Glossary of Selected Medical and Chemical Terms will provide assistance in this regard.

## ABSTRACT

This study tested the efficacy of hypnosis in the treatment of both migraine and aura. The hypnotic treatment, administered to 25 migraine sufferers, comprised an integrated four-modality treatment. The modalities were: (a) group hypnosis, (b) hypnotic relaxation, (c) vascular manipulation type A for migraine, and (d) vascular manipulation type B for aura. Thirteen of the participants (52%) also suffered aura. During the pre-treatment period of 12 weeks, participants recorded details of their migraines, aura, and quality of life. This was followed by 12 weeks of treatment, at the beginning of which participants received group hypnosis. This was administered during two training sessions for self-hypnosis. During the treatment period participants practiced relaxation on a daily basis, and used the vascular manipulation procedures A and B, in response to the manifestation of migraine or aura symptoms.

The dependent measures were, duration, frequency, and severity for both migraine and aura. Depression, quality of life, and medication were non-hypothesised measures. Significant pre and post-treatment improvement differences were found for each of these nine measures. Migraine duration was reduced from a group mean of 54 migraine hours per fortnight for the pre-treatment phase to 26 hours at post-treatment. The duration of aura was reduced from a group mean of 9.8 hours per fortnight for the pre-treatment phase to a mean of 4 hours at post-treatment. Pre-treatment migraine frequency was reduced from a group mean of 3.8 attacks per fortnight to 2.8 attacks at post-treatment, and the pre-treatment group mean of aura frequency was reduced from 1.68 attacks per

fortnight to 0.68 attacks at post-treatment. The severity of migraine and that of aura were both reduced from a group mean of 1.99 to 1.37, and 1.42 to 0.44 respectively.

Despite these statistically significant results overall, the consistently less significant treatment responses from some participants raised the possibility of some form of treatment resistance, and further, that such resistance may be occasioned by comorbidity. Investigations led to the finding that the participants concerned (n=13) (52%), suffered from comorbid psychological disorders, classified as somatoform disorders (DSM-IV). Such comorbidity is likely to have a depleting effect on self-perceived coping ability, thereby increasing some patients' reliance on pain as a coping mechanism. It is suggested, in view of these and related factors, that the consistently lower treatment responsiveness of the comorbid somatoform group is ascribable to comorbidity related treatment resistance.

## INTRODUCTORY CHAPTERS

### CHAPTER 1

#### The Disorder of Migraine

##### 1.1 Definition and Overview

###### 1.11 Social and Personal Impact of Migraine

Migraine imposes major personal and social costs upon our community, both in personal suffering and in lost contributions. Parry (1993), in an Australian study on the cost of migraine, based on the National Health Survey of Australia (NHSA) Report 1989/1990, revealed that 68,000 work days and 33,200 school/university days were lost due to migraine and other headaches, and 167,300 days were of reduced activity. He also revealed that while migraine sufferers do not always seek medical help, there were nevertheless some 566,000 reported visits to doctors, specialists, and hospital clinics.

Worthington di Marzio (1992) reported that direct medical/hospital costs for treating migraine during the (one year) 1989/1990 period totalled \$38.28 million. He also showed that indirect work costs such as lowered productivity and time loss, totalled \$264.31 million. The combined totalled cost of over \$300 million per annum is a substantial and ongoing cost to the Australian society.

Osterhaus, Townsend, Gandek, and Ware (1994), who measured the functional status and well being of migraine patients, found that headache is one of the ten most common complaints presented to physicians, accounting for more than 18 million outpatient visits per year in the United States. Migraines in particular, account for substantial morbidity and cost, resulting in an estimated 3 million days spent bedridden

each month, and lost labour cost ranging from \$6.5 to \$17 billion per annum. Clouse and Osterhaus (1992) found that migraine sufferers generated 1.7 times more medical claims, 3 times more pharmacy claims, 3.8 times more emergency room visits and 5 times more diagnostic procedures than an age-matched control group.

Osterhaus, Raymond, Townsend, Gandek, and Ware, (1994) point out that while the economic cost of migraine has received attention and is well documented, its human cost in terms of impact on 'health related quality of life' (HQL) has not been examined extensively. HQL represents the overall effects of illness and therapy on a patient, as reported and evaluated by the patient. Factors which contribute to a health related quality of life include physical and mental functioning and well being, social and role abilities, and general health perception.

Results of their research showed significantly lower scores on the Health related quality of life (HQL) scale for migraine sufferers relative to age and sex adjusted norms and after adjustment for comorbid conditions. Although migraine sufferers scored lower on all eight HQL scales, pain scores were best able to distinguish between patients at varying levels of migraine severity and the pain of the general U.S. population without chronic conditions. For example, the HQL score of patients with very severe migraine was only 41.7, those with mild migraine scored better at 73.0, but their score was still significantly lower than that of the general U.S. population at 84.7. This means that all migraine patients, irrespective of the severity of their attacks, were shown to have significantly lower HQL scores than the general population on measures of pain, social functioning, role disability due to physical health, physical functioning and mental health.

Solomon, Skobieranda, and Gragg (1993) conducted a study into the quality of life and the well being of headache patients. Their findings showed that migraine sufferers had poorer physical and social functioning, and poorer mental health than patients with other chronic conditions. Migraine sufferers are four times more likely to suffer depression and are twice as likely to have anxiety disorders such as agoraphobia and panic attacks (Australian Brain Foundation, 1996). These findings clearly show that migraine has a high personal impact as well as a costly social impact.

### 1.12 Definition of Migraine

The term migraine is derived from the Greek word 'hermikrania.'. Hermikrania means literally 'half the head.' Terms such as heterokrania and holokrania were also used to describe what we know today as migraine. Later derivatives such as myegrym, megrome, and midgramme, were used in the English language in the fourteenth century, the word migraine however was used at least as early as the thirteenth century in France (Oxford English Dictionary, 1969).

Migraine is a symptom complex comprising, among other symptomatology, periodic headaches usually temporal and unilateral, often with irritability, nausea, vomiting, diarrhoea or constipation, and photophobia (Dorland's Medical Dictionary, 1982). The Classification Committee of the International Headache Society (1988), defined migraine attacks as having at least two of the four symptoms set out below, as well as nausea with or without vomiting, and sensitivity to light or sound. The four symptoms require that the pain is unilateral, pulsating, of moderate or severe intensity, and that it becomes aggravated through exertion. This definition has since been broadened to admit bilateral headaches for classification as migraine, with the proviso

that these bilateral headaches share all other requisite symptoms of unilateral migraine (Mountier, 1994).

### 1.13 Historical Overview

The earliest records of unilateral headache date back nearly 3700 years to 1700 BC. The headache was described on a papyrus, (an ancient Egyptian writing material), as 'a sickness of half the head' (Evans, 1988). However, the earliest detailed descriptions of *hemikrania* are those by the Greek physician and philosopher Claudius Galen 131-201 AD, and those by Aretaeus who also lived in the second century and who referred to unilateral headache as *heterocrania* (Low, 1987). Aretaeus was subsequently to practice at the Court

The medical writings of Aretaeus which included descriptions of heart disease, epilepsy, and tetanus, were rediscovered in 1552 by Junius Paulus Crassus who undertook the translation into Latin (Low, 1987). The later English translation of 1856 by Francis Adams gives Aretaeus's descriptions of *heterokrania* under the heading of *Cephalaca* as follows,

... and in certain cases the whole head is pained; and the pain is sometimes on the right and sometimes on the left side... This is called *heterocrania*, an illness by no means mild...it occasions unseemly and dreadful symptoms: spasms and distortions of the countenance take place; the eyes either fixed intently like horns, or they are rolled inwardly to this side or that.. vomiting of bilious matters; collapse of the patient...the patients, moreover, are weary of life, and wish to die (p. 7-8).

Two classical theories on the nature of migraine have dominated medical thinking since the time of Hippocrates (Sachs, 1985). Known as the 'humoral' and the

'sympathetic' theories, they have survived through millenia and were still seriously debated at the end of the eighteenth century; even today, their more evolved variations still demand credibility. The humoral theory, arguably the least credible of the two, claims that an excess of yellow or black bile can bring about 'black humor' or a 'jaundiced view of life,' bilious vomiting and gastric upset. Accordingly, the prescribed treatment consisted of a 'drawing away' of the bilious humor by means of blood letting and heavy purging.

The sympathetic theory holds that migraine has a peripheral origin in the viscera, such as the stomach, bowel, or uterus, and that it is transmitted through the body by way of subconscious communication. This perceived sympathetic connection between the subconscious and physiological symptomatology expressed in Freud's 1916 to 1932 theories on hysteria, anxiety, instinctual life, and neuroses (Strachey, 1975, 1977), clearly shares elements expressed in current theories of psychosomatic medicine (Burrows & Dennerstein, 1993; Graham, 1990; Leshner, 1978; Meares, 1977; Petty, 1987), and in some psychological theories on pain management (Frankel, 1976; Hilgard & Hilgard, 1975; Milne, 1983, 1995; Rose, 1989, 1990; Shone, 1982).

While the basics of migraine symptomatology have not changed since ancient times, scientific investigations have provided some more revealing, if not conclusive, insights into the biological nature of migraine symptoms, specifically in the areas of vascular and biochemical changes (see sections 1.41 and 1.43). A more rigorous diagnostic grouping of symptoms has also led to a distinction between 'common' and 'classic' migraine, and some rarer types of migraine which are variously known under the description of migraine equivalents (Sachs, 1985).

The cardinal symptoms of common migraine are unilateral or bilateral headache and nausea, with or without vomiting. The pain is often of a violent, throbbing nature earlier in its course and then gives way to a steady ache. It is usually accompanied by an array of other symptoms, detailed in section 1.22. Great variability of symptoms is the rule, not only between patients but between attacks, and that some symptoms are conjoined to form characteristic constellations (Sachs, 1985).

The differential feature of classic migraine is that it incorporates in addition to the usual symptoms of common migraine, certain neurological features called 'aura' (see section 1.31). Although the term aura has been in use for nearly two thousand years to denote sensory hallucinations preceding epileptic seizures, it has for the last one hundred years, been applied to denote variously debilitating visual, and sometimes auditory, sensory hallucinations preceding and accompanying migraine attacks.

#### 1.14 Age of Onset and Epidemiology

Relatively early onset of migraine is reported by Lance (1993), who found that 20% of the 500 migraine patients interviewed in his 1960 study, suffered their first migraine by the age of 10 years. He also found that onset at 50 years of age or older is very rare, but that migraine equivalents may appear in the later years of life. Low (1987) reported that 33% of migraine patients experienced their first migraine by the age of 10 years, 56% by the age of 16, and 90% by the age of 40 years. Onset beyond 40 years is rare.

The estimated prevalence of migraine varies according to the diagnostic criteria applied. According to Lance (1993), a survey of nearly 15,000 people was conducted by the British Migraine Trust in 1975 which showed that 10% of males and 16% of females

suffered from unilateral migraines. A 1970 survey in Denmark, found that migraine became progressively more common in early adulthood, increasing from 9% at 15 years of age to 11% for men, and 19% for women during the reproductive years (Waters, 1975).

Only 10% of migraine sufferers are aged between 15-24 years, with the majority of migraine sufferers being aged between 25 and 54 years, and that approximately 70% of all migraine sufferers are women (Australian Brain Foundation, 1993). It is generally accepted that women's greater susceptibility to migraine is influenced by hormonal changes associated with menstruation.

The National Health Survey of Australia (NHSA) 1989/1990 conducted by the Australian Bureau of Statistics revealed that some 540,000 people aged 15 years and older reported migraine as a chronic condition, this figure represents 4% of the 1989 Australian population in that age group. Based on the results of a study undertaken by the International Headache Society (IHS), the Society recommended that a further 4.8% of a country's population be added to the figure of chronic sufferers to derive an estimate inclusive of non-chronic migraine sufferers. Applying that formula to Australia for 1989/1990, it is estimated that 12% of the Australian population aged 15 or older, suffered from migraine during that period. Dr. John King of the Australian Brain Foundation indicated in the Foundation's National Newsletter (Winter, 1996) that the number of migraine sufferers in Australia had reached 1.5 million.

A 1992 National Migraine survey conducted in the United States of over 20,000 people reported that 17.6% of women and 5.7% men suffered from severe migraines. Further, a telephone survey conducted in the U.S. in 1989, in which 10,000 people

between the ages of 12 and 29 years were interviewed, revealed that 7.4% of women had suffered a migraine in the month preceding the interview (Lance, 1993)

## 1.2 Clinical Symptoms

### 1.21 Aura Symptoms

Aura is associated with classic, as opposed to common, migraine. The manifestations of aura are various and intense. They include not only simple and complex sensory hallucinations, predominantly in the visual field, but deficits and disturbances in other areas such as, intense affective states, disturbances of speech and ideation, dislocations of space and time perception, and a variety of dreamy, delirious and trancelike states involving olfactory, auditory and visual sensations (Lewis, 1988; Milne, 1995; Sachs, 1985).

Visual hallucinations are described as scintillating scotoma of flickering luminous spectra usually with a perfect geometrical pattern such as spots of different shapes, sizes and colours, and zigzag lines. These hallucinations are known as ‘specification spectra.’ The scotoma’s brightness can be felt as almost blinding, and objects seen through the spectral colours may be edged with multi-coloured iridescence (Lance, 1993).

A questionnaire study of 100 migraine aura patients, conducted by Queiroz et al. (1997), found that the most common visual scotoma (42%) were small bright dots. Next in this hierarchy were: flashes of light (39%), blind spots (32%), and foggy vision (27%). The authors also found, that for the majority participants, aura duration did not exceed 30 minutes, and that in 57% of cases, aura occurred exclusively prior to the onset of full migraine.

Another characteristic of scotoma is that they are frequently accompanied by analeptic excitement and arousal very similar to the hyperactive prodromal phase which can precede migraine attacks. Conversely, scotoma may also be present with drowsiness and inhibition (Sachs, 1985).

A distinction is made between positive and negative migraine aura. Negative aura is distinguished by its deficit, usually manifested cortically by partial blindness, deafness or speech impairment. Portions of sound may disappear or portions of the visual object may be blocked out, such as, part of a face or a section of print on a page may be absent. This phenomenon is known as hemianopsia (Herzberg, 1994; Low, 1987). Intense negative aura can be particularly frightening to a patient.

Lance (1993) cites the distinguished Harvard Psychologist Dr. Lashley who, being a migraine sufferer and subject to negative visual hallucinations, pursued his scientific enquiry into his own symptoms. He meticulously plotted his reducing field of vision by sticking pins into the screen in front of him. As the perimeters of his vision reduced further, he outlined the contours on the screen to enable himself subsequently, to calculate the rate at which his vision decreased.

His calculations showed that his cortex was temporarily incapacitated by some process which moved across its surface at the rate of three millimeters per minute. Lance continues, that there is a process well known to physiologists, which slowly spreads across the cortex at precisely that rate; it is called 'spreading depression' and is caused by a change in the membrane of nerve cells. It seems highly probable that this process underlies the gradual onset of cerebral symptoms many migraine sufferers experience in connection with aura.

Tactile hallucinations may co-exist with scotoma and may be positive (paresthetic) or negative (anaesthetic). The investigation of these hallucinations may shed light on the nature or origin of aura. The paresthetic hallucinations are said to have a thrilling vibrato of the same frequency as the scotoma and manifest at the most excitable sites of the tactile field such as the tongue, mouth, hands, and less commonly the feet (Sachs, 1985).

Auditory hallucinations often take the form of hissing, growling, and rumbling noises, but at times there can be more organized versions involving, for example, pleasant music; however all can be experienced as excessively loud. Hallucinations of smell and taste can also be either pleasant or unpleasant, as well as very pronounced (Milne, 1995). Hallucinations of motion may be frightening through their sudden onset, especially when accompanied by vertigo, staggering, severe nausea and vomiting. The most intense hallucinatory manifestations may cause patients to confuse their aura experience with reality or dreaming (Low, 1987).

It is estimated that aura is experienced by about 20% of the Australian migraine population (Australian Brain Foundation, 1993). However, the Foundation's most recent Report (1997) states that 64% of migraine patients suffer visual disturbances in association with their migraine. Although the extent to which this percentage is representative of aura, is not stated, it does suggest, that the prevalence of aura may now be in excess of 20% of the migraine population.

### 1.22 Migraine Symptoms

Unlike classic migraine, common migraine is not associated with aura and is usually less severe. In all other respects, the symptom range is identical. Both classic

and common migraine may be preceded by certain emotional symptoms indicating that a migraine is likely to follow. These symptoms are known as prodromes (Karle, 1992).

Lance (1993) points out, that the prodromal symptoms experienced by patients, might include changes in mood and appetite, and drowsiness. Some patients feel euphoric, extraordinarily confident and may become hyperactive during this phase. During other prodromal phases however, the mood may be one of irritability, hypoactivity and depression. Changes in appetite can range from intense hunger or mere craving for sweets which often leads to a consumption of excessively large amounts of cakes, biscuits and chocolates, the latter of which may then be blamed for actually causing the migraine (Low, 1987).

Prodromes arise in the hypothalamus, a deepseated structure in the brain which serves to activate, control and integrate the peripheral autonomic mechanisms, endocrine activities and many somatic functions (Gray, 1974). Prodromal symptoms may persist for up to 24 hours before the actual migraine pain begins. Migraine pain tends to involve the sides of the head more frequently than other parts.

When the actual pain begins, it is usually localized in the temple or in the eyeball on one side of the head. Occasionally it spreads downward to the neck and, in rare instances, to the arm. The sensation is one of intense, throbbing pain that is aggravated by exposure to light, noise, or movement (Low, 1987, p. 9).

The pain sites are notably temporal, parietal, and occipital, and the pain may also occur in the upper and lower teeth, at the base of the nose, in the neck, in the region of the common carotid arteries, and down as far as the tip of the shoulder (Wolff, 1963). While pain locations tend to be unilateral at onset, the pain frequently becomes diffuse

and bilateral as the attack takes its course. About one third of patients experience bilateral attacks at onset (Sachs, 1985).

Selby and Lance (1960) observed 500 cases of migraine and allied vascular headaches. They found that migraine commonly starts as a dull headache which rapidly becomes more severe with a throbbing, pulsating quality. The pain may be felt deeply behind the eye, but more commonly involves the frontal and temporal regions. It may extend over the entire head and radiate down to the face, and even the neck and shoulders.

The headache persists usually for less than a full day, although a feeling of exhaustion and lethargy may persist for several days afterwards. They also found that 72% of subjects experienced dizziness, lightheadedness and unsteadiness, and that 60 out of 396 subjects had lost consciousness in the course of a migraine attack.

Patients may experience numbness in the lips, tongue, and at times in the face and fingers. There may also be mental confusion and difficulty in speaking (Evans, 1988). In the latter instance it seems that the patient wants to say one thing and finds himself saying something quite different. Bickerstaff (1961b) points out that in addition to temporary paralysis on one side of the face or body (hemiplegia), impaired muscular coordination (ataxia) may also be present, indicating brainstem effect.

The onset of migraine pain is frequently sudden and severe, and is usually accompanied by nausea and sensitivity to light (photophobia). Sensitivity to sound (phonophobia) and smell (osmophobia) may also be present. Although migraine may start at any time of the day, it is more common for it to be present on waking in the morning (Rose & Gawel, 1981).

### 1.23 Duration, Frequency and Severity

Although the duration of an aura may outlast that of a migraine attack, auras more commonly begin one to two hours before the onset of full migraine, and subside within two hours after it. This brings the average duration of aura within a range of two to four hours. Patients who suffer aura, tend to experience them with most of their migraines, that is, with similar frequency. On rarer occasions however, auras have been known to occur without subsequent migraines; in this case, they are categorised as 'migraine equivalents' (Lance, 1993). For some patients, aura can be accompanied by such intense emotional disturbance and severe body distortions that they are too frightened to talk about them (Milne, 1995).

The duration of migraine pain may vary from a matter of minutes to several days. The average duration for common migraine is between 8 and 24 hours. The International Headache Society's migraine criteria acknowledges a migraine duration range from 4 to 74 hours (Prendergast, 1988). Duration of migraines attacks has been known on occasions to be prolonged in response to medication taken to reduce severity of migraine pain. Sachs (1985) cites a patient who, suffering from 'week-end' classic migraine, would inevitably face the choice of either enduring some two to three hours of excruciating pain and vomiting, after which he would feel extremely well for the remainder of the week-end, or dull the pain with medication and endure the dull ache until Sunday night.

Frequency of migraines might be more appropriately considered in relation to the duration of attacks. For example, 1 migraine which lasts some 12 hours or even several days cannot, be meaningfully equated with one or even two migraines of only two hours

duration each. For this reason some researchers prefer to speak of frequency in terms of number of migraine days per month (Emmerson & Farmer, 1996).

Selby and Lance (1960) found that more than 50% of subjects experienced between 1 and 4 attacks each month. They considered it highly probable that emotional factors become of great aetiological significance as the frequency of attacks increases. Furthermore, in 15% of subjects who reported more than ten attacks per month, tension headaches were often present as well, and the subjects sometimes found it difficult to distinguish between the two types of headaches.

On rarer occasions patients may progress to what is known as 'status migrainous,' when they awake each morning with recrudescence (recurrence of migraine symptoms after temporary overnight abatement or lack of awareness) (Selby & Lance, 1960). Status migrainous clearly illustrates the ambiguous connection between frequency and duration, for it raises the question of whether recrudescence constitutes continuity of one migraine or the start of a new one.

Severity of migraine pain is proportionally related to, and synchronized with, arterial pulsation. It is easily aggravated by head movements of any kind. In severe migraines, the pulsation of extracranial arteries may be visible, and it can be frequently so severe that the patient becomes greatly incapacitated and even immobilized (Petty, 1987; Sachs, 1985). The severity may also be aggravated by other, coexisting secondary symptoms such as, tension headache, fluid retention, profuse sweating, blurred vision, abdominal pain, diarrhoea, cartharr, dizziness, vertigo, lethargy, drowsiness and other accompanying symptoms already mentioned (Lance, 1978, 1993; Lewis, 1988; Milne, 1995; Petty, 1985). These coexisting secondary symptoms are normally associated with

anxiety disorders (DSM-IV, 1994), and substantiate the understanding that migraine is a psychosomatic disorder.

### 1.3 Clinical Features

#### 1.31 Vascular Changes

The blood supply to the brain flows from branches of the left and right internal carotid arteries which, together with the vertebral arteries, form a communication network around the base of the brain, known as 'the circle of Willis.' From this circle, branches spread to the front, the centre, and to the back of the brain, with finer vessels extending into the deep areas of the brain (Rose & Gawel, 1981).

States of anxiety, which cause an increase in the release of adrenalin and noradrenalin, are known to causally contribute to a constriction of these and other blood vessels. Electroencephalograms (EEGs) show that during the aura or the prodromal phase of migraine, the brain's blood vessels constrict substantially, thereby reducing blood flow by up to 50%. During the migraine attack itself, cerebral blood vessels become swollen and dilated (Lance, 1960, 1978, 1993; Lewis, 1988; Sachs, 1985). The connecting links in the temporal chain of anxiety states, prodromes, aura and migraine, reinforce again, the psycho-neurological nature of aura and migraine, and the appropriateness of a psychological therapy in the use of their treatment.

Bickerstaff (1961a) found that the diminished cerebral blood flow during prodromes may be global or may simply affect the symptom appropriate areas, and that ischaemia (inadequate blood supply) of the cortex or brain stem, may manifest itself in specific neurological deficits, such as negative scotoma. These findings were confirmed by Wolff (1972), who found that negative scotoma can be induced by intravenous

infusion of the vasoconstrictor agent noradrenaline, and can be relieved by the use of vasodilators such as amyl nitrite or the inhalation of 10% carbon dioxide in air oxygen, which is known to be effective in dilating intracranial blood vessels.

Lance (1993) described a case study of a patient who was observed during his prodromal phase. The patient had a slight skull defect leaving a small area of the scalp visibly depressed. When the attack began, the scalp depression started to fill up and continued to do so as the pain spread over the head. At the height of the attack, the swelling was visible as a bulging, palpable mass. This phenomenon further supports the findings that vasoconstriction accompanies the prodromal phase of migraine and is followed by vasodilatation during the attack itself.

Blood vessels, especially arteries, have a muscular coat and elastic fibres which enable them to constrict and dilate, thereby changing their diameter and affecting changes in blood flow. Vasoconstriction and vasodilatation are not uniform throughout the brain. Further, constriction involves branches of the left and right internal carotid arteries located inside the skull, whereas dilatation occurs in the branches of the external carotid artery including the temporal and occipital artery branches which extend outside the skull into the scalp (Grey, 1974; Prendergast, 1995).

Although vasoconstriction is associated with aura and prodromal symptoms, there is as yet no direct evidence that vascular changes such as vasodilatation, actually produce migraine pain, for although blood vessels are supplied with nerves, their function is not that of relaying pain sensations, but that of providing stimuli for the contraction of muscles and constriction of the blood vessel itself. These nerves are controlled by the

autonomic nervous system which comprises the sympathetic and the parasympathetic system (Drummond & Lance, 1983).

The sympathetic nervous system increases the tension of the vessel walls and increases heart rate and blood circulation. It also causes release of adrenalin from the adrenal gland to prepare the body for the 'fight or flight' demands (Rose & Gawel, 1981). Stimulation of the sympathetic system thus affects a decrease or increase in the calibre of blood vessels and blood flow. Other factors which control the diameter of blood vessels are local changes in the concentration of carbon dioxide and acidity. Increases of these chemical elements will cause vascular dilatation (Wolff, 1992).

Rose and Gawel (1981) point out the apparent paradox, that pain, like stress, produces increased sympathetic activity, yet contrary to expectations of the resultant vascular constriction, it increases blood flow to the brain instead of decreasing it. Their explanation, presented only as a possibility, is that the sympathetic nervous system controls the larger, so-called resistance vessels, whereas pain in this context activates local capillaries only. In other words, while migraine pain activates the sympathetic nervous system, and thereby indirectly causes the larger blood vessels to constrict, the local capillaries at the site of the pain dilate to facilitate an above normal blood flow.

Although this explanation accommodates the understanding that migraine pain itself is associated with dilatation of the smaller capillaries situated between the skull and the scalp, it provides no answer to the question of why these local capillaries, in their supposedly compensatory function, facilitate an excessive increase in blood flow? Maybe migraine pain itself is a causal factor, but if so, why is it that other, non-migraine pain is not associated with vascular dilatation?

Evans (1988) points out, that the complexity of cerebral blood flow is compounded by the fact that the system which controls it, is affected by a large number of factors. These include nervous activity, circulating biochemicals (amines), and extra vascular factors such as pain and mental processes. For example, radioactive tracers have demonstrated that during speech there is an increase in blood flow to those parts of the temporal lobe now known as one of the speech centres.

Positron emission tomography (PET scanning) studies have shown that the brain uses less oxygen during migraine, and that as a consequence, brain functions slow down at these times. This may lead to a secondary reduction in cerebral blood flow, since blood flow changes automatically to supply the brain with the precise amount of oxygen it needs.

Motivation and concentration have also been shown to have a direct effect on the blood flow to the brain, confirming the psychosomatic connection between emotion and physiological change (Torda, 1995). Lance (1993) comments that, "Emotion can induce a migraine within minutes" (p. 80). These and the previously mentioned psychophysiological aspects pertaining to both aura and migraine (see sections 1.33 and 1.41) combine to provide a theoretical basis for the use of hypnotherapy as a potentially effective and appropriate treatment modality.

### 1.32 The Pathways of Pain

An adequate understanding of the means by which migraine pain is transmitted and perceived, is of primary importance in selecting a specific and potentially effective hypnotic treatment approach. This section, therefore, addresses in requisite detail relevant neuro-anatomical, and physiological aspects of pain transmission. These aspects

however, are rendered with the qualification that pain transmission as well as pain perception, are also open to psychological influences (see sections 3.41, 3.42, and 3.43).

The bony skull itself, is insensitive to pain, but the inner periosteum, the connective tissue covering all bones, has pain receptors and is particularly sensitive over the brow, the temples, and the back of the head. Stretching of the periosteum, which may be caused by growths or inflammation, produces severe pain. The brain and its smaller blood vessels are insensitive to pain, but all the main arteries supplying its outer covering (the dura mater) are pain sensitive, as are some of the smaller branches and the blood vessels of the scalp.

The stretching of an artery on one side will produce pain on that side. The dura mater covering the floor of the skull in the frontal area is very sensitive and the pain produced spreads to behind the eye; a well known site of migraine pain. The pain associated with dilatation of the internal carotid artery is dull and throbbing and sometimes nauseating; it is usually localized behind the eye over the temple. When an artery becomes dilated, the small nerve fibres surrounding it stretch, and produce signals which may increase with each pulse, creating an increasingly severe throbbing sensation (Rose & Gawel, 1981).

The function of the nervous system is to transmit information about conditions inside and outside the body. It has two parts:

1. The central nervous system (CNS) which comprises the brain, the spinal cord, and the central pathways. Its function is to integrate and control many of the body's activities.

2. The peripheral nervous system (PNS) carries information toward and away from the spinal cord and brain.

Both systems play important roles in migraine.

The brain is the most important part of the nervous system, and certain of its components are highly relevant to an understanding of pain perception in relation to migraine (Drummond & Lance, 1983). The brainstem connects with the spinal cord leading to the higher parts of the brain, the cerebral cortex, it has three main parts known as the midbrain, the pons, and the medulla oblongata.

The midbrain acts as a communication centre for both conscious and reflex messages. It contains nerve cells which when stimulated, cause release of the neurotransmitter serotonin (5-HT), which affects the blood supply of the cerebral cortex. The pons contains the sensory nucleus of the trigeminal nerve, which is important for transmitting the characteristic head pain of migraine. It also contains nerves which can cause the release of the neurotransmitter noradrenaline, which also affects the blood supply of the cerebral cortex.

The medulla oblongata contains nerves involved in the control of blood pressure and breathing. Branches of the trigeminal nerve pass down through the medulla into the upper part of the spinal chord, which is a column of nerve tissues also instrumental in the conveyance of pain signals (Gray, 1974; Prendergast, 1992).

The thalamus is also involved in pain perception. It consists of nerve cells which act as relay centres for messages, including those of pain, which seem to be perceived in the thalamus without being relayed to the cerebral cortex. The hypothalamus plays an important role in pain perception because it contains relay centres dealing with messages

from the autonomic nervous system controlling emotion, breathing, blood pressure, heart function, body temperature, and metabolism (Prendergast, 1992; Torda, 1995).

Torda (1995) maintains that the conduction of impulses through the spinal cord and brainstem to the thalamus, is the simple aspect of pain, and that the processing of this physiological signal into the precepts of pain and the individual's pain behaviour are under the influence of psychological factors. Torda maintains that optimal pain management needs to take all these factors into account.

Rose and Gawel (1981) commented that much of what is known about the pathways of pain, and the nerves involved in conveying pain impulses, was gained from experiments on volunteers receiving mild shock treatment or undergoing operations. Today, this very concept creates abhorrence, or at the very least, raises serious questions about procedural ethics. The problem remains however, that human pain sensitivity and responses can only be ascertained from live human beings.

Pain impulses from the face and the front half of the head, as well as from the front half of the scalp, are conveyed by the trigeminal nerve. The trigeminal nerve is the largest of 12 cranial nerves. When stimulated, it conveys pain impulses from those areas, producing pain in the forehead, jaw, and cheek, depending on which of its three divisions is involved. Pressure on the fine nerve branches in the sinuses of the forehead for example, produces pain impulses which travel along its first division. Pain from the cheeks and upper jaw is conveyed along the second, and pain from the lower jaw travels along its third division (Gray, 1974).

The trigeminal nerve is also responsible for sensations arising from the blood vessels in the scalp. All of its divisions convey pain impulses downward to the brain

stem and then further to the upper spinal cord where they synapse on the same cells as the fibres conveying pain from the back of the head. After this convergence, the combined pathway crosses to the opposite side, and travels upward to the cerebral cortex. The pain sensation is mediated by 'fast' and 'slow' nerve fibres. The fast fibre mediates a sharp pain sensation, whereas the slow fibre conveys a dull and diffuse pain (Lance, 1993; Rose & Gawel, 1981)

The sensory nervous system can distinguish between innocuous low intensity stimuli, and potentially noxious high intensity stimuli, and that low and high threshold sensors are activated correspondingly. Signals generated at high threshold nociceptors apparently communicate with the central nervous system via different fibres (Torda, 1995). In describing the chain action in the conveyance of pain impulses, Lance (1993) points out, that to pass on its pain signal to the next cell, the nerve fibre releases a chemical to stimulate it. Although this chemical is thought to be a peptide, its true character is not yet known; therefore, it is tentatively referred to as 'substance p.'

When the next cell has been stimulated, an electrical wave passes through it upward to the cerebral cortex. This electrical process is then followed by a chemical transmission facilitated by neurotransmitters at the site of the nerve cell synapse, that is, at nerve cell contact. Neurotransmitters may be viewed as keys which unlock nerves or receptors thereby triggering biological responses.

The release of 'substance p' (SP) is controlled by interneurons through their release of enkephalins. Interneurons in turn fall under the pain control system involving the midbrain and brain stem. This system releases the monoamine neurotransmitters serotonin and noradrenaline. More specifically, nerve fibres from a fluid located in the

midbrain, known as periaqueductal grey matter, release the monoamine serotonin over the interneurons. At the same time, fibres from another area in the midbrain, known as the locus ceruleus, release noradrenaline to control interneuron activity and alter the transmission of pain impulses (Lance, 1993).

The release of serotonin and noradrenaline may, in response to emotional factors, be released excessively and facilitate a constriction of blood vessels, which in some individuals may lead to auras and other migraine prodromes. In other psychological conditions, the release of serotonin and noradrenaline may be partly inhibited, leading to vasodilatation, and in some predisposed individuals, to a full migraine attack. Low levels of serotonin are also associated with diminished pain control (Carlson, 1986) and, as more recently discovered, with depression, the prevalence of which has been found to be significantly higher among migraine sufferers (Comer, 1995; Graham, 1990; Snyder, 1996). These factors highlight again, the psychosomatic nature of the migraine syndrome.

The human pain control system modulates the perception of pain, by releasing enkephalins, also known as endorphins, and often described as 'the body's natural opioids.' They are morphine-like substances. The so-called 'high' experienced by marathon runners is attributed to released enkephalins in response to the pain associated with extreme exertion. The endorphins were first isolated in the 1970s (Petty, 1987).

In their function to reduce or block the transmission of pain impulses, the enkephalins are assisted by another chemical, gamma aminobutyric acid (GABA) also released by interneurons. GABA reduces the excitability of the second neuron in the pathway chain and thereby reduces its response to pain messages. The two chemicals

enkephalins and GABA thus guard the gateway against entry of excessive pain signals into the nervous system.

This capacity of the brain to alter the transmission, and with that, the perception of pain, has contributed to the distinction between 'sensory discriminative' pain (which is informative about the location and intensity of nociceptive stimuli, that is, the perceptual quality of the pain) and 'motivational-affective' pain (which reflects the aversive impact and negative emotional resonance of pain). This distinction is now generally accepted at both psycho-physiological and neuro-anatomical levels (De Benedittis et al. 1989; Melzack & Casey, 1968).

Certain aspects of these distinguished dimensions of pain, namely the perceptual quality of pain, and its associated negative emotional resonance, lend themselves, in particular, to psychological and hypnotic manipulation as evidenced by studies researching the effectiveness of hypnosis in the treatment of pain (Hilgard & Hilgard, 1975, 1976; Knox et al. 1974).

### 1.33 Biochemical Factors in Migraine

Sachs (1985) points out that blood levels of serotonin and noradrenaline increase before the onset of migraine and decrease during the attack. This leads to the conclusion that vasoconstriction is accompanied by a rise, and vasodilatation, by a fall in serotonin. The fall in serotonin seems specific to migraine attacks, as it is not a general response in other headaches.

Further, vasodilatation and constriction are also uniquely characteristic of migraine pain and not found in other types of headaches (Herzberg, 1994). Changes in serotonin levels appear thus linked with changes in the diameter of specific blood vessels in the

brain, and hence both of these physiological factors are seen as playing key roles in the pathogenesis of migraine (Lance, 1978, 1993; Mountier, 1994; Prendergast, 1992).

Serotonin levels also fall in response to stress and low blood sugar, and rise as a result of vomiting, consumption of amines such as found in cheese, chocolate, oranges, and the consumption of the amino acid tryptophan contained in milk and turkey (Low, 1987). This tryptophan is transported to the brain by insulin and then converted into serotonin, thus contributing to its increase.

Serotonin is also affected by hormones, where an increase in oestrogen triggers a rise in serotonin, which may then trigger vasoconstriction and migraine prodromes. Conversely, the decrease in serotonin produced by a decrease in oestrogen, may trigger vasodilatation and be instrumental in producing, what is generally referred to as menstrual migraine (Herzberg, 1994). Serotonin's implication in the migraine syndrome, is further substantiated in that drug therapy for migraine, relies substantially on the way certain chemicals affect the production of serotonin. Ergotamine and methysergide are only two of such drugs. These drugs are discussed in section 2.11.

A number of other chemicals are also implicated in migraine, two of these, noradrenaline and prostaglandins, are of particular interest. Noradrenaline is a neurotransmitter of the sympathetic nervous system. It is a powerful vasopressor which stimulates contraction of the arteries and capillaries. Prostaglandins are hydroxy fatty acids. They stimulate the contractility of the smooth muscles and regulate platelet aggregation.

Platelets are constituents of blood and have the capacity to form sticky clumps, an essential process in the coagulation of blood. Platelets of migraine sufferers have been

shown to be present in greater numbers, particularly during migraine attacks (Lance, 1993; Rose & Gawel, 1981). Prostaglandins are the most potent of platelet aggregators. They also perform a large range of other functions, with their action mostly occurring at the site of their production. Petty (1987) points out that a particular type of prostaglandin found in the 1970s named E1, was found to cause headaches and nausea.

The headaches were often preceded by some visual symptoms characteristic of migraine. However, another type of prostaglandin called I2, did not have this effect. The responses and effects of the different types of prostaglandins vary considerably. For example, serotonin is known to cause the release of at least one prostaglandin in the brain, whereas the anti-migraine drugs ergotamine and methysergide have been found to inhibit it. Although prostaglandins do not, at this stage, provide clear indications about their exact function in migraine, their role in the aggregation of platelets, and in turn, the role of platelets in the production of serotonin and in migraine, clearly establishes prostaglandins as having some function in migraine (Amano & Meyer, 1982).

Blood histamine levels are known to increase during migraine. An increase in histamine has been shown to provoke pain. Evans (1988) and Lance (1978) cite Sicuteri's 1967 study, which showed that the histamine freeing substance consisting of bradykinin and serotonin, causes intense pain when injected into the external carotid artery.

The application of this same combination causes extreme pain when applied to the base of blisters. It is now thought that histamine may contribute to the local inflammatory response involved in vasodilatation and migraine. Whatever the biochemical interactions which give rise to local inflammatory responses may be, it is

appropriate to inquire about their antecedent psychological conditions which are involved in headaches and other pain (Carlson, 1986; Milne, 1995).

A current research approach focuses on the gas 'nitric oxide,' which has been found to contribute to the dilatation of cranial blood vessels. It is also involved in the transmission of pain impulses. This discovery has directed efforts to the identification of pharmacological means to inhibit the natural production of this gas (Migraine Foundation of Australia, 1996).

## CHAPTER 2

### The Pathogenesis and Treatment of Migraine

#### 2.1 Aetiology and Contributory Factors

##### 2.11 Aetiological Hypotheses

Many theories about causal factors in migraine have been advanced over the years, including vasomotor, nerve cell derangement, and electrical (nerve storms) theories bordering on epilepsy (Evans, 1988; Herzberg, 1994; Sachs, 1985), hypoxic theory (Lance, 1978; Low, 1987), and endothelial cell theory (Appenzeller, 1991).

Prendergast (1992) lists five of the major causal theories put forward over the past four decades, namely:

1. The vascular theory in which the prime cause is thought to be actual changes in the blood vessels.
2. The neurological theory in which the prime cause is believed to be related to the nervous system.
3. The neurovascular theory which postulates an interaction between blood vessels and nerves as the prime cause.
4. The seroenergic theory in which serotonin (5-HT) is thought to be the prime cause.
5. The so-called unifying theory which, as the title implies, postulates some causal involvement of all the above.

Although the pathogenesis of migraine is still not fully understood, it has long been thought that the pain is of vascular origin. Wolff found in 1938, that the amplitude of pulsation of the scalp arteries increased with the onset of migraine, and that this increase

correlated positively with the severity of the pain at each stage of the attack (Lance, 1978).

### On Vascular and Chemical Involvement

The vascular theory as 'prime cause' for migraine pain is now challenged by a growing number of neurologists for the seemingly obvious reason, that vasodilatation in the brain occurs under other conditions such as exposure to heat or strenuous exercise, without causing migraine pain. Petty (1987) suggests that the vascular theory, although very persuasive, is "...almost certainly wrong" (p. 88). He goes on to say that although vascular dilatation plays an important role, it now appears very likely, that migraine is caused by a chemical disturbance in the brain. This chemical imbalance leads to a change in the way sensations are perceived, and also to a change in the blood flow around the head, and that when both occur simultaneously, a migraine develops.

Notwithstanding the importance of vascular changes in migraine itself, Lance et al. (1967) drew attention to a chemical involvement in the actual production of vascular pain. This pain appeared to be due to an accumulation of various pain sensitizing substances around the dilated arteries; for example, polypeptides containing amino acids which are known to cause pain, are also found in periarterial fluid during migraine attacks.

Sicuteri (1967) found that the vaso-neuroactive substances bradykinin and serotonin free histamines and cause vascular pain (see section 1.43). Wolff (1972) found a related substance which he called 'neurokinin' and which he found to be responsible for triggering an inflammatory response in blood vessel. He thought it possible that this inflammatory response could in turn produce vascular pain.

Sachs (1985) supports this view of chemical involvement and points to the evidence that there are upwards of a dozen neurotransmitters in the central nervous system which can show signs of changes during a migraine attack and often during the prodromal phase as well. Changes can be witnessed, for example, in levels of adrenaline, noradrenaline, acetylcholine, histamine, and predominantly serotonin, all of which are linked to anxiety states.

The more recently advanced endothelial cell theory, based on Ph.D. research into the functions of this cell (Appenzeller, 1991), also draws on chemical involvement. Endothelial cells line the blood vessels of the entire body. They act to protect the circulation and, when disturbed, cause an increase in prostaglandins.

As mentioned in the preceding section, prostaglandins stimulate the contractility of smooth muscles, regulate platelet aggregation and control inflammation and vascular permeability apart from their other functions. Although their involvement in migraine pain has been generally acknowledged, the claim made in this theory, is that the endothelial cell is the most likely site of 'primary abnormality' leading to migraine.

Considering the scientific evidence of neuro-chemical involvement in producing vascular pain, the question of interest now, is no longer whether such involvement exists, but which chemical messengers in the bloodstream, interact with the nerve pathways to produce the sequence of painful vascular constriction and dilatation.

There is no doubt that calibre changes in the skull's blood vessels occur during migraine but, it is argued, perhaps these are produced not by a breakdown in the nervous or neuronal control mechanism but by substances circulating in the blood as it flows through the arteries. Is migraine a disorder of the bloodvessels or of the nervous system? No one is yet quite sure whether it is neural or vascular, so a

compromise description is used that, for the present at least, satisfies all parties:

migraine is 'neuro-vascular' (Evans, 1988, p. 54).

### The Unifying Theory

The hypothesised sequence of causal interaction under a neuro-vascular 'unifying theory' is as follows: In migraine sufferers, either internal or external stimuli cause cortical/hypothalamic activity in the brainstem which may then respond by producing a particular neuro-vascular reaction. This may occur, either by stimulation of trigeminal nerves causing dilatation of the extra-cranial blood vessels and migraine pain, or by stimulation of nerve pathways in the midbrain, causing constriction of the internal carotid artery accompanied by aura or other migraine prodromes. Stimulation of the brainstem also results in the release of noradrenaline which in turn can cause oxygen starvation in the occipital cortex and visual scotoma; it can also contribute to a release of platelet serotonin stores (Prendergast, 1992).

The appeal of this hypothesised causal sequence, may be ascribed to its simplicity, lack of ambitious claims, and that it follows logically from what is scientifically known about migraine to date. It combines vascular, neurological and chemical factors, and although it is too general, and leaves many aspects of migraine unexplained, within the restricted confines of the neuro-vascular field, it does embody the most advanced knowledge of migraine.

Neuro-vascular aspects of migraine however, constitute only secondary links in the causal chain of migraine and aura manifestation. And therefore, a satisfactory causal theory of migraine, needs to give account also, of its primary antecedent conditions, which are psychological in nature.

## Psychological Aspects of Migraine

Research conducted by Levor, Cohen, Naliboff, and McArthur (1986), and Mathew (1990), has shown that chronic headaches such as migraine, are associated with an interaction of psychological conditions such as environmental stress, feelings of inadequacy and helplessness, hostility, compulsiveness, and a passive or depressive personality style. More specifically, psychological conditions have also been shown to interact with such migraine relevant, physiological factors as serotonin imbalance, and vascular and musculo-skeletal deficiencies (Blanchard & Andrasik, 1982; Raskin, Hobobuchi, & Lamb, 1987).

Anxiety and depression, are further examples of the wide range of psychological conditions that trigger these vascular, neurological, and biochemical responses, but they, like other psychological antecedents, have yet to be incorporated into the aetiology of migraine (Comer, 1995; Jackson, 1989; Siegel, 1991).

Schwartz (1982) proposed a 'Disregulation' model to explain how psychological and physiological factors combine to produce illness. He suggests that the body and the brain, work on 'negative feedback loops' which normally guarantee a smoothly regulated function of the body. The model may be explained in simple terms as follows: The brain receives and processes information from the external environment and then stimulates the body organs into action. Mechanisms in the organs then provide critical negative feedback to the brain, which helps to regulate the brain's stimulating activities.

This process can be seen in the blood pressure feedback loop (Egan, 1992; Julius, 1992). In one part of the loop the brain receives information about existing danger, and in the next part of the loop, the nervous system is alerted to elevate the blood pressure in

readiness to deal with the danger (fight or flight response), while the following stage in the loop regulates to maintain the appropriate blood pressure level.

According to this model, if one part of the loop falters, the body enters a state of disregulation, causing further problems throughout the loop, which may ultimately result in a psychophysiological disorder. Schwartz (1982) continues, that three areas of potential difficulties may contribute to disregulation and psychophysiological illness: (a) the person's environment may create extraordinary stress; (b) the person may have idiosyncratic reactions to the environment or to the way the information relating to it is processed, and (c) body organs or feedback mechanisms may be deficient.

It is also possible that certain idiosyncratic needs, personal attitudes, emotions, and coping styles, influence and increase the potential for psychophysiological or psychosomatic dysfunction. Some of the emotional disorders associated with migraine and aura, are classified as Somatoform Disorders (DSM-IV, 1994), and are discussed in this thesis in chapters 4 and 5.

## 2.12 Dietary Influences

It is generally recognized in the medical and biochemical professions that naturally occurring chemicals in food can cause physiological reactions. Selby and Lance (1960) reported that 25% of their 500 participants believed that eating certain foods precipitated their migraines.

Novick (1994) draws a distinction between side effects, intolerance, and allergy. Side effects are predictable reactions to medications or foods, for example, the heaviness felt in the head after taking antihistamines is an expected side effect, not an allergic reaction. An intolerance is an exaggeration of an expected side effect, whereas an allergy can be sufficiently severe to be life threatening. An allergy to penicillin can result in life

threatening respiratory problems. The distinction between these three categories pertains essentially to degrees of severity.

Novick goes on to say that although allergies are true physiological disorders, heightened nervous tension, anxiety, fear and emotional stress may be contributory factors in the development and triggering of allergies. Acceptance of this view leads to the further conclusion that a psychological therapy directed at building skills to improve self-management of anxiety and stress, has the potential for reducing the allergies implicated in triggering aura migraine.

The term 'allergy', when used in the medical sense, means an altered response to a stimulus. It is usual that the presence of an allergy is established on the basis of immunological tests; although in migraine this is not often the case, which is partly due to the fact that allergenic substances in food are difficult to isolate. This difficulty is further compounded by migraine reaction times varying from 2 to 48 hours. Comparative studies are therefore the more frequent means adopted for testing possible links between suspected food chemicals and migraine.

Tea and coffee are just two of the chemicals frequently suspected of precipitating migraine in some patients. Their active ingredients include caffeine and theophylline which prevent the breakdown of the high energy compound cyclic AMP (Rose & Gawel, 1981). The build up of theophylline makes the body highly sensitive to the actions of the sympathetic nervous system, which affects blood vessels and blood flow, and is also involved in the communication of pain impulses.

Because of their capacity to constrict and dilate blood vessels, vasoactive amine chemicals in food are named most frequently in migraine literature as triggers of migraine. Some researchers suggest, that migraine patients suffer an inherited deficiency

of the enzymes which metabolize these amines (Sandler et al. 1974). The focus has largely concentrated on tyramine as the vasoactive amine most likely to trigger migraine attacks. Tyramine is found in cheeses and in a number of alcoholic drinks as well as in a wide range of common foods, including bananas, chicken, pork, tomatoes, yeast, and fermented soy products.

Hannington, Horn, and Wilkinson (1970) undertook a study in which 100 mgs. of tyramine was administered to 50 migraine patients on 100 occasions, resulting in 80 attacks (80% response); whereas the placebo (lactose capsule) administered 66 times, was followed by only 6 attacks (9% response).

Sandler et al. (1974) tested sensitivity to phenylethylamine, the incriminated substance in chocolate. The researchers found that 18 of their 36 'chocolate sensitive' subjects developed migraines some 12 hours after eating chocolate. Wolff (1972) reported on experiments in which incriminated foods such as chocolate, citrus fruit, and fatty foods were ingested by patients without their knowledge and without ensuing headaches. Ingested placebo capsules however, presented as containing the offending substances, were frequently followed by a migraine attack.

Egger, Carter, Wilson, Turner, and Southill (1983) conducted a double blind study of diet treatment and were able to reduce the frequency of migraine attacks. Monro, Brostoff, Carini, and Zilkha (1990) had similar success. However, Medina and Diamond (1978), and McQueen, Loblay, Swain, Anthony, and Lance (1989) found dietary modification had no significant effect on migraine.

These inconsistencies and diverging results demonstrate a regrettable absence of clear indicators which could identify food sensitivities as causal factors in triggering

migraine. They also prompt awareness of the possibility that other, necessary conditions, may have to be present for the food sensitivity to produce its trigger effect.

Rose and Gawel (1981) cite a case study where a patient would only get migraine attacks after eating pork when he was very tired or under emotional stress. This case illustrates that it is not necessarily the allergy or food sensitivity by itself which activates the migraine response, but other stimuli or conditions may also be necessary. This fact may well explain the paucity of compatible results between original and replicated studies and with that, the lack of clear indicators regarding the relationship between the ingestion of certain food chemicals and migraine responses.

Professor Low, a biochemist and former migraine sufferer himself, argues persuasively in his book 'Victory over Migraine' (1987) that a causal connection exists between hypoglycemia and the onset of migraine in predisposed individuals. He argues that in these individuals the consumption of refined sugar stimulates the pancreas to produce excessive amounts of insulin which increases the metabolism of sugar, resulting in hypoglycemia (low blood sugar) and the onset of migraine in migraine sufferers.

The unique system of checks and balances in normal individuals restores blood sugar levels automatically, whereas in hypoglycemic individuals this is not the case. Instead, when the catecholamines cause blood sugar levels to rise by converting glycogen into glucose, the pancreas in hypoglycemic people is overstimulated to produce excess insulin, which once again lowers blood sugar to a dangerous level, and thus the vicious circle repeats itself, producing a prolonged state of hypoglycemia in response to sugar consumption. Low contends that the pancreas of hypoglycemic individuals is extremely sensitive to even small quantities of glucose, no matter what the source.

The culprit in this sequence of events is an overactive pancreas, a condition, without which the ingestion of glucose would not result in prolonged hypoglycemia and would not, by extension of Low's (1987) argument, result in triggering migraine in individuals who are also predisposed to suffer migraine attacks. The hypoglycemia/migraine connection is supported by a large number of writers (Lance, 1993; Lewis, 1988; Mountier, 1994; Prendergast, 1992; Sachs, 1985).

Low's hypothesis is further strengthened in that adrenalin, one of the catecholamines secreted in an attempt to rectify the hypoglycemic situation, is a very potent vasoconstrictor, reducing the calibre of blood vessels symptomatic of the aura and prodromal stage in migraine. Prostaglandins involved in the dilatation of blood vessels symptomatic of the migraine attack proper, are secreted to counteract the adrenalin induced constriction in order to restore the vascular balance. A conclusion could be made, that the excessive dilatation found in migraine is a catecholamine overcompensation in response to the constrictive powers of adrenaline.

What makes Professor Low's theory appealing, apart from its logical connectedness, is that it does not simply rest on a single allergy, such as sensitivity to phenylethylamine (in chocolate), tyramine (in cheese), or refined sugar, per se. Instead, it demonstrates (of importance to future research), that apart from the predisposition to migraine and the suspected food substance, there is an extra, necessary condition operative in this migraine provoking sequence, and this necessary condition is an overactive pancreas. Without this necessary condition, research aimed to establish whether sugar is a migraine trigger in migraine subjects, is destined from the start to

produce less than reliable indicators, because not all migraine sufferers have an overactive pancreas and suffer prolonged hypoglycemia.

This consideration also illustrates the point that whatever food allergies may be responsible for triggering migraine in some people, they cannot have this effect in all migraine patients. Future researchers need to take this into account in the selection of subjects. Further, even hypoglycemia prone migraine patients with an overactive pancreas, may not react with migraines under conditions, where prolonged hypoglycemia is knowingly or unknowingly prevented by frequent ingestion of food. This demonstrates, again, the relative ease with which uncontrolled variables or conditions, can produce counter-effects leading to diverging outcomes in the research of dietary triggers of migraine.

### 2.13 Hormonal Factors

The hormone epinephrine, also known as adrenaline (discussed in section 1.33) merits mention here because of the strongly implicating nature of its various functions and their known effects on neuro-vascular responses. Adrenaline is a highly potent vasoconstrictor and strong stimulator of the sympathetic nervous system, two strongly implicated factors in the pathogenesis of migraine. In addition, adrenaline is also released in response to hypoglycemia increasing such metabolic activities as glycogenolysis and glucose release. This further substantiates Low's (1987) theory of hypoglycemic precipitation of migraine.

Hormonal migraine is a particularly troublesome reoccurrence. Up to 25% of women are said to experience hormonal or menstrual migraine at some time during their reproductive life. The regularity of the menstrual cycle, and with that, the predictability

of menstrual migraine for those prone to it, create a sense of inevitability of migraine pain that may not be avoided. Women in this group are likely to have started their migraines at puberty or after pregnancy, or while on the contraceptive pill (Mountier, 1994).

Lance (1993) suggests that the character of migraine frequently undergoes a transformation at puberty. He writes:

In pubescent girls, the incidence of migraine increases until twice as many girls are affected as boys. In some 60 percent of female patients, headaches occur before or during menstruation. In the last six months of pregnancy, the majority obtain some relief, only to have the headache attacks recur shortly after delivery of the baby (p. 61).

Somerville (1971, 1972) studied the role of progesterone in menstrual migraine, and that of oestradiol withdrawal in the etiology of menstrual migraine. He found that the attacks most frequently started when the levels of these hormones, specifically that of oestrogen, were low before the onset of menstruation. Prevention of a fall in progesterone did not prevent the onset of migraine, but replacement of oestrogen to maintain high blood levels delayed the attack until the oestrogen level was allowed to drop naturally again at mid-cycle, or prior to the next menstruation. This has generally been taken to indicate that a fall in the level of oestrogen, and not progesterone, in some way triggers the onset of migraine in women. It may also account for the mid-cycle migraine experienced in addition to menstrual migraine.

Several studies investigating the hormonal effects of oral contraceptive on migraine (Dalton, 1975; Gardner et al. 1967; Somerville 1972), have shown that oral contraceptives which cause a lower fall in oestrogen than experienced in natural

menstrual cycles, usually exacerbate migraine. For example, the Somerville (1972) study showed that menstrual cycles treated with a plasma oestradiol of only 2ng/100ml, triggered migraines of 12 hours duration, as opposed to those of only eight hours in the higher 5ng/100ml natural cycle. The onset of migraine coincided with falls in oestrogen levels in all these studies.

The emphasis here is on cyclical falls, that is, on relatively sudden decreases in oestrogen levels which may be seen as causal factors in migraine. It is generally accepted that women who suffered migraines during their oestrogen fluctuating years, are often totally relieved of their migraines at menopause, when their oestrogen levels are very low but stable.

This does raise the question however, why do complete hysterectomies, which after all plunge women into immediate menopause, do not provide this relief? Contrarily, they have even been known to increase migraines. The answer, given by Lewis (1988), is that the changes in natural menopause are very slow, spanning several years with gradual atrophy of the uterus and ovaries, thereby allowing for a slow decrease in hormone levels, whereas the opposite is the case in artificial menopause. This explanation also supports the above findings in which the relative suddenness of falls in oestrogen levels, is itself implicated in the triggering of menstrual migraines.

#### 2.14 Genetic Predisposition

Migraine is part of the human condition and of our genetically inherited physiology (Sachs, 1991). This conclusion rests not simply on the familial incidence of migraine, which by itself does not imply genetic inheritance, it rests also on results of comparative studies showing correlations between migraine and genetic sensitivities, genetic chemical

dysfunction, and genetic disorders. For example, Lennox and Lennox (1960), found a constitutional relationship between epilepsy and migraine (Sachs, 1985). Of the 2,000 epileptic patients in that study, 23.9% had a family history of migraine, a figure substantially in excess of that in the control group.

A study conducted by Friedman (1978) which involved 564 patients, revealed that 46% of patients suffering from common migraine and 40% of those with classical migraine had a family history of headache in 1 or more close relatives (Low, 1985). Lance (1978, 1993) speaks of a 'hereditary neurovascular instability' factor, and of a 'family tendency' for migraine, supported by the finding that 60% of migraine patients have at least 1 family member who suffers from migraines, whereas only 16% of the general population have a migrainous relative. And further, it appears that the pain control system and corresponding neurotransmitters in migraine sufferers are more sensitive to changes within and outside the body.

Henryk-Gutt and Rees (1973) compared 100 migraine patients with two other groups, one of which comprised matched subjects who suffered from other types of headaches, and the other group consisted of subjects free of headaches. The findings showed that although migraine sufferers had not been exposed to more stress than the other two groups, they had, nevertheless, a stronger reaction to all forms of stress. The psychological anxiety expressed in the heightened stress response may, like the sensitivity to environmental changes, be a familial or genetic trait.

Theories ascribing a range of specific personality traits such as 'A Type' personality to migraine sufferers have not been substantiated in studies (Kohler, Dulz, Buck-Emden, & Peters, 1991; Lewis, 1988; Prendergast, 1992). The indications are,

according to (Evans, 1988; Low, 1987; Mountier, 1994; Prendergast, 1992; Sachs, 1985), that migraine patients are more sensitive to their internal and external environments. This view is also supported by Milne (1995), who suggests that a physical predisposition to migraine may interact with genetic sensitivity traits and environmental events to produce migraine attacks. Migraine is thus recognised as being a complex disorder, involving not only multiple genes, but also specific environmental and psychological factors.

Research conducted at the Genomics Research Centre, Griffith University, Australia, is focusing, in particular, on chromosome 19 in which the familial hemiplegic migraine gene is thought to be located. The current estimate is, that migraine genes will be identified within the next five years (Migraine Foundation of Australia, 1996). While the identification of migraine genes will be very important for the advance of tailored pharmacological treatment; it needs to be understood, that such identification is not likely to have any effect on the psychological factors which seek expression in the manifestation of migraine pain.

### 2.15 Psychological Factors

The most frequent and important trigger for migraine is psychological stress (Henryk-Gutt & Rees, 1973; Siegel, 1991). Psychological stress in distinction to physical stress is understood to be anxiety based, and brought on by the individual's feeling of uncertainty and perceived lack of requisite means or power, vis a vis needs and responsibilities in a given situation. The ensuing struggle and search for alternative ways of coping (psychosomatic pain may be one such way of coping), are aimed to normalise

this imbalance, to restore control and security, and ultimately, to maintain self-preservation.

Although systematic adaptation can occur to chronic physical stressors, this is not the case in chronic psychological stress. The only defence against emotional stressors is to learn some appropriate coping skills (Graham, 1990; Montgomery & Evans, 1984). Where a normalization of stress levels is denied over extended periods, the stress effect may compound rapidly, with the likelihood of it affecting many other areas of the individual's life, including his mental and physical health (Jackson, 1989; Siegel, 1991).

Health and psychological well-being may be negatively affected in various ways. For example, some migraines occur regularly at the conclusion of prolonged periods of physical or psychological stress. The so-called week-end migraines are typical cases in point. Migraines occurring at the end of stress periods are said to be biologically analogous to recuperative sleep and are interpreted as self-preservative reflexes (Prendergast, 1992; Sachs, 1985; Wolff, 1963). They are thought to be triggered by sudden change brought on initially, by a sharp fall in activity pressure. Adaptation to the external change then leads to biological change involving vasodilating prostaglandins, which will end the vascular constriction, and may, if the compensation is excessive, result in migraine in some migraine predisposed individuals (Stanton, 1988).

The majority of stress related migraines however, appear to occur at the time of stress rather than after it; nevertheless, the common physiological trigger under either condition is thought to be reducible to biological changes of extreme conditions. Lance (1993) has this to say:

The common factor in the wide variety of precipitants of migraine appears to be the rate of change within the body or its environment. This can be as direct as a blow on the head or as indirect as sleeping late on Sunday morning. If the only aim in life were to prevent migraine, either a life of unrelieved monotony or one maintained at a steady pitch of feverish excitement would be the solution (p 89).

The works of other writers (Kohler et al. 1991; Mountier, 1994; Prendergast, 1992; Rapoport & Sheftell, 1991; Sachs, 1985) further illustrate the general acceptance of the idea that stress is a very common precipitator of migraine. A study conducted by Mitchell and Mitchell (1971), which investigated whether migraine frequency can be reduced by more effective stress/anxiety management, can be seen to have been based on the presupposition that stress is involved in migraine.

Stress is the most frequently mentioned emotional trigger of migraines, but just as negative emotions or psychological stress has been shown to trigger attacks, so has a sudden change to positive emotion such as elation, been shown to abort it. Evans (1988), and Lance (1993) cite the example of General Ulysses Grant of the Union Army in the American civil war, who was 'suffering very severely from a sick headache' and spent the night bathing his feet in a hot mustard bath and putting mustard plasters on his wrists and neck. The next day he received a letter from Robert E. Lee, the Confederate General, announcing his willingness to surrender. General Grant wrote in his journal, "I was still suffering from the sick headache: but the instant I saw the contents of the note I was cured" (Lance 1993, p.56). Seen from the perspective of ego-state theory (Emmerson & Farmer, 1996; Watkins & Watkins, 1990), this incident clearly illustrates

the impact a changed ego-state can have on the psychophysiological well being of an individual.

Several case studies (Lance, 1993; Mountier, 1994; Sachs, 1985; Walji, 1994) further demonstrate that sudden emotional change, even of a negative kind, such as may be experienced in sudden fear or shock, can instantly abort a migraine attack or produce a long term remission if not cure. Sachs (1985) relates the case history of a man who had suffered migraines since the age of seven until he was imprisoned in Auschwitz during World War II. During his six years in the camp he suffered no migraines, even though he lost his wife, parents and other close relatives during that time. Since his liberation, however, now many decades ago, he has suffered six to ten severe attacks of classic migraines per month, as well as guilt and chronic depression.

Sachs goes on to say that the so-called habitual or situational migraines arise not as expressions of acute emotional disturbance, but as expressions of chronic, usually repressed emotional needs. Habitual or situational migraines are not simply emotional reactions, they also have the function of restoring emotional equilibrium.

Freud, in his 1916-17 Introductory Lectures on Psychoanalysis (Strachey, 1995) assigned this function of emotional equilibration to what he called 'symptom formation,' a category, for which migraine may well qualify as member. Also in the Freudian manner is the following view expressed by Professor Sachs (1985).

...the symptoms of migraine and many other psychosomatic syndromes, in their symbolic employment, may be seen as a reversion to an ancient and universal mode of expression, a primordial language of the body - implicit in the structure and functioning of the nervous system, and available for use when required (p. 209).

This view about psychosomatic or psychophysiological syndromes, and the implication that migraine symptoms are part of it, have been shared by the psychoanalytic school for many years. Horney (1950) spoke of 'neurotic or functional suffering' as having a wide range of functions. It is a plea for attention, for care and for sympathy, it serves to maintain the individual's solution to demands and needs, and therefore also has an integrating function.

Adler (1927), attached more sinister purposes to psychosomatic suffering. He saw it primarily as means to avoid responsibility, and to attain devious superiority. Reik (1941) maintained that the suffering of psychosomatic pain had dual functions, it could be the means for the attaining of love and for expressing vindictiveness.

Viewed from a cognitive-behaviourist perspective, psychosomatic suffering may be described as an adopted behavioural response to stressors. Montgomery and Evans (1987) contend that behavioural responses remain part of the fight or flight syndrome, part of the self-preservation drive in the face of stress. If, in keeping with the DSM-IV classification of migraine, it is accepted that migraine is a psychosomatic behavioural response, then it follows that migraine patients will be particularly well served by psychologically oriented therapies such as hypnosis.

Unlike the complexities involved in the psychological aspects of stress triggered migraine, the neuro-vascular explanation for the stress trigger itself, can be offered in simple terms: During stress, cranial vessels are maintained in a state of constriction. A sudden decrease in emotional tone and in the circulating agents which regulate vasoconstriction may permit unrestrained or excessive vasodilatation resulting in subsequent migraine.

It is not fully understood whether stress triggers migraine by increasing only the production of adrenaline, or whether, as is more likely the case, other catecholamines are also involved. Based on psychological theory, and by extension of the hypoglycemia argument advanced by Low (1987), the series of likely events leading to migraine may begin and develop like this:

1. Emotional stress or some drugs taken to combat it, can cause the pituitary gland to produce hormones which stimulate the pancreas to generate excessive amounts of insulin.
2. Unless the insulin balance is restored promptly, low blood sugar or hypoglycemia results.
3. The adrenal glands react to hypoglycemia as they do to stress itself, by releasing adrenaline and other catecholamines, resulting in vascular constriction.
4. Prostaglandins, are released as a countermeasure to dilate the constricted blood vessels.
5. The change from vasoconstriction to vasodilatation manifests as migraine in migraine prone individuals.

Stages one to five in this series of events are applicable independently of hypoglycemia, because hypoglycemia is not a necessary condition for the release of the vaso-constrictive agent adrenalin. Its release also occurs in response to emotional stress.

Stages one to five, preceded by stress, can therefore apply to migraine sufferers in general.

Serotonin plays a major role in the body's pain control mechanism and in registering emotion, including stress, anxiety, and depression. Low serotonin levels have

a negative effect on mood and level of pain control (Carlson, 1986; Guidano and Liotti, 1986). This neuro-biological understanding helps to explain why stress, anxiety, migraine and depression are frequently found to be co-existent.

Consistent with the above are the findings of Henryk-Gutt and Rees (1973) (discussed within the context of genetic predisposition in section 2.14) which are of relevance here, as they offer an additional perspective on the stress/migraine connection. Those findings revealed that without being exposed to greater stress, migraine sufferers have a stronger reaction to it than individuals who suffer other types of headaches, or no headaches.

#### 2.16 Considerations in Hypnotic Treatment of Migraine

The major factors for consideration in the formulation of hypnotherapeutic treatment arising out of the discussion on migraine so far, may be summarized in point form as follows:

1. Stress is a frequently cited trigger for migraine. The use of self-hypnosis for relaxation as a stress management technique can be recommended.
2. Stress and anxiety induced by the patients awareness of lack of control, is addressed when they are provided with self-intervention skills such as self-hypnosis.
3. Anxiety and depression are part of the migraine profile. The practice of hypnotic relaxation regulates the production of serotonin involved in these emotional states and is therefore appropriate (Spiegel, 1965).
4. Physiological triggers of migraine are understood to be chemico-vascular in nature. Hypnotic relaxation, especially when combined with imagery focus, lowers and normalises levels of adrenaline, noradrenaline, and serotonin, all of which are increased in

response to stress and known to be involved in the vasoconstriction of aura and early phase of migraine.

5. Vasoconstriction of aura and early migraine, and the vasodilatation of full migraine, can be addressed with specified imagery to normalise these opposite extremes.

6. The brain's capacity to alter the transmission and the perceptual quality of pain, also facilitates its psychological and hypnotic manipulation (Hilgard & Hilgard, 1975; Knox et al. 1974).

7. Migraine has been classed as one of the traditional psychophysiological disorders (Comer, 1995). The practice of hypnotic self-intervention skills such as relaxation, specified imagery, and ego enhancement, has the potential to impact positively on the psyche and physiology of the individual (Jackson, 1989; Rose, 1989; Spiegel, 1989, 1992).

8. The pathogenesis of migraine in so far as it is understood, does not contraindicate hypnosis.

## 2.2 The Treatment of Migraine

Although the scientific purpose of this research is to test the analgesic effectiveness of hypnosis in the treatment of migraine and aura, it is also of clinical relevance to view its effectiveness in the light of other available treatments. The potential clinical role of hypnotherapy in this context, and its selection by doctors, psychologists, and migraine patients, as a treatment of choice, is dependent on a number of factors. From a practical perspective, these factors relate directly to the effectiveness, public awareness, cost, and convenience of hypnotic treatment compared with all other treatments commonly

available to migraine patients. In view of the relevance of these factors, physiological and psychological migraine treatments are reviewed in sections 2.2 and 2.3 respectively.

### 2.21 Drug Treatment

The often extreme nature of migraine has over the centuries generated an equally graphic range of alleged treatments. By the turn of the eighteenth century, patients were treated with a range of concoctions including Peruvian bark, camphor, hemlock, and opium, while treatments such as opening the temporal artery, teeth extractions, skull surgery, and ‘blistering behind the ears,’ were claimed to be particularly effective in resistant cases (Evans, 1988).

Guided by scientific advances, the pharmaceutical and medical professions of more recent times, have been able to adopt a more informed approach. Pharmacotherapy for migraine is today based on the practicality that the disorder to be treated is one of painful dilatation of cranial blood vessels. A distinction is made between treatment for migraine prevention, and treatment for acute attacks. The objective of the former is to prevent vasoconstriction of cranial arteries, that is, the first stage of migraine. This however, necessitates a continuous administration of the preventive medication. The objective in treating acute attacks is to induce vasoconstriction of the already dilated scalp arteries symptomatic of the painful second stage of migraine, and to relieve pain and vomiting.

Generally speaking, a drug may produce pain relief via four possible routes:

1. It may elevate the pain threshold, where physical processes remain the same but the person’s perception of pain is blocked or reduced (analgesics).
2. It may modify muscle tone, causing tight muscles to relax or prevent them from contracting in certain ways (tranquillizers).

3. It may reduce inflammation of the brain, nerves, and blood vessels, encouraging the latter to constrict or dilate (beta-andrenergics).

4. It may also increase the availability of certain neurotransmitters and stimulate certain nerves to counteract the usual biochemical processes that produce headaches (antidepressants).

All of these effects however, may also be achieved to some extent, by drug free methods (Rapoport & Sheftell, 1991).

The wide range of drugs used for migraine prevention includes drugs commonly prescribed for psychological and psychiatric disorders, including anxiolytic benzodiazepines such as Diazepam; antipsychotic beta-andrenergics such as Propranolol; anti-depressant tricyclics like Amitriptyline, or monoamine oxidase inhibitors (MAOIs) such as Phenelzine; as well as anti-convulsants used in the treatment of epilepsy, like Carbamazepine (Lance, 1993). These drugs are often supplemented with anti-inflammatory compounds such as Naproxen to reduce swelling of blood vessels, anti-emetics such as Maloxon to reduce vomiting, and opium derivative compounds like Pentazocine and Codeine to reduce pain.

Benzodiazepines are variously referred to as anti-anxiety agents, anxiolytics, or minor tranquilizers. They sedate, temper excitement, and reduce pathological anxiety. They are frequently prescribed for panic disorder, phobias, and bipolar disorder. Benzodiazepines are anti-convulsants and muscle relaxants; their exact effects in the treatment of migraine are not fully understood. In general, benzodiazepines act as hypnotics in high doses, as anxiolytics in moderate doses, and as sedatives in low doses. They are addictive and some patients develop an increasing tolerance to the drug,

requiring higher doses over time. Depending on the rate of withdrawal, the range of withdrawal symptoms may include anxiety, insomnia, dizziness, muscle twitching, headache, and concentration difficulties.

Beta-andrenergics, also referred to as beta-blockers, act on the circulatory system, originally designed and prescribed for a range of psychiatric disorders and to reduce somatic symptoms of anxiety, they inhibit the 'fight and flight' mechanism triggered by the release of adrenalin, they slow pulse rate and reduce blood pressure. Because of their capacity to stabilize blood vessels in and around the brain, they have been deemed suitable for migraine, although their exact action is not known.

They tend not to lead to addiction, and their side effects include ataxia (lack of muscle co-ordination), weakness, blood disorders, insomnia, contraction of airways around the lungs, nausea, vomiting, and hallucination. Heart failure may also occur. They are contraindicated for asthma sufferers and must not be taken with ergotamine medication (Julien, 1995; Kaplan & Sadock, 1991).

The actions of tricyclic, and monoamine oxidase inhibitors antidepressants are different from that of anxiolytics, they reduce the reuptake of norepinephrine and serotonin thereby raising the pain threshold. They are used for a variety of conditions where chronic pain is a feature. The fact that they are non-addictive, may be considered a big advantage, however, withdrawal from the drug needs to be gradual as sudden withdrawal may cause irregular heartbeat (Julien, 1995).

Special dietary precautions are indicated when monoamine oxidase inhibitors are taken. Foods such as cheese, red wines, meats, chicken livers, and broad beans, for example, need to be eliminated from the diet. It is particularly important, that no other

drugs with a noradrenaline type action be taken in conjunction with MAOI medication. The reason is that this group of drugs prevents the breakdown of noradrenaline in the body, and an excess of noradrenaline in the bloodstream can cause an increase in blood pressure which could have very serious consequences (Lance, 1993).

The shared effect of the diverse pharmacological actions of these drugs is their capacity to control vasoconstriction, directly or indirectly, and produce some measure of pain relief. Their side effects can be worse than the symptoms they are supposed to camouflage. These include: hallucinations, addiction, hypotension, hypertension, blood disorders, and arterial spasms, (Kaplan & Sadock, 1991; Lance, 1993; Walji & Kingston, 1994).

The drug traditionally used in the treatment of acute attacks for the past 50 years has been ergotamine. It is capable of constricting the external carotid arteries but the efficacy of this depends on the pre-existing vascular resistance. Ergotamine is effective as a vasoconstrictor when vascular resistance is low, but is transformed into a vasodilator as vascular resistance is increased (Aellig & Berde, 1969; Prendergast, 1992; Rose & Gawel, 1981; Walji & Kingston, 1994). This indicates that a migraine attack can become worse when ergotamine is administered to patients with average or high vascular resistance. Cases have been reported of increased frequency, severity and duration of migraine in response to prolonged administration of high doses (Walji & Kingston, 1994).

Low (1987) claims that in most cases ergotamine has no effect at all. He also draws attention to the fact that its administration is contra-indicated in cases of hypertension, cardio-vascular disease, thrombophlebitis, and renal disease. Evans (1988)

adds that the minimal effective dosage is often enough to produce the very effects of headache and nausea it is supposed to eliminate.

Ergotamine is a potentially harmful drug and can cause the severe illness known as 'ergotism,' which produces burning pain and even the loss of limbs through gangrene. Edmeads (1973) found that minimal, migraine effective doses of between 0.25 - 1.00mg are capable of causing uterine contractions, therefore ergotamine is contra-indicated during pregnancy.

Another powerful drug is methysergide. It has anti-serotonin effects and also acts as an anti-histamine agent reducing inflammation of the arteries. Its full actions and effects are not yet fully understood. While methysergide does not itself constrict cranial blood vessels, it does make them more sensitive to the constrictor effects of noradrenaline (Fozard, 1975). Methysergide is generally considered to be effective in about 50% of non-acute migraine cases, but is deemed as dangerous as it can be effective (Evans, 1988).

The possible side effects of methysergide include abdominal pain, nausea, vomiting, blood disorders, mood changes, skin rashes, and weight gain. It is contra-indicated for patients with kidney or liver disease, heart disease, circulatory disorders, oedema, and hypertension. Cady and Farmer (1993) point out that methysergide has been associated with a rare idiosyncratic fibrosis, which if left untreated, can result in death.

Along similar lines, Lance (1993) comments that if the drug is administered continuously and in large doses, its serotonin stimulating action has the potential of producing fibrosis in susceptible individuals, including retroperitoneal fibrosis, pleural

fibrosis, and cardiac valvular fibrosis. Current practice prescribes that the patient remain under constant medical supervision and that medication be completely stopped one month out of every four to prevent these fibrotic side effects.

The extraordinary variety of drugs used to treat migraine attest to the multifactorial nature of the migraine syndrome, and lead to the conclusion that there is as yet no single agent sufficiently effective to make all the others with their shortcomings obsolete. A recent vasoconstrictor drug used in migraine is Sumatriptan. This drug is based on the serotonin molecule, it has little or no effect on other than the 5-HT<sub>1D</sub> receptor responsible for vasoconstriction, and it has fewer side effects than serotonin (Goadsby et al., 1991). Its side effects include, sensations of numbness, tingling especially in the head and neck area, flushing and numbness (Cady & Farmer, 1993). There have also been reports of chest pains caused by spasms of the coronary arteries. As a consequence, the drug should not be used by patients with suspected coronary artery disease or uncontrolled hypertension (Lance, 1993).

Although the side effects may be fewer than those of other drugs, they still warrant concern. The drug is, at best, effective in 75% of cases, and despite its so-called rapid onset of action, this figure is only reached after a lapse of some four hours. Finally, the cost of the drug at \$100.00 per injection or up to \$42 per tablet, constitutes a considerable burden on the Australian community, as most migraine sufferers experience more than one attack per week. There is no doubt that pharmacotherapy for migraine is improving, and that it brings relief to many migraine sufferers. However, given the side effects, the inherent dangers, the high cost, and the restricted range of its effectiveness, some alternative treatments are clearly needed.

## 2.22 Surgical Intervention

The use of surgery is controversial: obviously the wholesale extirpation of viscera and other organs, once recommended, is quite monstrous; but the excision of the affected superficial artery in cases of intractable migraine may sometimes be justified (Sachs, 1995, p. 229).

Contrary to such expected justification however, surgical procedures on nerve pathways in the sympathetic and parasympathetic nervous systems, or ligation on branches of the external carotid or middle meningeal arteries, have not produced the hoped for, lasting benefits for migraine sufferers. The short term relief that has been achieved, has come at a price. For example, coagulating or dividing the trigeminal nerve produces some relief, but it leaves the treated side of the face numb, causing the patient to lose the protective blink reflex and leaving the eye prone to infections. For this reason, the operation is considered a last resort measure, and then only for those patients whose migraines reliably affect only the same side of the head (Lance, 1993).

Patients in this category also have the option of local anaesthetic procedures involving the sectioning or cutting of forehead nerves which may provide short or perhaps long term relief. Similarly, patients whose migraine is always restricted to one side of the back of the head, may be given temporary relief by injecting a long acting type of cortisone, or possibly longer relief by sectioning the appropriate nerve. If the migraine originally occurred in response to injury to the scalp, surgical removal of local nerves and blood vessels may provide permanent improvement. All of these options, as drastic as they are, are open only to the small minority of migraine sufferers whose migraines are localised to the same section of the head.

It may be concluded that while this type of surgery may be a worthwhile option for a small number of patients, its potential benefits need to be assessed in the light of its likely side effects. Some medical experts also point out that because migraine is a disorder of function, and not one of structure or organic disease, there is little justification for surgical intervention (Lance, 1993).

Surgical intervention will do nothing for the migraine sufferer except perhaps fulfil some deep masochistic need to be operated on in order to get better. Even more specific surgery, such as that done on the nerves of the temporal artery in the case of severe one-sided migraine, seems to be of questionable value and is best avoided because any relief it brings seems to be very short-lived (Evans, 1988, p. 136-137).

### 2.23 Dietary Control

Dietary influences (see section 2.12) led to the conclusion that despite the diverging results obtained in studies examining food sensitives, there is some general consensus, based largely on anecdotal evidence, that under certain conditions, some food chemicals have the capacity to trigger migraine in predisposed individuals. Expressed in other words, a food sensitivity or allergy may not of its own accord, but in the presence of certain other conditions, such as anxiety, lead to migraine.

Dietary control as a treatment of migraine usually takes the form of one of three traditional approaches: (a) desensitization to suspected food substances such as histamine, (b) selected vitamin supplementation, and (c) exclusion diets.

Antihistamine drugs remain the 'workhorses' of anti-allergy therapy (Novick, 1994). Novick also contends, as does Snyder (1996), that active desensitization to histamine, despite the fact that it is a laborious and drawn out process, becomes the

preferred choice when antihistamines have to be avoided because of their sedative side effects. Antihistamines produce general co-ordination impairment, and are contraindicated in cases with a history of glaucoma or inflammation of the prostate.

Desensitization, also known as immunotherapy, is often adopted when drug therapy is contra-indicated, or when it has not been successful. The desensitization procedure is aimed to make the body more tolerant or less reactive to the offending substances. The procedure usually involves injecting minute, but ever increasing, amounts of the allergen over a period of weeks and months or even years, until a dosage is reached which, when maintained with periodic booster shots will prevent symptomatic reaction to the allergen. Maximum benefits may require several years of desensitization treatment, but the main drawbacks, apart from the possibility that the desensitization injection may itself cause an allergy response, are costs and the inconvenience of regular, frequent visits to medical clinics.

Although a desensitization approach works well in cases of hay fever, and in some cases of asthma where there are definite identifiable allergic responses, the situation in migraine is not as clear. Desensitizing migraine sufferers to the offending food substances, such as tyramine in cheese, or phenylethylamine in chocolate, does not always confer a benefit. This low level response may be due to the fact that histamines are potent dilators of intracranial blood vessels, but only marginally so of the scalp arteries associated with migraine (Julien, 1995). Lance (1993) point out that desensitization procedures are difficult to adapt to controlled trials and that it is questionable whether the results warrant the time and the inconvenience involved.

The advice usually given to migraine sufferers is to eliminate those foods from their diet, which, based on their own experience, may be involved in triggering their migraine. Special procedures are laid down for this task, demanding strict adherence to an elimination diet such as the rotary diversified diet, the four-day rotation diet, the five-day fast on bottled water diet, the two-food lamb and peas diet, or the modified exclusion diet (Mountier, 1994).

Considering these contemporary theories on dietary triggers and treatment of migraine, involving as they do, most known food chemicals and vitamins, one is led to the inevitable conclusion, that whatever the merits of the recommended diets may be, and that of their respectively recommended desensitization, eliminations and supplementations, the unabated continuation of migraine suffering, attests to the general failure of dietary control as a generally effective treatment of migraine.

#### 2.24 Hormone Therapy

Somerville (1971, 1972) (see section 2.13) revealed that progesterone replacement to maintain high levels of the hormone prior to menstruation, does not prevent menstrual migraines. It seems reasonable to conclude that if a fall in progesterone were implicated in causing migraine, then the prevention of such a fall would most likely result in some preventive effect, but this was not the case. It was found, that high level maintenance of oestrogen delayed the onset of migraine until the level of oestrogen was allowed to fall. The conclusion reached was that a fall in oestrogen can trigger migraine attacks.

Mountier (1994) contends that lack of progesterone is not likely to be corrected by the artificial progestogen of hormone replacement therapy (HRT). As with the contraceptive pill, the progestogen will tend to deplete the body's natural progesterone

even further, making HRT as risky and as likely to trigger migraine as the contraceptive pill. Mountier's emphasis on progesterone as a trigger of migraine, could, in the light of Somerville's (1971, 1972) findings, possibly be misplaced. Unlike oestrogen which falls markedly at mid-cycle and again prior to menstruation, progesterone remains at a steady, very low level up to mid-cycle, then rises during the second half and falls only prior to menstruation. As such, there is no marked fall in progesterone at mid-cycle, and therefore its 'fall' cannot be implicated in triggering mid-cycle migraines.

The discovery of the connection between falls in oestrogen levels and migraine has renewed interest in the possibilities of hormone replacement therapy (Lance, 1993). However, treatment with synthetic oestrogen and progesterone has proved disappointing although it is thought that oestrogen implants and the application of oestrogen gels to the skin during menstruation may suppress the migraine in some women.

Lance (1993) pointed out that various forms of contraceptive pills, particularly high dosage contraceptives, increase both frequency and intensity of migraines in most instances. This is explained on the basis that oral contraceptives contain different combinations of synthetic oestrogens and progesterones which do not really simulate pregnancy as they allow hormone blood levels to fall each month and menstruation to take place. They also cause lower falls of oestrogen than occur in natural menstrual cycles.

There is a further concern, this is, that permanent neurological deficits have been reported in patients whose vasoconstrictive prodromal phase was prolonged while taking oral contraceptives. Lance (1978) understandably concluded; "The only hormonal treatment which is at least 60% effective is pregnancy" (p. 187).

Hormone therapy can aggravate migraine, and it may also increase the risk of heart disease, stroke, and cancer. Oestrogen causes spasms in the arteries, and this danger becomes compounded when HRT is used in conjunction with vasoconstrictive drug therapy, such as ergotamine based compounds. Hormonal changes also affect the liver which has the task of breaking down excess oestrogen and progesterone. Further, migraines are capable of producing stroke symptoms, so the combination of HRT and migraine is seen as doubling the risk of stroke (Rapoport & Sheftell, 1991).

Rapoport and Sheftell (1991) suggest that cyclical hormone replacement therapy (21 days on and 7 days off) is more likely to increase migraine and other possible dangers, than is non-cyclical or daily hormone therapy. It is also thought that synthetic oestrogen, although imbued with its own dangers and not very effective in the relief of migraine, is nevertheless safer than that produced from animal hormone. The writers' final recommendation, which is widely shared (Lance, 1993; Lewis, 1988; Julien, 1995; Mountier, 1994; Prendergast, 1992), is that hormone replacement therapy should be avoided if at all possible.

### 2.25 Acupuncture

Acupuncture treatment is aimed to restore lost equilibrium, using specific points of the body's meridian network where, theoretically, the yin and yang forces can be influenced, diverted, and strengthened (Rose & Gawel, 1981; Walji & Kingston, 1994). There are about 500 acupuncture points joining to form 12 major meridians. Particular points are said to relate to specific organs and body functions such as the liver, stomach, lung, heart, and circulation, and so forth.

The acupuncturist is trained to feel the 12 meridian 'pulses' (one for each meridian, and six to each wrist) to detect abnormalities and to arrive at a diagnosis. The diagnostician is said to be assisted by the patient's personal gestures, voice tone, and distinctive body odour. Fine needles may be used in conjunction with electric currents or laser energy to activate specific points, remove blockages, and to rebalance the system into an harmonious whole (Herzberg, 1994; Lewis, 1988).

Acupuncture practitioners believe headaches are the result of an energy blockage of the yang channel in the head (Lewis, 1988; Prendergast, 1992). Herzberg (1994) cites the Nan Jin, one of the classics of Chinese medicine, which states that when anger rises to the head but does not descend, the liver is damaged. 'Liverfire' has hot, angry yang energy, therefore the headaches are triggered by anything hot, a hot temper, bright sunshine, or hot spicy foods. Headaches are hence thought to be more common in people who do not resolve their anger, who allow their 'liverfire' is allowed to linger.

The explanation offered for the pain reduction achieved with acupuncture, is that acupuncture, as opposed to the placebo effect, encourages the release of pain reducing endorphins and enkephalins (Rose & Gawel, 1981). The analgesic capacity of acupuncture is also ascribed to its stimulation of the fast-conducting fibres in the pain gate mechanism. This mechanism, according to the pain gate theory, can block out pain impulses by transmitting other information along the same pain pathways (see section 3.41) (Rose, 1990). However, the pain gate theory itself has not been scientifically substantiated.

Although acupuncture is used in the treatment of migraine and in the treatment of other headaches, very few studies into its effectiveness have been documented.

Information, particularly in regard to its effectiveness in the treatment of migraine, is largely based on anecdotal evidence. However, a study conducted at the National Hospital for Nervous Diseases London, revealed that after 3 months treatment, 24 out of 41 migraine patients derived some benefit from acupuncture, and 17 remained unchanged.

Lance (1993) comments that acupuncture is time consuming for both doctor and patient, and that it can be quite unpleasant for the patient, as well as dangerous, unless procedures for adequate needle sterilization are strictly adhered to. On the question of long term effectiveness of acupuncture, Lance (1978) comments, "...my own experience has been that patients improve while undergoing treatment and relapse shortly thereafter" (p. 186).

Acupressure, which is also used in headache treatment, is distinguished from acupuncture by its use of finger pressure instead of needles, and by the fact that this pressure can be administered by patients themselves. The claim is that, when specific points on the meridian are pressed, a series of relaxation responses are triggered. These relaxation responses release muscular tension, promote circulation, relieve stress, aid mental relaxation and promote a general sense of well-being (Herzberg, 1994).

## 2.26 Summary Overview

While the physiological treatments reviewed under section 2.2, have been used with some, if varying degrees of success, many of these treatments are accompanied by adverse side effects, inconvenience, or expense. Surgical intervention is a radical measure, generally not recommended by the medical profession. Selective drug treatment offers some relief by reducing the severity of migraine pain. Its capacity to

prevent the onset of migraine however, can only be put to the test with continuous daily ingestion.

The role and effectiveness of dietary control in the treatment of migraine remains uncertain. There is the further consideration that, management of migraine by exclusive focus on the avoidance of foods associated with allergies, overlooks the need to deal with the psychological aspects involved in the manifestation of food allergies.

The use of hormone therapy is, by definition, restrictively directed to reduce migraines associated with oestrogen falls at menstrual mid-cycles. Independently of the dangers that accompany Hormone Replacement Therapy (HRT) when used in conjunction with vasoconstrictive migraine drugs, the effectiveness of HRT as a migraine treatment is limited. It is further restricted in that it can only be considered in cases of menstrual migraine.

The availability of documented scientific data on the effectiveness of acupuncture in the treatment of migraine is sparse. It appears that much is still to be learned by Western therapists about the use and the full potential of this treatment modality. A decided advantage, in the view of many therapists, is its drug free nature.

The foregoing review and summary of physiologically based migraine treatments, demonstrates the need for an effective treatment modality which also addresses the psychological aspects associated with migraine. Ideally, such therapy should have no adverse side effects, should build skills for coping with the underlying anxiety associated with aura and migraine, should enhance self-esteem and general well-being, should facilitate self-administration, and be financially affordable.

## 2.3 Alternative Psychological Treatment of Migraine

### 2.3.1 Psychological Therapy

The ways in which human needs and functioning are viewed largely determine theory and practice of psychological therapy. A holistic approach of body, mind, and spirit in social context, linked to the three aspects of our personal world of psyche, relationships, and culture, is these days considered necessary to avoid an overemphasis on physiological mechanisms (Pitty, 1994).

Hilgard (1993) comments that pain is not only a somatic problem, the consequence of nerve injury or the secondary consequence of muscular tensions, but that it has many other roots in the psyche of the individual which warrant the attention of the therapist. As clinicians have become more aware that psychological factors are frequently the basis of physiological disorders, the use of psychological therapy and intervention techniques has substantially and beneficially increased.

The most frequently used intervention strategies, such as relaxation training, biofeedback training, cognitive behaviour therapy, meditation, and hypnotherapy have involved components of operant conditioning (Hilgard, 1993; Lehrer et al. 1993). Although these therapies were initially used only for traditional psychological or psychosomatic disorders, their use has now increased to cover a much broader area of medical complaints, which has given rise to the term 'behavioural medicine' (Blanchard, 1994).

While each of the above therapies is considered an independent psychological therapy in itself, their mutual compatibility has proved to be a further advantage in that they can be effectively used in combination. The practice of hypnosis helps to

demonstrate this point, for although hypnosis can be effectively used by itself, it is more commonly used as an adjunct to other psychological treatments, such as Cognitive Behaviourism, or Ego-state therapy (Emmerson & Farmer, 1996; Milne, 1995; Phares, 1991; Watkins & Watkins, 1990). This finding is supported by the results of a survey initiated by the Migraine Foundation of Australia (1997) which revealed, that hypnosis by itself, was used by only 2% of migraine sufferers.

The most successful psychological therapies, in relation to pain, have involved conditioned responses based on the classical conditioning of Pavlov (1951), the operant conditioning of Skinner (1960), and the social learning theory of Bandura (1969). More specifically, Hilgard (1993) writes that the chronic pain patient is taught that pain responses have been learned and can therefore be unlearned, and that:

The unlearning, based on operant conditioning principles, is very ingeniously arranged, so that non-pain responses are rewarded, and pain responses are countered or extinguished. For example, a person with joint pains can walk only so far before the pain becomes so great as to require rest. Looking forward to that rest is like expecting a reward for experiencing pain. ... the patient is at first requested to walk less than this distance, so that the pain is not reinforced following the walk. Each day the walk is lengthened a little, and, not surprisingly, the person walks beyond the original tolerance limit without experiencing the rest-demanding pain (p. 261).

The rising popularity of Behaviour Therapy, particularly during the 1950s and 1960s, helped to open the way for a comprehensive range of therapies, which, unlike Psychoanalysis, seek to produce within short periods of time, therapeutic change in the

patient's psyche and behaviour by focusing treatment primarily on presenting symptomatology. Combined with Cognitive Therapy, and being spared the often very lengthy and therefore costly treatment of Psychoanalysis, Cognitive Behaviourism has become the dominant therapeutic approach for a broad spectrum of psychological complaints during the past 30 years (Comer, 1995; Graham, 1990; Guidano & Liotti, 1983).

This trend, to pragmatically de-emphasise the 'absolute' need for psychoanalytic causal insight, is further demonstrated by the fact that in recent editions of the DSM publication, disorders are defined by symptoms without reference to specific causes (Comer, 1995). The current focus is on more immediate, self empowering management techniques such as biofeedback, relaxation and hypnosis (Pitty, 1994).

Ego-state therapy differs from other psychodynamic therapies in that it adopts the unique approach of using hypnosis to facilitate communication between the various ego-states of the individual, seeking thereby to restore a harmonious and balanced functioning of individuals and their coping mechanisms. Ego-state therapy has been found to be effective in the treatment of a comprehensive range of psychological ailments (Phillips, 1995; Watkins & Watkins, 1990), including migraine pain. Emmerson and Farmer (1996) conducted an ego-state treatment based study with 10 menstrual migraine patients. Results showed that the average number of migraine days were reduced from 12.2 to 2.5 per month; significant improvements were also achieved in lowering depression and anger, and increasing extroversion.

Successful treatment of psychosomatic pain disorders such as migraine, is particularly dependent on an holistic approach incorporating, or totally comprising

psychological therapy (Comer, 1995; Ivey, 1988; Lance, 1978, 1993; Rose, 1990; Siegel, 1991). The general effectiveness of hypnosis in the management of pain (see section 3.45) further substantiates this approach.

### 2.32 Biofeedback

Biofeedback is a visual and auditory feedback technique about physiological changes produced at will by the patient. It focuses on muscular relaxation and biological processes associated with headaches, including migraines. Evans (1988) refers to it as a type of electronically based extension of relaxation training. The principle involved in providing this feedback is a motivational one based on the concept that given a goal to be achieved it helps to know how successful we are in reaching that goal, so that continuity can be incorporated to ensure success.

Biofeedback may be facilitated with an electromyogram (EMG) of the frontal and temporal muscles, or from charted skin temperatures taken of the scalp and hand, or alternatively, from recordings of the temporal artery pulsations. Electrocardiograms (ECG), computers, and storage oscilloscopes can extend this range of biofeedback apparatus. Although some researchers may consider one procedure superior to others, the general consensus seems to be that all methods help promote relaxation (Lance, 1993).

Using biofeedback techniques in their ground breaking study, Friar and Beatty (1976) trained 19 migraine patients suffering at least 5 migraines attacks each month, to constrict their temporal arteries. The control group was taught to constrict the arteries in their hands. This was expected to lead to a non-specific placebo effect.

Pulse rates were recorded from the skin surface with two pressure transducing plethymographs, one of which was held in place over the temporal artery with an elastic band, and the other was taped onto the ventral side (nearest to the thumb) of the index finger. An electrocardiogram was recorded simultaneously, and the skin temperature was obtained with the use of surface thermistors adjacent to the pressure sensors.

The pulse rate from the temporal artery of the experimental group and the finger artery of the control group, together with the readings from the electrocardiograms were fed into a computer which then produced a stable pulse pressure wave form on a storage oscilloscope. This wave form was used as visual feedback for each subject, and an auditory signal from the computer was given when pulse amplitudes were less than the average of the 20 preceding amplitudes.

Subjects received a total of eight training sessions over a three week period. The results showed that the experimental subjects were able to autogenically reduce the pulse amplitude in their temporal arteries, and significantly decrease the total number of migraine attacks, with an even greater decrease in the number of fully established migraines.

The control subjects, though able to reduce the pulse amplitude in their peripheral artery, did not affect the blood flow in their temporal arteries. They were only able to achieve insignificant decreases in each of the above measures. Neither group reported a decrease in the intensity of the pain, yet both groups used significantly less medication for the period of the study.

A more recent development facilitates the monitoring of blood flow through the temporal artery by measuring the light reflected from the skin above it (Rapoport &

Sheftell, 1991). The photoplethysmograph, which monitors the light reflected, uses a photo-electric cell to transmit light to a diode which in turn emits light. The greater the flow of blood through the artery, the more opaque the skin, and the lower the intensity of light reflected.

Rapoport and Sheftell (1991) report a study comprising four groups of subjects. Each group received training in one of the following four conditions: (a) a combination of biofeedback and relaxation techniques, (b) biofeedback only, (c) relaxation only, or (d) placebo only. Results showed that 65% of the subjects in the combination condition found headache relief, 52% of the subjects trained in biofeedback alone, also found headache relief, 53% benefited from relaxation alone, and 17% even benefited from the placebo condition. Results of a second study reported by the researchers, showed that of 154 headache patients, 95% reported that biofeedback had given them some ability to remain calm during the headache itself. Nearly 50% had some success in stopping their headaches, but only 8% of those patients who suffered migraines were successful in doing so.

Herzberg (1994) comments that the Neurologic Centre for Headache and Pain in La Jolla California has used biofeedback with considerable success. After training patients for an average period of 14 sessions, patients were able to reduce the frequency and severity of their attacks by 87%. There can be little doubt, that biofeedback training is a useful and effective therapy in the treatment of headache pain and muscular relaxation (Andreychuck & Skriver, 1975). Evans (1988) points out that biofeedback, like relaxation, is an active form of therapy. A large measure of its effectiveness, may be ascribed to this fact of actively involving the patient, and to the emotional reassurance

and increased self-esteem gained from witnessing one's exercised control over certain physiological functions.

De Good (1995) points out that biofeedback, relaxation, and self-hypnosis, have the advantages of being self-regulatory behavioural techniques, which empower the individual. He also states that about 50% to 70% of chronic pain sufferers derive benefits from these techniques, and further, that these figures are similar to the response achieved from drug therapy. The effectiveness of biofeedback, particularly electromyograph biofeedback training, in the treatment of chronic headaches is also supported by Blanchard (1994).

### 2.33 Relaxation

One of the most significant things that has happened on the health front in more recent times is the realisation that stress can have very damaging effects on the body as well as the mind.... If you wish to master stress, the answer lies in learning techniques that help you relax in all situations of life (Jackson, 1989, p. ix, x).

There is now increased awareness and general agreement among physiological and psychological health professionals, that relaxation is a very effective way of coping with stress and its related disorders (Herzberg, 1994; Lance, 1993; Rapoport & Sheftell, 1991; Sheehan, 1983; Stanton, 1988). The physiological response to stress, or to perceived danger, is one of arousal. It prepares the body for 'fight or flight,' a process also referred to as the 'general adaptation syndrome.'

This adaptation, which occurs with remarkable speed and intensity, relies on the release of vasoconstrictive chemicals such as adrenalin and noradrenalin into the bloodstream. Intended as short term responses, these increased chemical levels are

normally depleted through the extra muscular activity required to fight the danger or flee from it.

When such muscular activity does not follow, as in cases of chronic stress, the chemicals build up producing a chronic state of muscular tension, and with it, a wide range of stress related diseases. For example, if blood vessels remain constricted and hypertension remains high, predisposed individuals may respond either with tension headaches, migraine, or another serious illness, such as cardiovascular disease in the form of heart attacks or strokes.

Even individuals not so predisposed will ultimately respond to prolonged stress with physical and mental fatigue, and a debilitated immune system which leaves the individual susceptible to invasive organisms, and to physiological and psychological burn out. "The importance of deep physical and mental relaxation is now well recognized as an important antidote to stress and its accompanying physical and psychopathology" (Brooking et al. 1992, p. 588).

Evans (1988) points out that the use of relaxation for combating physical and psychological ailments is not a new concept, and that Edmund Jacobson successfully used these techniques in America during the 1930s. Jacobson studied the effects of mental activities on muscular tension. He found that in a relaxed state, muscles register a different electrical potential to that found in states of muscular contraction. He subsequently taught patients to recognize their degree of muscle tension and control it with relaxation.

De Good (1995) stresses that from a cognitive-behaviour perspective, it is important that patients think of their self-relaxation as a coping skill rather than treatment. This

emphasises their self-empowerment rather than their role as patient. Andrasik, Blanchard, and Neff (1984) have shown that reinforcement of relaxation and biofeedback training, as well as regular contact with patients enhances long term effectiveness.

Comer (1995) cites a tension headache study which compared verbal progressive relaxation training, electromyograph (EMG) feedback training, and placebo medication. Each group comprised nine participants. The relaxation was focused on easing muscle contraction in the neck and scalp, commonly associated with tension headaches. The biofeedback group and the relaxation group each received eight one-hour training sessions. The placebo group received placebo medication only.

Results showed that the relaxation and biofeedback groups achieved similar, statistically significant improvements. The improvement achieved by the placebo group, however, was not statistically significant.

Evans (1988) also reports on a study by Hay and Madders into the effects of group relaxation therapy on migraine. All of the 98 patients participating in the study suffered at least two severe migraine attacks per month, and none had responded to drug therapy. Treatment for the participants consisted of six evening sessions in relaxation training followed by group discussions.

Results showed that 69 patients (70%) experienced fewer, shorter, and less severe attacks. The remaining 29 patients (30%) reported no benefit. This was attributed primarily to the difficulty some patients experienced in achieving muscular relaxation at will. Such difficulty, which is not uncommon according to Dr. Ainslie Meares (1977), may be due to some people having a basic reluctance to 'let go,' and others find it

difficult to believe that simple relaxation can help where more sophisticated methods such as drug therapy, have failed.

Stanton (1988) has defined three processes for successful relaxation: (a) mental relaxation, (b) muscular relaxation, and (c) slow deep breathing. Muscular relaxation is often seen as the means to attaining control of thoughts and emotions. This belief is based on the premise that it is difficult to be emotionally upset without muscular tension. The author also contends that relaxation of some parts of the body is considered more vital than others. Hands, forehead, jaw, and the abdomen are the key areas for muscular tension, and are therefore most in need of relaxation.

Stress responses, in a deeply relaxed state, are negated. The sympathetic nervous system slows down, noradrenaline and adrenaline levels return to normal, vasoconstriction is relieved, blood pressure and oxygen consumption decrease, heart rate stabilizes, brain waves become more synchronized and may register deep rest alpha patterns, and energy levels are replenished. Several techniques may be employed to achieve relaxation. Rapoport and Sheftell (1991) list a number of effective techniques, the best known of these are the following four techniques:

1. **Deep Breathing.** This technique is essential for relaxation and for providing oxygen to the blood which in turn aids circulation of nourishment to body tissues. The extra oxygen intake facilitated by deep slow diaphragm breathing effectively relaxes and energises brain cells.

2. **Progressive Relaxation** is a process by which first one, then another set of skeletal muscles are relaxed. The method of achieving relaxation may involve a direct 'letting go' of specific muscular tension, or the indirect method of 'deep muscle

exercises' which calls for the muscles to be first contracted and then relaxed. The latter method is employed to create awareness of tension contrasts and of the individual's own ability to influence that tension at will.

3. Autogenic Relaxation involves repetitious use of self-directed phrases such as, 'I am very relaxed,' or 'I feel calm and at ease.' This method is often used in conjunction with muscular relaxation, and like hypnosis, is based on the experimental evidence that the mind is particularly open to suggestions in a relaxed state. Rapoport and Sheftell (1991) refer to autogenic relaxation as "...a type of self-hypnosis. You talk yourself into a relaxed and positive state" (p. 155).

4. Passive Relaxation with guided imagery is, strictly speaking, hypnosis. This will become evident in the course of chapter 3, and more specifically in sections 3.31 and 3.32. It has long been the practice of some health and counselling professionals, who, despite no specialist training in hypnotherapy, use these techniques under the description of 'relaxation.'

Rapoport and Sheftell (1991) describe passive relaxation as a state of mind in which the individual no longer actively tries to relax, but simply allows thoughts to pass through without addressing them. Passive relaxation is the ultimate goal of many forms of meditation. Guided imagery is described as a process of imagining oneself in restful and relaxing surroundings such as lying on a beach. The authors point out that with practice, simply calling up the image can trigger a relaxation response.

This behavioural conditioning aspect, in the elicitation of hypnotic relaxation, can be simply expressed in terms of: Given repeated exposure to a relaxing stimulus, relaxation may be spontaneously elicited as a conditioned response. It is primarily the

conditioned behavioural response, which forms the simple, yet unique, basis of post-hypnotic suggestions discussed in section 3.33.

With hypnosis, guided imagery need not be restricted to the mere attaining of a relaxed state. It may also be effectively used while in that state to produce suggested physiological and psychological change. It is largely this two-fold capacity of hypnosis which makes it an effective therapy, particularly in the control of pain. This important aspect of hypnosis is discussed in sections 3.43 and 3.44, and its use in the treatment of migraine pain, in section 3.5.

## CHAPTER 3

### Hypnosis and Hypnotherapy

#### 3.1 The History of Hypnosis

The history of hypnosis, although relatively short in the clinical sense in which it is known today, reveals the many struggles which beset its early days prior to full professional recognition. To do justice to its historically important developments, even a highly condensed version of these difficult, but enlightening periods, requires exposition of its major historical events. With this in mind, and recognizing the need for selective brevity regarding some issues discussed in research theses, this history exposition has been divided into two parts, namely, early and recent history. An understanding of the progress of hypnosis to the present, places in context the usage of this modality as treatment for migraine and aura.

##### 3.1.1 The Early History of Hypnosis

The early history of hypnotic trance goes back to ancient forms of civilization. The healing temples of Egypt in the fifth century BC, also known as 'sleeping temples,' are known to have served not only as places for spiritual cleansing and enlightenment, but for performing healing rituals involving suggestions and curative sleep. The success of these healing services are believed to have contributed to their spread to Greece and the Roman Empire (Emmerson, 1987).

Medical historians have been inclined to tie the origin of hypnosis to the magnetic healing practices of Franz Anton Mesmer during the second half of the nineteenth Century. There is some evidence, however, that belief in the healing powers of the

loadstone magnet was widespread during the Middle Ages, and 'qualified' physicians who used this treatment were known as magnetisers (Karle, 1992). Nevertheless, there can be no doubt that the publicity and notoriety surrounding Mesmer's healing practices ultimately led to a better understanding and development of the curative effects of particular aspects of his practices, namely that of trance induction (Milne, 1995; Shone, 1982).

Anton Mesmer who was a practicing physician in Vienna during the 1770s, lived from 1734 to 1814. He conducted his first magnetic treatment in 1774 as an alternative to traditional medicines or surgery. He shared the belief of many of his contemporaries that the human body is greatly influenced by the planets and the magnetic powers of the solar system. Mesmer also believed that a healthy body depended on an uninterrupted flow of a magnetic fluid, which he called 'animal magnetism,' and therefore, disease was the result of blockages of the magnetic fluid in the system (Jackson, 1989).

These premises led to the conclusion that the blocked magnetic fluid could be freed and made to flow again by passing magnets over the body. Mesmer also believed that magnetic cures could be achieved in indirect ways by adopting rituals involving patients tying themselves to 'magnetised' objects such as trees, or touching iron rods partly immersed in tubs containing water, glass bottles and iron filings (Karle, 1992).

Records about these rituals state that patients frequently experienced seizures or other violent reactions, or fell into a deep sleep. Many patients are said to have made remarkable and dramatic recoveries from a wide range of illnesses. In the light of these descriptions, there can be little doubt that Mesmer's patients entered into a state of

hypnotic trance during these rituals, induced by any one or a combination of ritualistic factors.

Mesmer moved to Paris in 1787, where he achieved considerable fame during the next five years, but notoriety and suspicion was to follow. Although he was not to obtain recognition from La Societe Royale de Medecine, the French Government, supposedly at the suggestion of Marie Antoinette, offered him a life pension and funds to set up a clinic. This offer, however, was made with the proviso that the management of the clinic would be subject to the supervision of Government representatives, Mesmer declined the offer (Karle, 1992).

During the years following, controversy and rumours flourished with spectacular cures being claimed and just as frequently denied until 1784, when Louis XVI appointed a Royal Commission to investigate these claims. The Commission comprised of five members from l'Academie Franchise and four doctors from the Faculty of Medicine at the University of Paris (interestingly, the American scientist and statesman Benjamin Franklin was among them). Mesmer's animal magnetism procedure was demonstrated to the Commission as part of many diverse experiments.

The Commission's final report stated that the claimed effects of animal magnetism and magnetic fluids were ascribable to the imagination and nothing else. The report from the Commission and that from the Committee of La Societe Royale exerted considerable influence throughout the world. The practice of mesmerism was banned and Mesmer returned to Vienna a disgraced practitioner (Milne, 1995).

Although the Royal Commission of 1784 failed to recognize the true nature of hypnotic trance and its benefits, it nevertheless made an accurate assessment of the role

of the imagination in hypnotic trance. In hypnosis the imagination is actively involved in producing physiological changes, but this is not to say as the Commission's finding was meant to imply, that any healing and cures effected in this way, are mere figments of the imagination. Today, many practitioners of the medical and psychological professions understand this vital difference and make excellent therapeutic use of it. Milne (1995) comments that since the imagination is one of the important elements in the production of therapeutic trance, it was most unfortunate that the Commission chose to investigate the wrong aspects of Mesmer's work.

Although Mesmer believed strongly in his theory of magnetic fluids, it is clear that he was also aware of the important and constructive role played by the imagination. Karle (1992) quotes one of Mesmer's pupils, Charles d'Eslon: "If the medicine of imagination is best, why should we not practice the medicine of imagination?" (p. 14). But it seems that the belief in magnetism distracted Mesmer as well as the Commission from what was really happening. The Commission as well as the Committee of La Societe Royale appear to have been blind to the remarkable physical changes had occurred in patients treated by Mesmer's method, and that the cause of this warranted further investigation.

The indications are, that without being aware of it, Mesmer created hypnotic trance states in his patients. His rituals of having the patient lie quietly while hand passes were made repeatedly over the length of the body, were accompanied by implicit and explicit suggestions of curative powers. These rituals would most likely have focused the imaginative powers of his patients away from their surroundings and singularly onto their impending cures, thus facilitating a state of hypnotic trance, and with that, the

opportunity for healing suggestions to work. There exists little doubt among health professionals that Mesmer's methods induced a state of hypnotic trance (Fisher, 1992; Shone, 1982; Straus, 1988).

Although the failure to fully investigate the underlying causes of Mesmer's healing successes has held back the development of hypnotherapy by nearly a century, interest in mesmerism was to be revived by a number of physicians in Europe in the 19th Century (Karle, 1992). In Britain, three major figures are traditionally associated with the revival during this period, namely, John Elliotson, John Esdaile, and James Braid.

John Elliotson, was born in 1791, and trained as physician and surgeon in Edinburgh, and later practiced in England. He used trance states for their analgesic powers, particularly during and after surgery. Although Elliotson was considered one of Britain's most able physicians, his growing and public interest in Mesmerism led subsequently to his rejection by the medical establishment, including the *Lancet*. This led Elliotson to publish his own medical journal the *Zoist*. The journal specialized in articles about the central nervous system and its functions, and related subject matter about practices in mesmeristic trance states. He is said to have been the last Mesmerist and forerunner of medical hypnotists soon to emerge.

Emmerson (1987) quotes Elliotson in his capacity as editor in the first issue of the *Zoist*:

The science of mesmerism is a new physiological truth of incalculable value and importance; and though sneered at by the pseudo-philosophers of the day, there is not the least certainty that it presents the only avenue through which is discernable a ray of hope that the more intricate phenomena of the nervous system-of-life will

never be revealed to man. Already it has established its claim to be considered a most potent remedy in the cure of disease; already enabled the knife of the operator to traverse and divide the living fibre unfelt by the patient (p. 25).

The Scott, John Esdaile, who lived from 1808 to 1859, was another surgeon interested in mesmerism. He practiced in India during the 1840s and is credited with having demonstrated the analgesic effectiveness of hypnotic trance most conclusively, although for him, the credit for the outstanding analgesic effects belonged to the realm of magnetic fluids. Like Mesmer before him, he used 'Mesmeric passes' over the body, believing that doing so enabled him to diffuse the magnetic aura and thus produce insensitivity to pain. It is obvious that the true nature of this method eluded him, as it did Mesmer (Milne, 1995).

Esdaile performed many surgical procedures, including limb amputations and the removal of tumors of the scrotum due to Elephantiasis, which was endemic in the region. Some of those tumors are said to have been so large that other surgeons refused to operate. Esdaile's success is ascribed to his use of hypnotic trance, which reduced, if not prevented, pain and shock to the patient so that the resulting mortality rate was only 5% instead of the usual 50% (Milne, 1995).

### 3.1.2 The Recent History of Hypnosis

James Braid is considered the 'Father of modern Hypnosis' and is credited with having put hypnosis on a scientific footing. Braid was also a surgeon. He lived from 1795 to 1860. His book published in the middle of the nineteenth century was titled 'Neurhypnology or the Study of Nervous Sleep.' From 'neurhypnology' he derived the

words Hypnosis and Hypnotism. Braid too, experimented with magnets but found them lacking in therapeutic benefits.

Recognizing that the phenomena of trance can produce analgesic and healing effects, he rejected mesmerism and animal magnetism, calling his method 'neuro-hypnotism.' He did, however, initially believe that hypnosis is a form of nervous sleep, which he likened to a state of 'fascination' or 'paralysis,' sometimes witnessed in animals when confronted with extreme danger. Unfortunately this misconception still exists today in large parts of the community. Scientific evidence based on Positron Emission Tomography (PET) has clearly demonstrated that hypnosis is not sleep (Sarbin & Slagle, 1993). Details of this aspect are discussed in section 3.2.

Braid correctly stressed the importance of the patient's responsiveness, rather than the power of the hypnotist. His most important contribution to modern hypnosis, however, was his recognition that hypnotic trance can be induced and deepened without rituals and objects, by using verbal suggestions only, and further, that suggestibility increases with the depth of hypnosis (Karle, 1992; Milne, 1995).

Milne (1995) suggests that the increasing practice of hypnosis in response to its demonstrated effectiveness as a natural anaesthetic suffered a set back with the introduction of chemical anaesthetics. Chloroform had found favour with Queen Victoria in 1853 after it was administered to her during the birth of her eighth child, Prince Leopold. However, despite this set back, hypnosis continued to be practiced and taught by a large number of physicians both in Britain and on the continent. In France, hypnosis was studied extensively, leading, in the last quarter of the nineteenth century, to the formation of two separate schools of thought (Karle, 1992).

The Salpetriere school, under Professor Jean-Martin Charcot from the Faculty of Medicine, and Professor Babinski, advocated explanations about hypnosis primarily in physiological terms, rejecting out of hand such psychological aspects as the imagination, but admitting the psychological stimuli of suggestion. The Nancy school under Professor Hippolyte-Marie Bernheim took a pragmatic, therapy oriented view, admitting psychological aspects only, particularly with regard to suggestion and the imagination (Chertok, 1993).

Charcot conducted extensive work researching a perceived link between hysterical disorders and hypnotic trance states. He presented a paper to the French Academy of Sciences in 1882, which led to further investigation of his theories some years later, and to the finding that they were inaccurate. His scientific contributions on the subject remain acknowledged however, more so, since they inspired interest and research into the use of hypnosis in the treatment of mental illness in the last quarter of the nineteenth century (Karle, 1992).

The results of Bernheim's research, published in 1884, scientifically supported the hypothesis that hypnosis works through the power of suggestion. His treatment appears not to have been very different from modern practices, even though scientific contributions to current knowledge about the psychological and physiological responses occasioned by these treatments is understandably, quite different from his given explanations (Chertok, 1993).

During the period from August 8, 1889 to August 12, 1889, the First International Congress of Experimental and Therapeutic Hypnotism took place in Paris. During this summer, Paris also played host to the International Congress of Physiological

Psychology, an important part of which dealt exclusively with hypnotism. Mirroring the continuing controversial debates between the Salpetriere and Nancy schools, the latter congress was concerned only with psychological aspects of hypnosis, whereas the interest of the former was largely restricted to medical aspects (Milne, 1995).

These international forums offered excellent opportunities, as they do today, to discuss work and scientific research on the subject of hypnosis. Attendance records revealed the names of many historically notable persons present at both of these congresses. They included Babinski, Bernheim, Charcot, Freud, Janet, and Korsakov (Chertok, 1993).

Chertok (1993) reports that at the First International Congress, Bernheim stated clearly that the hypnotic state is a particular mental state which, once induced, increases suggestibility; that is, it produces the tendency for the brain to be influenced in accepting an idea and to transform it into action. At the Congress of Physiological Psychology Bernheim maintained similarly, that Hypnosis is nothing but suggestion, and that all the effects of hypnosis can be obtained without sleep.

Chertok goes on to say that Babinski, representing the Salpetriere school defined hypnosis as a bodily state with objectively identifiable physical signs which could be induced by either psychological or physiological stimuli from outside the organism. Despite the incompleteness of their explanations, both schools contributed to hypnosis and beyond, to the development of psychoanalysis and psychotherapy, primarily through their influence on Freud and Janet (Karle, 1992).

Freud had studied the use of hypnosis in the treatment of psychological disorders under Charcot at the University of Paris. He made use of the teaching by Charcot that

hysteria is primarily a mental disturbance in which traumatic memories play a fundamental role, and he learned from Bernheim about the existence of so-called powerful mental processes 'hidden from the consciousness of man.'

Freud's acceptance of these theories led him to develop his theory on the Unconscious. Although Freud practiced hypnosis for a number of years using Bernheim's techniques to recall traumatic memories into consciousness, or to retrieve them from the unconscious, he was soon to replace this practice with another of his new developments; the method of free association (Karle, 1992; Milne, 1995).

During an experiment on post-hypnotic amnesia conducted by Bernheim, the existence of a superficial type of amnesia was demonstrated; thus building evidence supporting the psychological aspects of hypnosis. It was shown that on the mere strength of the experimenter's suggestion and his touching the subject's forehead, the amnesia was removed and the forgotten facts and connections were readily recalled by the subject. This experiment thus demonstrated that memory could be manipulated. This led Freud to formulate his dynamic notion of repression and such related concepts as symptoms formation, and psychosomatic (hysterical) pain disorders.

Janet, like Freud, was similarly inspired by the work of Charcot and Bernheim. His interest too, went beyond mere physical manifestations of hypnosis into the psychological aspects of treatment. Travelling a similar path to Freud, he came to distinguish different modes of mental distancing. His theory *L'Automatisme Psychologique* was published in 1889, and has been credited with laying the groundwork for the psychological unconscious, and with having anticipated certain ideas current in modern psychosomatic theories (Chertok, 1993). For example, Janet believed that the same psychological

disorder can manifest in physical and in mental form; paralysis and amnesia can be different manifestations of the same disorder, so that paralysis can be the amnesia of a limb.

The work of Janet and Freud and other contemporaries like Breuer in Austria, (who gave the name ‘catharsis’ (Greek for cleansing) to his method of hypnotic age regression, and the German word of ‘abreaktion’ (abreaction) to the painful release of the symptom causing stimuli), did much at the turn of the twentieth century to advance both the professional credibility of hypnosis and the development of psychoanalytic theory. Hypnosis, despite its recognition by the British Medical Association in 1892, continued to wane and wax in professional acceptance and popularity, whereas the practice of psychoanalysis, despite its comparatively slow and uncertain effectiveness, embarked on a steady, if slow climb to recognition, at least for the next four decades (Karle, 1992).

The British Medical Association published, in 1892, the results of an investigation it had commissioned into ‘the nature of the phenomena of hypnotism, its value as a therapeutic agent, and the propriety of using it.’ The Committee conducted experiments which satisfactorily demonstrated that hypnosis was real and that it did have physical effects which could be of benefit to some patients.

It was also concluded that “hypnosis has real value in the control of pain, in inducing sleep, and in alleviating many ailments not stemming from physical damage” (Karle, 1992, p. 16-17). These findings clearly illustrate the knowledge shared today, that hypnosis is particularly effective in the treatment of psychophysiological or psychosomatic disorders.

Despite the Committee's recognition and recommendation that hypnosis was a proper treatment modality to be used by medical practitioners, interest in it, at least on a larger scale, only emerged during the 1930s. This revival involved academics, scientists, and medical practitioners. Since then a great deal of scientific research and clinical studies have been carried out in university departments and hospitals throughout the world (Chertok, 1993; Karle, 1992).

The Society for Clinical and Experimental Hypnosis was founded in the United States in 1949. Officers of that Society later founded the International Society for clinical and Experimental Hypnosis (ISCEH) with worldwide membership. The ISCEH was itself reconstituted as the International Society of Hypnosis (ISH) (Watkins, 1995). The British Medical Association in 1955, commissioned another investigation into hypnosis, and as a result, the Association reiterated the findings and conclusion of its 1892 report, giving full approval of the use of hypnosis in the treatment of both physical and psychological disorders.

The American Medical Association followed suit in 1958. Milne (1995) comments that before the end of the 1950s, both the British and the American Medical Associations had issued policy "It was some years before the infant discipline of psychology was to be legitimised in this way, especially in Britain and Australia" (p. 9).

Despite the extensive and increasing use of hypnosis by doctors, dentists, and psychologists, and the growing body of research undertaken in universities and hospitals, there remains a lot to be learned about how the psychological and physiological powers of hypnosis operate to produce benefits. We still do not understand the causal sequences involved in psychological/physiological interaction. But what is empirically supported, is

that physiological changes do occur in response to psychological, hypnotic suggestions; these may include changes in temperature, blood pressure, or pulse rate (Benson, 1975; Sarbin and Slagle, 1993).

### 3.2 The Nature of Hypnosis

#### 3.21 The Hypnotic State

Hartland (1982) described hypnosis as a state of mind in which suggestions are more readily accepted and more powerfully acted on than would be possible under normal conditions; in other words, the hypnotic state is characterised by an increase of suggestibility on the part of the subject. This interpretation is a generally accepted one and is supported in Dorland's Medical Dictionary (1982), where hypnosis is defined as: "...an artificially induced passive state in which there is increased amenability and responsiveness to suggestions and commands" (p. 293).

A concurring but more detailed definition is provided by the Australian Society of Hypnosis in its Hypnosis Registration Act 1996 which states that:

Hypnosis means the act or process by which... the hypnosis practitioner induces... in an other person, an altered state of attention or degree of awareness, in which a variety of phenomena appear spontaneously or in response to verbal stimuli. These include; alterations in consciousness and memory; increased suggestibility.. production of responses and ideas unfamiliar to [the subject's] normal state of mind; or changes in the behaviour... or physiological processes of that person.

Responding to the broad term, 'altered state of consciousness' Fromm and Hurt (1993) pose the legitimate question "...how are we to begin to understand these alterations?" (p. 14), and suggest, that these alterations are [temporary] reorganisations

of the cognitive structures which serve as the foundation of all our conscious experience. The cognitive structures are perception, cognition, alteration, memory organisation, and emotion of effects. They control the ways representations of the inner and outer worlds are structured in all forms of consciousness. The authors contend further, that an understanding of these alterations of consciousness, can be achieved via the Freudian distinction between 'primary processes' and 'secondary processes' in mental functioning.

The primary process is interpreted as characteristic of internalised instinctual gratification, of uncomplicated thinking more in the forms of fantasy and imagery than logical sequence and analysis. By contrast, the secondary process flows from the impact of external reality and is characterised by cause and effect analysis and conceptualisation, and by adaptation to the external world. Both types of processes continue to interact and can facilitate cognitive reorganisation resulting in altered states of consciousness.

This theory stresses the importance of a genetic component which attaches to the instinctual gratification drive of primary process thinking. Responding to the absence of the real gratification object, the instinctual drive attempts gratification through the imagined object or, by extension of this line of thought, by transference. It is further suggested by the authors, that this genetic aspect of the primary process clarifies the importance of the imagination and fantasy in hypnosis, as well as its seeming reality and emotional impact.

Freud believed unconscious motivational factors, and transference, to be causes of hypnotic trance. But if, in accordance with the generally accepted current view that the essence of hypnosis can be reduced to a state of suggestibility, then neither the concept of transference nor that of unconscious motivational factors, is a sufficient explanation of

this suggestibility. Some hypnotists might even be inclined to argue that within the context of hypnosis, transference, like suggestibility or age regression, is the result, rather than the cause of the hypnotic state (Chertok, 1993).

What is stressed as important in achieving and maintaining a state of hypnotic trance, is attentive focal concentration on the suggestion given. This is best facilitated in relaxed states in which patients execute, or let their body execute, the succession of ideas presented to them. At this point patients may ignore, or at will, misinterpret messages from their own body and accept as real, the suggestions presented to them by the hypnotist (Sacerdote, 1970).

Sarbin and Slagle (1993) support this view by suggesting, that hypnosis calls for expression of belief in a counterfactual proposition, even in the face of evidence to the contrary. And further, that hypnotic subjects perform actions consistent with their perception of their current role, given the limitations imposed by identifiable dependent variables. McConkey (1990) writes that even when hypnotic subjects process information which is consistent with the suggested condition, there is a strong tendency on their part to process the given data in a way that will reinforce, rather than challenge their belief in the suggested condition.

Considering these views, it may be said that hypnotised subjects interpret suggested data in a way which is biased toward reinforcing their perceived role in the hypnotic state. This view raises the question of role play in hypnosis. If hypnotised subjects perform actions in accordance with their perceptions of their current role, to what extent, if any, does this role play constitute hypnosis? Is it a mere compliance with the subject's perceived expectations of the hypnotherapist?

Regarding the influence of artefact in hypnosis, Sheehan and Perry (1993) commented, that demand characteristics in test situations may influence subjects' responses, and that the effect of subjects perceiving cues as to what they should do, is now widely recognized. Sarbin and Coe (1972) presented the argument that role play is likely to occur at both conscious and non-conscious levels, and that the role play of hypnotised subjects may vacillate between these levels.

Erickson, Rossi, and Rossi (1976) expressed their view on some functional aspects of the hypnotic state, maintaining, that it allows the patient to function adequately and directly in an unconscious level of awareness without evaluation interference from the conscious mind. Orne (1993) expressed the view that, from a phenomenological perspective, mixtures of conscious and non-conscious role play are frequently encountered, especially in subjects in the mid-range of hypnotisability, but unlike Sarbin and Coe (1972), he does not claim conscious role play may occur in a state of hypnosis.

The issue of artefact, while also of interest in clinical settings, is, a minor concern. In clinical practice where patients have a vested interest in getting effective treatment for their problem, they are less inclined to feel a need for compliance with the perceived needs of the hypnotist. Given these circumstances, the therapeutic results achieved in clinical settings may be taken to be relatively free of artefact, attesting both to the reality and the effectiveness of hypnotherapy.

Orne (1993) suggests that the best clinical evidence for the subjective reality of hypnotic effects derives from the effectiveness of hypnosis in the treatment of chronic pain and as an anaesthetic. In these circumstances it is difficult to accept that simulating subjects would tolerate major surgical procedures without anaesthesia, or pretend that

their chronic debilitating pain has abated when it has not. Hilgard and Hilgard (1975) wrote that although other psychological and physiological factors may affect the expression of pain, the repeated choice of hypnosis as an analgesic, when alternative treatment options are available, is difficult to explain without accepting that the analgesic suggestions successfully change the individual's perception of pain.

Psychotherapists and experimental psychologists have made several theoretical attempts at synthesising physiological and psychological elements of hypnosis. Kraines (1969) advanced the theory that the monotonous, rhythmic, and repetitive sounds of the hypnotist's voice, act to synchronise input from the reticular system to the cerebral cortex, causing an actively inhibited state in that tissue. This cortical inhibition allows for evaluation processes to be by-passed and for verbal suggestions to set up conditioned reflexes (such as heaviness of limbs); thus paving the way for a post-hypnotic suggestion to act as a conditioned stimulus for a conditioned sensory and behavioural response.

Roberts (1960) suggested that the central area of excitation in hypnosis, is the central integrating system of the brainstem, and that the inhibition of interaction between projection and motor systems occasions and conditions hypnosis. Hypnotic induction according to Roberts, involves attentiveness to monotonous, repetitive, and rhythmical stimuli of one sensory modality. This hypnotic stimulation may be seen to suppress afferent input, which in turn inhibits behavioural arousal, but not electrophysiological arousal, which may account for the presence of normal EEG activity in the hypnotic state.

Viewed from the perspectives of ego-state theory, hypnotic phenomena are the result of a dissociation of the ego-state. This dissociation is triggered in the induction

process which facilitates a narrowing of focus and awareness of a single ego-state.

When this dissociation is achieved, certain sensations such as pain, may be experienced by another ego-state while the executive ego-state is unaware of the stimuli (Emmerson lectures, 1997).

### 3.22 Sensory and other Phenomena of Hypnosis

The sensory phenomena which may be experienced in hypnotic states include olfactory, auditory, tactile, taste and temperature variations, and visual hallucinations such as colour blindness. Distortion of physical and temporal dimensions, age regression, amnesia, analgesia, and anaesthesia remain of particular interest to clinicians and researchers, primarily because of the unique nature of these phenomena, and in the case of analgesia and anaesthesia, also because of their recognized treatment value (Graham, 1990; Milne, 1995; Rose, 1990).

The actual experience of these phenomena is accepted as subjectively real in the sense, that hypnotically induced colour blindness is not colour blindness in any objective sense, and that hypnotic analgesia and anaesthesia do not, according to Hilgard and Hilgard (1975), in themselves eliminate the source of pain. The change of specific sensory awareness occurring in these hallucinatory states is taken to be restricted to the psychological perception of the suggested experiences, a perception which becomes distorted in accordance with the suggestion given.

A clear case in point is the phenomenon of hypnotic age regression (Foenander & Burrows 1993). When an individual is regressed to an earlier age, it is clearly not the case that intellectual capacities have become functionally regressed in any real sense, or that the individual shrinks in physical dimensions. Age regression may also serve as a

prime example of non-conscious role play. As in age regression, the question awaiting clarification in time distortion is not whether it is objectively real, but how, and the extent to which the neurological conditions which facilitate distortions of age or time perception, undergo physiological changes during these and similar hypnotic experiences, such as amnesia.

Spiegel and Spiegel (1978) comment that amnesia, for the suggestion or instruction given, is part of what they have termed 'the compulsive triad' of responses which hallmark the hypnotic state; the other responses being compulsive compliance, and rationalization. For example, a subject responding to a signal that his right hand will float up in the air will compulsively comply with this suggestion, but rationalize his compliance with a self statement of the kind that this is due to the circulation in his right hand being different from that in his left hand because of the way he was resting on the chair. The authors continue, that it is not merely the change produced by the response which is important in hallmarking or typifying the hypnotic state, but the compulsiveness of the responsiveness to the instruction, the amnesia for the context of the original signal, and the rationalization for the change itself.

The phenomena of hypnotic analgesia and pain control which may be experienced in light to deep levels of trance are of particular interest to this research are. One aspect of the psychological complexities involved in hypnotic pain control is demonstrated in a series of 17 caesarean section case studies using only hypno-anaesthesia. Twelve of these new mothers requested chemical anaesthesia only after their baby had been extracted. One mother explained that her request was occasioned by her emotional

excitement and elation after the birth of her child, when she could no longer control her pain (Stone & Burrows, 1993).

There are many reports, in addition to surgical anaesthesia, on the efficacy of hypnosis in minimizing and eliminating pain resulting from such other causes as dentistry, back pain, burns, and headaches (Gardner & Hinton, 1993). Finer, Hallin, and Torebjorg (1978) were able to demonstrate some aspects of hypnotic and physiological interaction by showing that activity in sympathetic nerve fibres is related to the experience of pain under hypnosis. They suggested that this was indicative of modulating effects of hypnosis on both central and autonomic components of the pain response.

### 3.23 Physiological Changes during Hypnosis

The hypnotic suggestion, “your right hand is getting colder and colder,” can actually facilitate the temperature of that hand to decrease, or a suggestion to the contrary, can facilitate its increase. Unlike achieving a mere change in pain perception by focusing or attending to other stimuli, in this case it is not just a matter of changed psychological awareness of cold or heat on the part of the hypnotised individual. The changes produced by these hypnotic suggestions include verifiable, physiological changes in the vascular system, and in blood flow (Comer, 1995; Shone, 1982).

The theoretical difficulties encountered in the search for a causal theory which explains the psychological and physiological interactions in hypnosis within the parameters of materialist scientific structures, are at once evident and challenging. Sarbin and Slagle (1993) write that in an age when a description of the neural mechanism underlying even the simplest behavioural event is a monumental undertaking, “...it is indeed presumptuous to expect that a phenomenon so complex and controversial as

‘hypnosis’ can at this time be neurophysiologically elucidated” (p. 60). It appears clear that research into the psychophysiology of hypnosis can but hope to contribute to a causal elucidation in a slow manner.

Considerable research interest has been generated over the past two decades in hemispheric activity and differential hypnotic brain waves. Gruzelier (1986) argues convincingly that hypnosis is a right hemisphere function, more specifically, that subjects in hypnotic states exhibit behavioural functions now attributed by neurological research to the right hemisphere of the brain. In support of this view are the findings of Ardila and Ostrosky-Solis (1984), who found right hemisphere behavioural functions to be predominantly emotional, creative, spatial, sensory, and non-analytical, whereas left hemisphere functions were found to be associated with critical analysis, and verbal and logical tasks, that is, the type of behaviour not exhibited in hypnotic trance.

Gruzelier (1988), in a later paper presentation, maintained that the mechanism of entering an hypnotic state involves either a shift into right hemisphere function or an inhibition of the left hemisphere, or both. And further, that the neurophysiological mechanism available in the brain to facilitate this effect resides in the inhibitory fibres of the reticular formation. Galin and Ornstein (1972) recorded EEG alpha signals from the left to the right hemispheres simultaneously while the subjects did verbal or spatial tasks, and found that people tended to do verbal tasks using the left hemisphere of their brain, and spatial tasks with their right hemisphere. While these findings were not directly related to hypnosis, they nevertheless lend support to the theory that hypnosis is a right hemisphere function.

Brain activity can be measured by placing electrodes at various places on the scalp. These electrodes record in graphical detail electrical activity occurring in the brain. Different activities produce different wave patterns, labelled alpha, beta, delta, and theta (Carlson, 1986).

Electroencephalographic (EEG) changes in hypnotised subjects have been reported by several researchers. Sabourin, Cutcomb, Crawford, and Pribam (1990) found increased theta activity in hypnotised subjects in both high and low categories of hypnotisability. They also reported increased beta activity in the left hemisphere of highly hypnotisable subjects. These findings were supported, for various levels of trance, by Bauer and McCanne (1980), Bick (1989), and Sabourin et al. (1990).

However, in contrast to those findings, De Pascalis and Penna (1990) reported increased beta activity in the right hemisphere of highly hypnotisable subjects. Their right hemisphere findings were taken to be indicative of highly focused attention congruent with a deep state of hypnosis and right hemisphere activity. This conclusion was supported by their additional findings that low hypnotisable subjects showed decreased beta activity in both hemispheres.

Sabourin et al. (1990) reported increased alpha activity in both high and low level states of hypnosis. These findings supported the results of an earlier study by Ulliyet, Akpınar, and Itill (1974), who maintained that alpha activity can be used as a direct measure of the level of hypnosis. The EEG readings of their study also showed alpha activity to be significantly increased in hypnosis.

Morgan, MacDonald, and MacDonald (1971) reported a hemispheric shift from left to right in highly hypnotisable subjects. This activated shift from the left to the right

hemisphere was also found by MacLeod-Morgan and Lack (1982), Gruzelier (1986), and Meszaros, Banyai, and Greguss (1986), and was thought to demonstrate a progressive left hemisphere inhibition normally associated with the hypnotic process. On the basis of the foregoing studies, it is reasonable to conclude that the hypnotic state can be identified as normally being a right hemispheric function, and that it can be scientifically differentiated from a normal state by its increased alpha activity.

Other systems believed to be involved in the hypnotic state, include the ascending reticular activating and the limbic systems. Under Arnold's (1959) neurophysiological theory of brain function and hypnosis, the 'imagination set' is mediated by the hippocampal action circuit in connection with the thalamic system which, when stimulated, can bring about a state of general cortical inhibition associated with subjective drowsiness and hypnosis. It appears that the diffuse thalamic system, can inhibit cortical activity when stimulated with slow frequency relays. This inhibition of cortical impulses, inhibits critical appraisal and evaluation, which in turn facilitates acceptance of hallucinated images as real. Arnold (1959) pointed out that the mere imagining of a sensation or movement, can result in experiencing that sensation and movement as real.

Presumably, hypnotic stimulation suppresses afferent thalamic input and inhibits behavioural arousal, but not electrophysiological arousal. The thalamic inhibition theory differentiates between the subjective drowsiness of the hypnotic state and sleep, and as such, also helps to explain why EEGs in hypnotic states register wakefulness as opposed to sleep. The presence of EEG arousal without behavioural arousal may be maintained by delta waves possibly originating in the anterior hypothalamus (Sarbin & Slagle, 1993).

Further differentiation of hypnosis from sleep is indicated by research results published in the National Health and Medical Research Council Report (1982), which show that oxygen consumption and the basal metabolic rate remain normal in hypnotic states, but are reduced by 10% during sleep.

Hess (1949), a German physiologist, identified biopsychological changes in the hypnotic state to which he gave the encompassing title, 'Organische Gesamtumschaltung' or 'biopsychological shift to a trophotropic state' (Langen, 1993). This trophotropic shift toward conservation and restoration of energy during hypnotic relaxation involves the following changes: contraction of pupils, lowering of blood pressure, slowing down of respiration, metabolism, and salivation, as well as hypodynamia of the skeletal muscular system.

Psychological changes occurred in terms of increased passivity, with deep relaxation bordering on the transition stage to sleep, and accompanied by typical protective reflexes. Finally, metabolic processes became geared toward assimilatory functions for the conservation and restitution of energy. All of these observations have become well recognized as regular psychophysiological changes of the hypnotic state (Carlson 1986; Graham 1990; Siegel 1991).

Recent studies have shown melatonin, a catecholamine hormone released by the pineal gland, to play an active role in hypnosis and its related physiological changes, such as the slowing down of cortical rhythm, and analgesic manifestations (Graham, 1990). It was found that focusing of the eyes, as frequently practiced in some form during hypnotic induction, triggers a release of melatonin. This, in turn, releases the melanocyte-stimulating hormone (MSH) from the pituitary gland which slows down

cortical rhythm, facilitates memory retrieval via the hippocampus and hippocampal gyrus, and releases the opiate lymphomorphine, thereby increasing stress tolerance (Maestroni & Coti, 1991).

It is thought that hypnotically induced analgesia, such as glove anesthesia, which is resistant to modification by the opiate antagonist naloxone, is most likely mediated by melatonin via lymphomorphine (Goldstein & Hilgard, 1975). Melatonin, especially in conjunction with the neurotransmitters dopamine and serotonin, is also thought to induce a feeling of safety and ease as opposed to anxiety and panic (Kaplan & Sadock, 1991).

Hypnotic experiments are frequently designed to elicit and measure autonomic responses, such as changes in cardiac performance, in blood sugar levels, in bladder pressure, and gastric secretions. These and other physiological responses may be elicited through a number of psychological stimuli such as direct or indirect hypnotic suggestions.

The chemico-physiological effects of hypnotic suggestions were demonstrated by an early study conducted by Lewis and Sarbin (1943). This study showed that gastric hunger contractions could be modified or eliminated by the hypnotic suggestion that subjects were actually eating a meal. The fact that the stomach is innervated primarily by the autonomic nervous system, and only indirectly influenced by the central nervous system, helps to facilitate psychological manipulation of physiological symptoms (Graham, 1990).

Notwithstanding these insights, the quest for further elucidation on the psychophysiology of hypnosis continues. Sarbin and Slagle (1993) point out that in order to make sense of the many clinical and experimental observations linking hypnosis

and physiology, the broader field of psychosomatics needs to be examined. The authors suggest further, that the questions to be asked in this context, should seek to identify the conditions which influence physiological responses when the antecedent stimuli are psychological in nature. This view lends direct support to that expressed by this researcher in relation to the need for psychological factors to be taken into account in aetiological theories of psychophysiological disorders such as migraine, and in the formulation and administration of their treatment (see sections 2.11, 2.15, 2.16, and 2.31).

### 3.24 Hypnotisability

Hypnotisability is defined as the ability to become hypnotised, to have the experiences characteristic of the hypnotised person, and to exhibit the kind of behaviour associated with hypnosis (Hilgard, 1965). Hilgard and Morgan (1975) found the level of hypnotisability to be directly related to clinical outcome. Although a person's attainable level, or hypnotisability score, may vary from day to day in a manner of moods, the individual's hypnotisability, in general, is taken to be relatively enduring (Bowers, 1976; Hilgard, 1965; Morgan, Johnson & Hilgard 1974). Spiegel and Spiegel (1978) reviewed and confirmed their thesis on the relative constancy of hypnotisability over time, with the additional claim that hypnotic capacity is basically physiologically determined and can be accessed by observing certain responses in subjects, such as the eye roll test described by Spiegel (1972).

There appears to be no marked 'hypnotic personality' type. Hilgard and Hilgard (1975) found no relationship between hypnotic susceptibility and personality on standard personality inventories, hysteria, and social gullibility. They did find, however, that

imaginative capacity, that is, the ability to become focused and absorbed in an activity, correlated positively with hypnotic susceptibility. This correlation is generally supported in the literature (Sheehan, 1979a).

Spiegel (1974) studied highly hypnotisable subjects and found they exhibited a preparedness to trust, to suspend crucial critical judgment, to exhibit readiness for new experiences, to accept logical incongruities, to have a telescoped time sense, an overall tractability, and paradoxically, a rigid core of private beliefs. The extent to which these traits may be found in low hypnotisable subjects was not determined in the study, and therefore no statistical comparison was possible.

The allocation of attention by hypnotised subjects is seen as a critical determinant of the degree to which subjects succeed in responding to the suggestions of the hypnotist. McConkey (1994) claims that, if highly hypnotisable subjects employ appropriate cognitions and attentional resources consistent with their level of motivation, they will experience the suggested effect in a compelling way.

Sarbin and Slagle (1993) support this view by negative example, commenting, that poor hypnotic subjects tend to continuously evaluate the suggestions given (reality-testing) and indulge in irrelevant action impulses. The necessary imaginative focus suggested by the hypnotist is thus avoided, and the requisite conditions for hypnosis of muscular relaxation and inhibition of sensory experience never occur.

The phrasing style adopted for suggestions in the course of induction may exert an influence in some circumstances, and is therefore considered of some importance in testing hypnotisability. For example, De L. Horne and Powlett (1993) reported on a study in which individuals who had been previously tested on a scale using direct

suggestions for the induction process, were re-tested with the use of indirect suggestions. The results indicated that whereas the direct method of phrasing and presenting suggestions had resulted in these individuals achieving scores indicative of only low hypnotisability, the indirect 'confusional' method used in the re-test study produced scores indicative of high hypnotisability.

Spiegel and Spiegel (1993), in a different sense, subscribe to the need for careful phrasing in testing hypnotisability, by pointing out, that the term 'hypnotic susceptibility' may add fuel to the popular misconception that hypnosis calls for being susceptible to falling under the spell of the hypnotist. They suggest, therefore, that the term 'hypnotic capacity' which avoids this connotation, is a more suitable term to be used.

The question of re-test practice effect was, however, not addressed in any detail, presumably because of the different techniques used. Nevertheless, it is still legitimate to ask about the possible effects of lowered apprehension occasioned by the insights of the previous hypnotic testing experience, and the resultant reassurance and relaxation facilitated by it.

Hilgard (1971) reported that there was no significant difference in susceptibility between the sexes, and that hypnotic susceptibility scores tend to reach their peak in the pre-adolescent years and then slowly decline with age. While being in agreement with the latter of these two findings, Milne (1995) qualifies this view by pointing to exceptions, namely that there are many senior citizens who have remained highly hypnotisable.

Considering the complex nature of hypnosis, the measurement of hypnotisability involves relatively simple procedures. Most hypnotic susceptibility rating scales seek to

isolate and evaluate a range of hypnotic responses to suggested stimuli. This multifactorial approach can be seen to yield a larger number of measures, and with that, more reliable assessment scores (De L. Horne & Powlett, 1993). It also accommodates subjects who tend to be more responsive in some areas of hypnosis than others. For example, while some subjects respond readily to visual stimuli, others respond more readily to sound pictures. Similar differences prevail between motor and cognitive responses.

Common hypnotic susceptibility scales seek to measure motor responses such as arm levitation or lowering, limb immobility, head falling forward, hands moving together, eye catalepsy, and so forth. Cognitive responses include amnesia, age regression, and hallucinated voices.

The Hypnotic Induction Profile (HIP) (Spiegel, 1973), is a short clinical assessment procedure starting with the well known 'Spiegel eye-roll'. Spiegel (1973) writes that the method of eye-roll induction has its origin in old eye fixation techniques, but that the patient's ability to maintain his eyes in a rolled up position while closing his eyelids marks a neurophysiological baseline from which his hypnotisability can be measured on a scale from 0 to 4. This score is then compared with the patient's performance on more traditional somatosensory criteria of the test. A high eye-roll during lid closure is a presumptive prediction for high hypnotic capacity, whereas a low eye-roll suggests little capacity to experience hypnosis.

Somatosensory responsiveness such as tingling sensations and numbness in the fingers, and hand levitation combined with inability to leave the hand in a down position when pulled down by the hypnotist, is measured by the number of verbal reinforcements

necessary to secure the patient's compliance, and by the patient's subjective differential control of the hand. A score combining the eye-roll and somatosensory performances is then determined on a scale from 0 to 5. The small number of individuals with zero score are deemed not to have the capacity for hypnotic trance, and according to Spiegel (1973), repeated or even more elaborate testing will not change this result.

Some individuals may do well in the eye-roll test but achieve only a zero rating in the somatosensory test. Evidence suggests, that these individuals have some measure of concentration impairment, or psychopathology such as disorder of thought, or some emotional affect, or some neurological impairment (Spiegel, Fleiss, Bridger, & Aronson, 1975).

Light to medium trance levels have generally been sufficient for non-surgical clinical treatment, such as pain management and psychosomatic disorders (Clarke & Jackson, 1983; McConkey, 1990). It is estimated that about 80% of the population are able to attain these levels, 9% are thought to be at the low end of hypnotisability, and 11% are thought capable of deep trance, such as may required for surgical intervention (Hilgard, 1968; Milne, 1995).

The foregoing review helps to illustrate the importance of hypnotisability in the theory and application of hypnosis. Although hypnotisability is, among other determinants, a very useful indicator of likely treatment response, it does not require measurement in all treatment studies. From a clinical perspective, acceptance of patients for hypnotic treatment does not normally depend on their hypnotisability level (patients requiring anaesthesia are the exception). In this study, which has a strong clinical orientation, the absence of pre-established levels of hypnotisability among participants is

therefore more representative of prevailing clinical conditions and practices, and thereby adds to the generalisability of the results obtained.

### 3.25 Self-Hypnosis

James Braid (1794-1860) has not only been described as 'the father of modern hypnosis' (Milne, 1995), but is credited with having been the first person to describe self-hypnosis. Braid is said to have experimented with hypnosis by letting his servant, and later several other people, focus on a small object. He found that this would usually result in a hypnotic state, which he later came to recognize and describe as self-induced hypnosis (Langen, 1993).

Self-hypnosis, also known as auto-hypnosis, may be simply and unambiguously defined as the process of inducing and maintaining one's own state of hypnosis. Jackson and Merrington (1993) comment that its use may improve the quality of life in numerous and important ways, especially when used in stress management and pain control. It is particularly valuable as an adjunct to clinical treatment and encourages patients' involvement in their treatment, facilitating empowerment and self reliance.

The authors also state that self-hypnosis may best be taught in the course of clinical hetero-hypnosis, yielding the additional benefit of removing resistance that might otherwise be present. Spiegel (1973) suggested that the use of a hypnotisability test is a useful adjunct in teaching self-hypnosis because it provides the hypnotist with diagnostic information on the basis of which self-hypnotic treatment may, or may not, be the preferred treatment.

Langen (1993) draws a distinction between passive and active teaching of self-hypnosis or 'auto-hypnotic procedures'. In the passive procedure, hypnosis is initially

induced by the patient's passive cooperation with the hypnotist. The belief here is that if the patient undergoes this procedure regularly, relevant hypnotic aspects of these experiences can be readily self-induced by the patient, simply by recalling and repeating the successive suggestions given by the hypnotist in the course of the hypnotic teaching procedure. The hypnotist may also incorporate supportive post-hypnotic suggestions to encourage the patient's success in self-induction. In the active procedure, patients are initially required to learn some structured introductory exercises under supervision, such as relaxation, before they advance to the induction procedure itself.

Research conducted by Hurt (1978), and expanded by Oberlander (1978), was designed to determine differences and similarities between hetero-induced hypnotic states, (induced by another person), and self-induced hypnotic states over a four-week period. Results indicated the presence of several experiential differences.

Selected participants, all of whom had a high hypnotisability rating, described their attentional focus during self-hypnosis as more expansive than in the hetero-hypnotic sessions. Their time during self-hypnosis was largely spent being actively engaged in becoming more broadly aware of their mental continuum, whereas their attentional focus in hetero-hypnosis was more directed to specific objects and events, resulting in fewer intrusions. Characteristics of trance entry and focus were individually manipulated by subjects, and it was found that success in self-hypnosis (as in hetero-hypnosis) is influenced by the ability to desist or dissociate from reality oriented information processing.

Concurrent with the shift toward expansive attentional focus in self-hypnosis, was also a shift toward greater ego receptivity allowing more unconscious and preconscious

material to surface to conscious level. Associated with this, was a greater amount of imagery, especially visual imagery. However, results also showed that as subjects became more experienced and settled in their self-hypnosis routine over time, these differences between hetero-hypnosis and self-hypnosis decreased.

Although physiological changes such as EEG alpha wave activity, and a lowering of blood pressure and oxygen consumption (as discussed in section 3.23), were not compared in the above research, these phenomena of the hypnotic state proper are present irrespective of the method used to induce that state (London, Hart, & Leibovitz, 1969; Travis, Kondo, & Knott, 1973).

Oberlander (1978) indicated that subjects continued to use induction schemes learned in hetero-hypnosis. These involved focusing attention on the induction stimulus. The repeated reinforcement of the behavioural interaction between focus stimulus and hypnotic response led to rapid autogenic induction for successful subjects, so that simply looking at, or visualising the stimulus elicited a trance response. This reinforcement aspect is supported by Kroger (1993), who, referring to a variation of the Spiegel (1973) eye roll technique writes:

After sufficient practice, autohypnosis is obtained by a triggering cue, as for instance, closing the eyes and letting the eye balls roll upward for a few seconds.

When they roll down, lid closure will follow, and the autohypnotic state will remain until terminated by dehypnotization (p. 512).

The foregoing suggests that although there are some minor differences between hetero-hypnosis and the early learning phase of self-hypnosis, differences of long term duration have not been identified. However, clinical experience suggests that the need

for self direction in self-hypnosis requires greater involvement of the left hemisphere, and that this occasions the level of hypnosis attainable and sustainable, to be of lesser depth than that in hetero-hypnosis. But even this difference is now readily overcome with the use of pre-recorded audiotapes containing the procedure and suggestions normally given by the hypnotist, thus freeing the user of directive left hemisphere involvement (Jackson & Merrington, 1993).

### 3.3 Hypnotic Suggestions and their Functions

#### 3.3.1 Suggestions and Methods of Phrasing

Suggestions perform the most important functions in hypnosis. They are the primary means by which modern day clinical hypnosis is induced, deepened, and terminated, and they are indispensable for purposes of therapeutic treatment, and for affecting post-hypnotic compliance. The form of phrasing or presentation of suggestions is important, as it is likely to influence the patient's response, such as when hypnotisability is being tested (Hamilton, 1984).

Allowing for differences in personal style, the phrasing adopted for the past 30 to 40 years may be said to fall into 2 broad categories, namely direct and indirect phrasing of suggestions. The direct suggestion makes a clear, unambiguous and encouraging statement, and may be expressed in the form of, "You will find it progressively easier and easier to relax into a state of great comfort." This statement may be reinforced with a further direct suggestion such as, "You will notice how easy and enjoyable it is now, to relax more and more deeply."

The confident, "You will," appears to leave little doubt that what is being suggested is actually the case. However, considering that patients remain conscious and

in control during hypnosis, the commanding and strongly authoritative tone formerly adopted vis a vis the perceived passive patient of the last Century, has largely given way to an encouraging and reassuring tone (Stanley, 1993).

The indirect suggestion adopts a more covert and deliberately ambiguous approach. It is designed to manipulate any resistance by giving the impression that the patients are always in full control, that they choose whether or not they will comply with the suggestions given, and that whatever the choice, it will occur at their leisure. An indirect suggestion to relax may be presented in the following form.

Soon, when you feel ready, you may want to start thinking about going into a deep comfortable state of relaxation. Sooner or later you may just want to enjoy the thought of relaxing sooner than later, and this you may do very suddenly or rapidly, feeling all the while more and more at ease and more and more relaxed (adaptation from Stanley, 1993).

The indirect form of suggestions made popular by Milton Erickson is thought to allow the patient more freedom for imaginative interpretation, of equal importance however, is the reassurance which the patient derives from suggestions which reinforce his or her feeling of choice in participation, and with that, a perceived sense of control (Emmerson, 1987). Research has shown the indirect form of phrasing to be particularly effective in inducing hypnosis in subjects of both low and high hypnotisability (Hamilton, 1984).

However, the confident and unambiguous approach adopted in the direct form of suggestions is generally considered the more appropriate form to be used in the treatment of pain control, although this need not apply to the induction procedure itself

(Burrows & Dennerstein, 1993; Daniels, 1976; De Benedittis et al. 1989). In the circumstances of pain control, a prescriptive tone and phrasing enhances the patient's subjective belief in the competency of the hypnotist and the efficacy of the treatment, thereby also enhancing its real effectiveness.

Chertok (1993) supports this view. He writes that the common method for producing hypnotic anaesthesia for surgery is authoritative. For practical and clinical purposes it may be expedient to use a combination of methods within one session where, for example, a form of indirect phrasing is used for induction and deepening purposes, and a direct approach is adopted for treatment, post-hypnotic compliance, and alerting (Hartland, 1971). In view of these considerations, a mixture of direct and indirect suggestions may also be advantageously used in this study.

One style of indirect phrasing is known as the confusional technique which is based on the idea that when communication ceases to make sense to a patient, he or she dissociates from what is being said, resulting thereby in self-induced relaxation. It follows, that the confusional technique is not likely to be suitable when the patient's full attention is required to focus on a specified stimulus, such as may be required in the course of treatment. This method is therefore better used to induce hypnosis, after which, any suitable treatment can be applied. The confusional technique has been found effective for inducing hypnosis in subjects in the low range of hypnotisability (Hamilton, 1984). A relatively short example of a confusional suggestion may be phrased in this form:

You may like to be free of tension, and relaxation, or relaxation with no tension, perhaps you also like tension only sometimes and relaxation always when not possible or

whenever possible. Who knows, there are many things to feel relaxed about or to remember, but a lot more to relax about and not to remember, and the more you relax the less you might remember to relax but the more you might not remember and not care about to remember, it is so good to relax.... (adaptation from Stanley, 1993).

It is generally accepted that suggestions for treatment, post-hypnotic compliance, and alerting, should be short, clear, and repeated several times, with key words being repeated in synonyms to aid clarity of meaning and to avoid misunderstanding. The delivery should be unhurried, with appropriate pauses between suggestions to allow the patient sufficient focal attention on the message given (Comer, 1995; Emmerson, 1987; Stanley, 1993).

To enhance their effectiveness, suggestions may conveniently incorporate and build on the changed physiological manifestations of patients which occur in response to the lowered state of arousal in their central nervous system. These include slower breathing, muscle lethargy, increased circulation, and diplopia. The last phenomenon occurs through sustained focus on a small object, so that when a patient is complying with the suggestion to focus on a small pencil dot, he or she will after a short while, see two dots. A spontaneous refocussing will readjust the vision only to lapse again into seeing two dots. This process continues to repeat itself, causing considerable fatigue of the eyes and eyelids. A suggestion utilising the diplopia phenomenon for deepening purposes can, for example, be phrased in a direct manner and expressed thus:

You will notice your eyes are becoming tired, and your eyelids too are becoming tired, and heavy, so very heavy, they will go on becoming heavier and heavier until you

will find relief, complete relief by allowing your eyelids to close gently (adaptation from Spiegel & Spiegel, 1993).

Alternatively, progressive muscular lethargy may be incorporated and enhanced in this manner: As your arms and legs become heavier and heavier you will notice yourself becoming more and more relaxed, and the heavier your whole body feels, the deeper and deeper your sense of relaxation and comfort will be (adaptation from Stanley, 1993).

### 3.32 Induction and Termination

An induction technique may be broadly defined as anything which invites the subject to pay attention (Spiegel & Spiegel, 1993). The primary and obvious objective of hypnotic induction is to facilitate a trance state. In so far as the hypnotic trance is a state of altered consciousness, dissociated from external sensory input, the procedure of induction employs strategies designed to reduce awareness of external stimuli (Erickson & Rossi, 1980; Erickson, Rossi, & Rossi, 1976), and associated left hemisphere cognitive processing (Rose, 1989; Shone, 1982; Stanley, 1993).

It follows, that the hypnotist engages the subject's attention on internal, subjective sensations. This process is frequently started with some eye stimulus, such as the Spiegel eye roll, or alternatively, with a narrowed eye focus, such as focusing on a small dot. This narrowed focus leads to subjective changes in the visual field (diplopia), and other internal changes such as slow, easy breathing, followed by relaxation. It may also be accompanied by a sense of heaviness or lightness in body and limbs. This, in turn, may be accompanied by tingling sensations, and sometimes by perceived distortions of the extremities, such as enlargement of hands or feet (Spiegel, 1973; Spiegel & Spiegel, 1993). The eye roll technique is an effective induction method. It has been effectively

used in hetero-hypnosis, self-hypnosis, and in group inductions (Spiegel, 1973; Stanley, 1993), and as such, it is suitable procedure for use in this study.

Although the involvement of the subject's eyes in the induction procedure is not essential to induce trance, eye closure helps the subject to focus inwardly on the suggested stimuli. It triggers the release of melatonin which is known to facilitate relaxation and memory retrieval, as discussed in section 3.23. Another factor of importance in inducing hypnosis, is the interpersonal relationship between the hypnotist and subject. Erickson and Rossi (1980) maintain that the level of trance elicited is largely the responsibility of the hypnotist and that it is a direct reflection of the relationship between hypnotherapist and patient.

It is also their well known view that deeper levels of trance may best be achieved by artful distraction of the subject during the induction procedure. Connected to this view is their belief that a conscious resistance or interference by the subject can be manipulated and used while the hypnotist communicates with the unconscious. And further, that this is best achieved with an indirect induction method, such as the confusional technique or suggestions of multiple stimuli which will keep the subject absorbed where a single stimulus suggestion might not.

Crasilneck and Hall (1975) believe that the motivation of patients, impacts on their pursuit of goals in spite of conscious resistance. More specifically, the authors state:

For motivation to work maximally, the patient must believe in the suggestion being given, must accept the integrity and well meaning of the hypnotherapist, and must (most importantly!) feel that the suggestion being given by the therapist echoes in a profound way, the patient's deeper inner voice (p. 109).

Spiegel and Spiegel (1993) advance the view that induction techniques may be conveniently categorized into three broad rubrics, (a) coercion, (b) seduction, and (c) guidance. The authors recommend the method of guidance as the most appropriate for therapeutic uses, suggesting that the therapist, like the Socratic teacher, helps the patient to discover what he already knows about himself. The guidance model, in turn, has three fundamental components, (a) aura, (b) psychophysiological enhancement, and (c) the 'plunge.' Each component is discussed below.

The aura consists of socially implanted beliefs, expectations and anxieties about hypnosis and the hypnotherapist which patients bring to the hypnotic treatment session. Depending on the anxieties evoked by these beliefs and expectations, the aura can hinder or enhance the induction process. Gruzelier (1988) and McConkey (1990) support this view, maintaining that patients who believe a given hypnotic treatment is likely to resolve their problem, are more likely to be fully attentive and use their full trance capacity.

A highly anxious patient is likely to offer resistance by being preoccupied with reality testing, and other left hemisphere activities, thus finding it more difficult to enter a trance state. Anxiety based resistance may well be something that the patient wishes to overcome, but is unable to control. This supports the view expressed by Erickson and Rossi (1980) that ultimately, the responsibility for by-passing or using the patient's resistance rests with the hypnotherapist.

Spiegel and Spiegel (1993) point out that the aura is not an unchanging factor. Clinical experience has shown that anxious patients can become relaxed in response to insights gained through their first hand experience of hypnosis, and conversely, that a motivated and trusting patient may become anxious in response to comments

unsuspectingly and inadvertently made by the hypnotherapist. The authors point out further, that highly hypnotisable patients have been found to be particularly sensitive to negative aspects of aura.

The second component of the 'guidance' induction model is physiological enhancement. It is a deepening technique, and refers to suggestions incorporating physiological phenomena experienced by the subject in the induction process. The plunge is the third component of this induction triad. It is the actual transition from normal, albeit relaxed consciousness, to trance state, and represents the subject's optimal shift toward focal attention with a concomitant constriction of peripheral awareness (Spiegel & Spiegel, 1993).

Concurrent with the practice of reducing evaluation and reality testing left hemisphere activities, is the practice of inducing motor changes in the subject. These may include arm rotation, arm levitation, and catalepsy in one hand. The attentional focus, the sensory, and motor changes which the subject experiences during these procedures deepen the trance state (Rose, 1989; Shone, 1982).

Other deepening techniques may also be employed. These include associative suggestions, linking progressive deepening with somatosensory awareness of warmth or buoyancy, or with guided imagery such as descending to deeper levels of trance by means of escalators, lifts, or staircases (Rose, 1989). Counting techniques are also used, where each progressive number is associated with increasing detachment from external stimuli, leading to deeper levels of trance. Guided imagery is an effective technique not only for deepening the trance state, but for inducing relaxation, and for facilitating the

treatment itself. Guided imagery is therefore a suitable procedure for the purposes of this study.

Whereas the procedures of induction and deepening of the hypnotic state aim at progressively reducing external sensory inflow and evaluation activities, the progression to deeper levels of hypnosis is not a continuous process; nor does it necessarily plateau at a lower level. Instead, it follows a cyclical trend which is in keeping with natural cyclic waves alpha, beta, and theta, and indicative of changing impulses in the brain. This means that the depth of hypnosis within any one session vacillates between various levels within the hypnotisability range of the subject (Carlson, 1986; Sabourin et al. 1990).

Kroger (1993) comments that physiological phenomena occurring during the induction process may be advantageously used to enhance the effectiveness of the treatment to follow. For example, eyelid heaviness, arm catalepsy, or relaxation of the entire body as experienced by patients, may be cited to them as examples of control over physiological responses. It may be suggested to patients that just as they can control these physiological responses, so they can control their problem behaviour. And thus, words, thoughts, and feelings can act as conditioned stimuli to elicit specific autonomic responses, even though the original stimuli may, in time, be forgotten.

Therapeutic treatment is the primary aim of clinical hypnosis, and although the effectiveness of treatment is understood to be enhanced when administered at deeper levels of hypnosis (Gruzelier, 1988; Hilgard & Hilgard 1975), it is generally agreed that even a light to medium trance level is sufficient for most clinical treatments (Clarke & Jackson, 1983; McConkey, 1990).

Clinical experience has shown that the principles of hypnotic induction procedures and suggestions, including post-hypnotic suggestions (as discussed sections 3.31 to 3.33), apply to group sessions as they do to single patients (Braun, 1993; Shone, 1982). However, the qualification is made that individuals in group settings may require more time for the initial 'settling down' period, but this is frequently offset by a greater preparedness to relax, presumably because of a subjective perception of 'safety in numbers' (Stanley, 1993).

The cancelling of all suggestions not wanted to have any effect in post-hypnotic periods is essential. Termination of the hypnotic state is readily achieved with alerting suggestions specifying that the patient will be in a state of full alertness at the end of a specified procedure. A common procedure for this involves counting backwards from five to one with the suggestion that the patient will become progressively more alert with each count, open his or her eyes in response to the count of 'two,' and feel fully alert and refreshed on the count of 'one' (Milne, 1995; Shone, 1982).

Clinical experience has also shown that some patients may, at times, require a longer counting procedure to become fully alert. This is usually associated with reluctance on the part of the patient to terminate the comfort of trance. When this occurs, repeating the counting procedure just once usually elicits the desired alerting effect. On rare occasions, and usually due to extreme and prolonged physical exhaustion, a patient may actually drift into light sleep, but will respond to a touch on the shoulder together, or an alerting suggestion. Alertness counting is usually administered at a faster rate and in a livelier tone than that used for induction (Milne, 1995; Stanley, 1993).

### 3.33 Post-hypnotic Responsiveness

A post-hypnotic suggestion refers to a suggestion which is given to the hypnotised subject for the purpose of eliciting a specified behavioural response under specified conditions when the subject is no longer hypnotised. Spiegel and Spiegel (1978) contend that post-hypnotic compliance consists of a predictable triad of reactions namely, compulsive compliance with the suggestion given, amnesia for having received the suggestion, and rationalization for the compulsive behavioural response. Their statement is made appropriately with the qualification that there is no evidence in the literature to suggest that behaviour can be elicited through hypnosis which the patient would not normally agree to perform outside hypnosis.

A behaviourist perspective of post-hypnotic compliance is offered by Kraines (1969), who suggests that the subject behaves on a stimulus-response basis, and that hypnotic suggestions can therefore be used to set up conditioned reflexes. Post-hypnotic compliance may therefore be simply expressed in behavioural terms as being a conditioned response to a pre-determined stimulus. Kraines' (1969) views are based on the Pavlovian theory of classical conditioning (Pavlov, 1951), the precursor to behaviourist theory. Pavlov's experiments have led to the now generally accepted theory that ideosensory activities are functions of the instinctual or primary signalling system. These functions can be mobilized rapidly since they are necessary to preserve the integrity of the organism, that is, they are functions of the self preservation drive (Comer, 1995; Kroger, 1977).

Kroger (1977) maintains that patients can be trained to elicit conditioned reflexes for clinical purposes. For example, responses such as temperature increases and

decreases in hands are easily elicited by appropriate imagery and can be used as preparation for more complex suggestions such as aversion conditioning. Aversive conditioning can be developed by pairing aversive stimuli with unwanted behaviour. Kroger continues that such symbolic activities are part of the secondary signalling system of higher nervous elaboration, and work by analogy.

The hypnotic method of conditioning is hardly new. It is similar to Eastern philosophical healing methods such as Yoga, and Zen. It differs from Pavlovian classical conditioning only in terms of its stimulus being internal as opposed to the external (whistle) stimulus of Pavlov's famous experiments. It was Pavlov's view that suggestion is the simplest form of a conditioned reflex (Pavlov, 1951). This view is supported by Austin (1994):

Every thought creates a physical reaction. The subconscious mind, too, is affected by thoughts with strong emotional content; once these have been accepted they become a program and trigger the same response over and over again (p. 36).

Like all post-hypnotic responses, post-hypnotic amnesia can be dramatic, considering the relative ease with which it can be induced, especially in highly hypnotisable subjects. The hypnotist merely suggests that on alerting from hypnosis, the subject will be unable to remember part or the entire hypnotic experience until a specified cue (stimulus) is given to the subject, or until the post-hypnotic suggestion becomes extinct of its own accord (Stanley, 1993).

Evans (1979) conducted a study into post-hypnotic amnesia using subjects who did not previously know the answer to the question what colour change a blue or purple gemstone undergoes when exposed to great heat. While hypnotised, the subjects were

given the answer yellow. Amnesia about how they learned the answer was then induced and the subjects alerted. A simple information test which included the 'gem' item was subsequently completed by the subjects, and the correct answer of 'yellow' was given by all. However, when they were questioned about the source of this knowledge, they invariably named some other event, the real source having apparently been forgotten.

Evans (1979) also points out that memories may remain functionally intact except for the context in which the event occurred, or for the circumstances under which a particular piece of information was learned. In the latter circumstance, subjects may be embarrassed at their inability to remember and be inclined to confabulate an answer. Post-hypnotic amnesia is not a functional deficit in the objective sense, for it is not an ablation of information and experience. It can, after all, be lifted as easily as it can be induced (Hilgard, 1965), or become extinct of its own accord in due course.

Historically, post-hypnotic amnesia has been likened to memory disturbances occasioned by hysteria and other clinical syndromes like fugue, multiple personality disorder, and Korsakoff syndrome. The denominators which are common to these three syndromes and post-hypnotic amnesia, are (a) compelling memory loss, affecting in particular, personal experiences and other autobiographical data, and (b) absence of any identifiable or recognizable disturbance of the central nervous system. Further, the essential intactness of critical memories during these amnesia periods is evidenced (as in the case of post-hypnotic amnesia), by their subsequent recovery (Evans, 1993). This recovery also attests to the fact that all of these amnesia episodes are not due to faulty memory processing or faulty memory storage, but rather to an inhibition of the retrieval process.

These common characteristics notwithstanding, Kihlstrom and Evans (1977) found that when post-hypnotic amnesia subjects were subsequently freed from their amnesia, they were able to recall a significantly greater number of test items than non-hypnotised control subjects. Evans (1993) comments that the inclusion of 'reversibility' as a criterion of amnesia not only provides a means for differentiating between the effects of amnesia suggestions and normal forgetting, "but also suggests that the subject may not really be amnesic unless he demonstrates reversibility, regardless of his initial level of recall during the amnesia test" (p. 86).

It appears that reversibility is not a reliable criterion for amnesia for a number of reasons. It can be as much of an artefact as feigned amnesia. It may be no more than a return to normal behaviour after completion of amnesic role play. Considered from an operational perspective, if a subject recalls one or several items of the test, the subject could be said to have reversed the amnesia and thus qualify for the title of amnesic subject with restored memory. It is equally plausible that the person is a former role playing subject now behaving normally, and thus would also qualify for the diagnosis of amnesia.

Alternatively, if the subject cannot recall any item of the test, that subject may be either a non-amnesic subject with a weak memory, a genuinely amnesic subject with a restored but normally weak memory, or a genuinely amnesic subject with uncancelled amnesia. This range of possible explanations demonstrates that reversibility is not a singular either/or condition. Reversibility can for these reasons not function as an amnesia determinant or as a differentiating criterion for subjects who are amnesic and those who are not, or just pretend to be.

Post-hypnotic suggestions play very important and beneficial roles in clinical treatment. For example, they can be used to shorten the induction and deepening processes by simply suggesting that when a certain cue stimulus is presented during future induction procedures, either in the course of self-hypnosis or hetero-hypnosis. Relaxation will follow spontaneously and will proceed readily to deeper levels. The cue may be selected by the patient and consist of a word or action. To prevent any unwanted elicitation of responses, it is usually suggested that the cue stimulus will only be operative within specified circumstances such as agreed to induction procedures or specified aspects of pain control. Post-hypnotic suggestions are also known for their efficacy in replacing unwanted sensations and behaviour such as anxiety and substance abuse with calming sensations and healthy behaviour patterns (Comer, 1995).

Commenting on the effectiveness of post-hypnotic suggestions in the control of post-operative pain and in the healing process, Siegel (1991) suggests that they are just one of several effective hypnotic methods used in the treatment of pain. The fact that they can be used in combination with other hypnotic methods such as 'vascular manipulation' discussed in sections 3.43 and 3.5, adds to their overall clinical usefulness, and their suitability for use in this study. For example, a patient may be trained to use a particular pain control technique and have its potency and effective duration progressively increased with a post-hypnotic suggestion that this will be, and continue to be the case every time the technique is used in response to a specified pain related cue (Stanley, 1993). Kline (1976) found conditioned stimulus-response behaviour established in hypnosis to be more durable and less likely to become extinct than that established in a normal state.

### 3.4 The Nature of Pain and its Response to Hypnosis

#### 3.41 Psychophysiological Components of Pain

Psychophysiological interaction can be initiated by either psychological or physiological factors. Physiological processes, whether biochemical, electrical, or mechanical, can effect changes in psychological states (Karle, 1992). The effects on moods and feelings of anxiolytic drugs or alcohol, for example, are well known, and demonstrate this point.

The influence of psychological factors on physiological states may be convincingly demonstrated with a scientifically monitored case study (Foenander & Burrows, 1993).

Details of this case study reveal that a patient suffering from convulsive seizures submitted to an EEG at the time of first onset of the seizures when he was 18 years of age. The EEG showed evidence of various abnormalities. Similar abnormalities were also recorded with a further EEG taken when the patient was 24 years of age.

Subsequent to this procedure, the patient was hypnotically regressed to his 12th birthday, (a period prior to onset of seizures) and the EEG recorded in this hypnotically regressed state showed no abnormalities. However, when the patient was progressed to 18 years of age, a repeated EEG procedure registered abnormalities corresponding to those recorded originally at the time of seizures. The EEG was then brought within limits by 'reassuring' the patient.

The authors point out that the age regression itself may not have been responsible for the EEG changes, that they may have occurred in response to the relaxed state of the patient, or in response to the recall of psychologically laden memories. While this may have been the case, it needs to be acknowledged that both the regression procedure and

the psychological memories experienced in the course of it, are variants of psychological experience, and as such, it may be said that psychological factors acted as stimuli and triggered real and measurable physiological effects.

The importance of psychological factors, such as attitude, faith, and belief in the potency of the treatment undertaken (including that of hypnosis), is well demonstrated by placebo effects. These have shown that in cases of covertly fictitious treatments mere belief in their potency is sufficient to contribute to the mobilization of immune resources, which in turn speed up natural healing processes (Karle, 1992; McConkey, 1990).

Relatively recent neurological discoveries have revolutionised ideas about the interaction between psychological thought and physiological blood flow. The former belief was that thinking did not use up any significant amount of energy, and that, therefore, blood flow was not affected by it. However, in the course of the past two decades the use of radioactive tracers has assisted in the discovery, that activation of a group of brain cells through thinking or talking produces a changed pattern in blood flow. This is evidenced by a marked decrease in frontal lobe areas and a marked increase in part of the temporal lobe (Graham, 1990; Rose & Gawel, 1981).

The system which controls blood flow to the brain is highly complex, as discussed in section 1.31, and is affected by a range of factors including mental processes, circulating chemicals (amines), nervous factors and pain (Julien, 1995; Lance 1993). Motivation and concentration have also been shown to have a direct effect on neurovascular blood flow "...confirming yet another psychosomatic link" (Rose & Gawel, 1981, p. 37).

Pain increases the metabolism and dilates blood vessels generally. The paradox, as pointed out by Rose and Gawel (1981), is that pain, like stress, produces increased activity in the sympathetic nervous system, and while this leads to vasoconstriction, it is also accompanied by increases in blood flow to the brain. It may be, that this paradoxical increase in blood flow, under the increased pressure occasioned by vasoconstriction, subsequently causes vascular dilatation and migraine pain. Contrary to this view, Rose and Gawel suggest that the answer may be found in that the sympathetic nervous system controls only the larger 'resistance' vessels, whereas pain, in this context, may only activate local capillaries.

The National Health and Medical Research Council Report (1982) indicates that there is some evidence of an endogenous pain inhibitory mechanisms in the central nervous system which utilizes opiate-like transmitters called pentapeptides. These pentapeptides can be partially inhibited or reversed by the anti-narcotic substance known as naloxone.

Importantly, and of special interest here, is the clinical finding that analgesia in hypnotised patients cannot be inhibited or reversed by naloxone. This irreversibility is in contradistinction to endogenous analgesia, or that produced by acupuncture or by transcutaneous electrical nerve stimulation. This strongly suggests that the pain inhibitory mechanism in hypnotic states differs from the inhibitory mechanisms in natural opiate, electrical, and acupuncture analgesia (Rose, 1990). It also supports the concept that hypnotic analgesia is a state of 'altered pain perception' rather than an absence of pain.

Rose further comments, that despite all that is known about pain, we are left with the conclusion that pain is a great puzzle and that the most accepted theory of pain today, is the gate theory. This theory suggests that a control mechanism exists in the spinal cord's inner structure which, when open, allows pain signals through to the brain.

The gate control theory of pain, advanced by Melzack and Wall (1965), can accommodate psychological processes affecting this spinal mechanism (see section 2.25). For example, activities such as distraction, cognitive explanation and belief, placebo, encouragement, and hypnosis, are regulated by the control mechanism as afferent processes. This, in turn, helps to explain why the perception of psychophysiological pain is readily altered by psychological treatment, such as hypnosis (Finer, 1993).

A distinction can be drawn between acute and chronic pain. Acute pain is sudden and usually brief, and as it is commonly caused by an accident or irritation, it is usually accompanied by local tissue damage. Psychologically, it is often associated with anxiety. Acute pain is more easily resolved with drug treatment. Pain qualifies for the description 'chronic', when it persists for six months or longer without any clear cause. It is associated with depression, is frequently treatment resistant, and often gets worse over time. Complaints such as migraine, and tension headaches fall into this category (Jackson, 1989).

### 3.42 Pain Perception

The psychological role in pain perception is readily acknowledged within the medical profession. The anaesthetist Thomas Torda (1995) describes the pain mechanism thus:

The conduction of impulses through the spinal cord and brain stem to the thalamus is the simple sensory aspect of pain. The processing of this physiological signal into the percept of pain and the individual's pain behaviour are under the influence of psychological factors. For optimal pain management, all these mechanisms need to be taken into account (p. 65).

The psychological factors which include awareness of self and immediate events, are open to manipulation by hypnotic suggestions. For example, a suggestion to the patient to focus on a specific pain reducing event, is likely to affect the processing of the physiological signal of pain, and with that, the awareness of pain.

De Benedittis, Panerai, and Villamira (1989) investigated the effects of hypnotically induced analgesia and hypnotisability on experimental pain, taking into account pain and distress tolerance as well as neurochemical correlates. They distinguished two distinctive dimensions of pain, namely 'sensory-discriminative,' which is informative of the location and intensity of pain, and 'motivational-affective,' which reflects the negative impact and emotional distress of the pain.

Twelve female and nine male university students participated in the study, comprising eleven high hypnotisable subjects and ten low hypnotisable subjects, as determined on the Stanford Hypnotic Susceptibility Scale. All subjects were administered ischaemic pain tests in both normal and hypnotised states by the application of a tourniquet to the subject's non-dominant arm. Three variables were tested, (a) pain and distress tolerance were measured on a ten-point scale, (b) anxiety levels were measured by the Italian version of the IPAT Anxiety Scale Questionnaire, and (c) plasma

concentration of beta endorphin and adrenocorticotrophic hormone (ACTH) was evaluated by radioimmunoassay methods.

Results showed positive correlations between relief of pain and distress, and hypnotisability. There were no significant changes in plasma beta-endorphin levels during experimental ischaemic pain between hypnotic and non-hypnotic conditions independent of hypnotisability, which could indicate that hypnotic analgesia is not mediated by opiate systems. No relationship was found between hypnotic analgesia and anxiety reduction. Subjects reported that they could readily make the distinction between pain and distress. They generally found stress to be less or equal to, the sensory pain. However, as the pain lasted longer and continued to rise, distress began to rise more rapidly than pain, so that in the final ratings, distress was generally reported as greater than, or equal to pain.

These findings are similar to those reported by Knox, Morgan and Hilgard (1974), and confirm that emotional or psychological distress occasioned by prolonged pain, can in turn, affect pain perception. Tolerance measures of high hypnotisable subjects revealed significant increases in both pain and distress tolerance during hypnosis compared to normal states. The low hypnotisable subjects failed to produce significant increases in these conditions. Overall, hypnotic analgesia was found to effectively increase tolerance for both pain and distress by 63%. These findings indicate that the reduction in pain perception and distress, correlate positively with hypnotisability, and in this, they also support the findings reported by Hilgard and Morgan (1976).

A hypnotically induced dissociation, between the 'sensory-discriminative' and the 'affective-motivational' dimensions of pain experience, was found only in high

hypnotisable subjects. In their discussion, De Benedittis et al. (1989) point out, that although hypnosis was effective in reducing both pain perception and distress, distress was significantly more reduced than pain perception. This latter finding is consistent with the apparent paradox associated with hypnotic pain reduction, namely that the felt or perceived pain is reduced (overt response), while the involuntary physiological indication of pain (covert response) remains at nearly normal levels.

Hilgard (1993) comments that the commonly occurring experience of pain relief in the course of some distractions indicates that the awareness of pain can be reduced if there is some kind of absent-minded attention paid to it. The hypnotised person, free of the experience of pain, may have registered the pain in some dissociative fashion so that the memory of pain can later be recovered when appropriate techniques are used such as automatic writing.

Hilgard cites the Estabrooks (1957) case study in which a hypnotised subject was sensitive to pain caused by a pin prick on her anaesthetised hand which was involved in automatic writing. While there was no outward indication of a pain response, the writing hand responded by writing a string of invectives directed at the hypnotist, indicating an apparent experience of pain at a different level of consciousness. This phenomenon was rediscovered by Hilgard (1973) in the Stanford Laboratory and led to a series of research studies under the now familiar term of 'hidden observer.'

The hidden observer findings suggest that while there is genuine pain relief under hypnosis, pain is experienced at a cognitively covert level corresponding to physiological indicators of pain persistence. It appears that this phenomenon is not exclusive to

hypnotic analgesia. There is some evidence that a patient under chemical anaesthesia may in fact be storing some information about what is happening (Hilgard, 1993).

The concept of hidden observer has intuitive appeal. It helps to illustrate the capacity of the human mind to covertly 'register' an event such as pain, and at the same time, change the overt experience of it through an altered state of focus. While this intentional or unintentional focus diversion is known as dissociation, and has long been identified as constituting the basis of hypnosis, Hilgard's neo-dissociative postulate has elucidated this concept.

Watkins and Watkins (1990) suggest that when pain is displaced into a cognitively covert structural system referred to by Hilgard (1973, 1977) as 'the hidden observer,' it is actually displaced into underlying Ego-States. Ego-state theory assumes that human personality develops through processes of integration and differentiation. Normal differentiation is adaptive, and Ego-States have permeable boundaries. In the intermediate range of differentiation/dissociation, covert Ego-States can be found in most normal subjects. At the more extreme end of the range however, differentiation develops into maladaptive dissociation, which may lead to the creation of dissociative identity disorder (multiple personalities).

Watkins and Watkins (1990) go on to make the point, that irrespective of their differentiation/dissociation states, all individuals can displace (dissociate) pain into covert ego-states. The difference, within the context of the pain displacement theory, is that whereas Hilgard's (1973, 1977) neo-dissociation theory makes the more general claim that real pain perception is displaced into some covert cognitive structures, Ego-state theory takes it a step further by specifying that this displacement occurs in the covert

cognitive structures known as ego-states. Watkins and Watkins also reach the telling conclusion that as the pain is not eliminated by hypnosis, we may indeed, not be getting away 'scot free' (p. 1).

### 3.43 Pain as a Function of Psychological Disorder

The range of psychologically based pain is substantial, it includes the disorders of irritable bowel syndrome, Menieur's disease, bronchial asthma, arthritis, and chronic pain. Headache pain is the most common of the psychosomatic pain disorders. Pain may be symptomatic of various underlying psychological factors and motives, and although these are circumstantially unique to each individual, theories based on emotional needs and their gratification, have found general acceptance (Comer, 1992; Rose, 1990). The following gratification gains, grouped into three categories will serve as examples.

#### Pain as Punishment and Expiation of Guilt

Engel (1968) suggested that certain individuals have a psychogenic proclivity for chronic pain, and that such pain-proneness is linked to abusive childhood experience. The typical profile of a pain-prone patient would include some harsh parental treatment, or even abuse. The child would in due course, come to associate pain with self-blame, fear of losing a love object (the parent), inwardly directed anger, and possibly masochism.

More specifically, it is theoretically associated with the childhood need for reassurance where communication of pain, through crying, elicited a comforting and gratifying response from the loved one. Pain is therefore seen as punishment for misdeeds, leading to the expiation of guilt, forgiveness, and reunification with the loved one. And in accordance with behavioural theory, any pain behaviour which elicits a

favourable response, becomes reinforced as behaviour leading to a reward. Elton, Stanley, and Burrows (1978) comment that a guilt-ridden, self-punishing personality is one of the contributing causes of chronic pain, and that clinical experience suggests that such displacement may be of value to some individuals.

### Pain as a Manipulative Tool

The instinctive and growing recognition of how these gratifying responses may be elicited, also leads to the awareness that pain is a powerful manipulative tool; a tool for eliciting certain desirable responses which reinforce a personal sense of power, control, and provide other emotional gratification. Pain can also be masochistically self-inflicted and sadistically inflicted on others for sexual gratification (Engel, 1968). It may also be a tool in the form of martyrdom, and a covert discharge of hostile impulses against self and others.

### Pain as Self-esteem Enhancement

Pain may restore self-esteem through the elicited belief that the individual could function at superior levels in most or all areas, were it not for his 'pain handicap.' Feelings of guilt over non-performance or failures are thus also lessened. A pain-prone patient may have a mood disorder, somatoform disorder, personality problems or a very poor self-concept. Rose's (1990) findings support this claim. He reports that 70% of patients who suffer chronic pain in their lower back, have accompanying psychological symptoms such as depression, mood swings, and a negative self-image, and 50% of depressed patients feature pain as a major symptom.

The above categories of psychological factors underlying psychosomatic pain, represent only some of the psychological motives which can induce individuals to seek

refuge in pain. Although the psychological motives cited are small in number, they clearly give some indication of the range of emotional gratification, which chronic pain patients may derive from their disorder in return for their suffering.

Ready acceptance of the 'sick role,' and clinging to it irrespective of its inconvenience, suggest the presence of self-doubt about having the coping capacities required to deal adequately with daily problems. Pain in this context, represents a 'way out' of personal difficulties. The desire for gain is usually at an unconscious level. Elton and Burrows (1979) comment that pain-prone patients may escape into pain rather than escape from it, despite their desire to be free of it.

#### 3.44 Resistance to Pain Reduction

The onset of pain-proneness, or gaining by being invalidated through pain and suffering, can also occur later in life. After suffering an illness or injury, some individuals may have found themselves not only absolved of their 'overwhelming' responsibilities, but to be the recipient of increasing attention. These are considerable gains, and can lead to an unconscious reluctance on the part of patients, to surrender these gains in exchange for the alleviation of their pain. Such reluctance commonly manifests in some form of treatment resistance (Comer, 1995).

Elton et al. (1978) conducted a study to examine personality variables which could explain the phenomenon of non-improvement of some chronic pain patients. The study compared placebo 'treatment' with psychological treatments, including biofeedback, and hypnosis. The variable of particular interest, was that of self-esteem and its correlation with non-improvement. Results showed a significant link between 'persisting' pain and low self-esteem. Non-improvement was related to the psychological needs of

'persistent' pain patients not having been met by traditional, non-psychological treatment.

The finding of a significant relationship between low self-perception and pain persistence therefore supports the belief that resistance to pain improvement is linked to a 'patient perceived' psychological cost factor. That is, the greater the psychological dependence on an escape into pain, the greater the perceived cost and reluctance to surrender this coping mechanism, this 'way out.' It is reasonable to accept that low self-esteem carries with it a low estimation of self-coping skills, which, unless counteracted, is likely to contribute to an unconscious resistance to relinquishing any coping mechanism.

It is equally plausible, that just as such reluctance or resistance can diminish through an enhancement of self-perceived coping skills, so it can increase, if self-coping skills are subjectively perceived as further decreased. Such a situation can conceivably arise through the compounding effects of a coexisting psychological disorder. To this end, it may be prudent to consider some somatoform disorders classified in DSM-IV (1994, p. 445), as possible comorbid disorders which are likely to compound unconscious resistance to migraine treatment.

The terms psychosomatic or psychophysiological disorder, have traditionally referred to conditions of medical or somatic damage stemming from psychological, emotional problems. Chronic tension headaches and migraine are among the best known and most prevalent of these 'traditional' psychophysiological disorders (Comer, 1995; Graham, 1990). The terms psychosomatic, and psychophysiological, have in the past

been ambiguously and misleadingly applied to emotional or somatoform disorders such as hypochondriasis and conversion disorder, neither of which involve medical damage.

Clarification is now facilitated through the use of more rigorously descriptive terms adopted by DSM-IV. In contrast to the former traditional psychosomatic disorders such as migraine, which now fall under the term 'disorder of psychological factors affecting a medical condition,' the DSM-IV clearly separates the following purely psychological disorders under the heading of Somatoform Disorders: Hypochondriasis, Conversion Disorder, Pain Disorder, Body Dysmorphic Disorder, Somatization Disorder, Undifferentiated Somatoform Disorder, and Somatoform Disorder not Otherwise Specified.

A somatoform disorder is a physical expression of emotions. The symptomatology of some somatoform disorders may include chronic pain, hysterical blindness, hysterical paralysis (conversion disorder), high blood pressure, depression, and anxiety. Mental disorders, such as anxiety, or mood disorders, or somatoform disorders, may predispose the individual to chronic pain. Such disorders may co-exist with pain, or result from it (DSM-IV, 1994). This official recognition attests further to the close relationship between mental states and their somatisation, of which chronic pain may be just one of several symptoms.

Early diagnostic systems classified somatoform disorders as 'neuroses,' together with anxiety, and mood or dissociative disorders. DSM-IV provides the following clarification:

The common feature of the Somatoform Disorders is the presence of physical symptoms that suggest a general medical condition ...and are not fully explained by

a general medical condition, by the direct effects of a medical substance, or by another mental disorder... The symptoms must cause clinically significant distress or impairment in social, occupational, or other areas of functioning. In contrast to Factitious Disorders or Malingering, the physical symptoms are not intentional (p. 445).

Somatization Disorder, Undifferentiated Somatoform Disorder, and Somatoform Disorder not Otherwise Specified, appear the most suitable as comorbid considerations in determining contributory factors for unconscious treatment resistance in migraine. The reason for this selection is based on the following considerations and inferences.

The objective in this selection process is to select those somatoform disorders which, by adding to the range of the already existing psychological factors associated with migraine pain, are likely to compound or increase some patients' dependence on their migraine pain as a coping mechanism, or for other secondary gains. With this in mind, 'Pain Disorder' is not sufficiently different from the pain in situ, and is therefore not likely to achieve a compounding effect.

The unique specificity of Conversion Disorder, which involves deficits in motor and sensory functions, reduces the likelihood of this disorder being found in statistically significant numbers in a small sample. The same can be said of Hypochondriasis and Body Dysmorphic Disorder. As such, selection of these disorders, for purposes of this study, is impracticable. The suitability of the three somatoform disorders selected above, is inferred on the basis of their different, non-pain psychological nature, and their general, more commonly encountered symptomatology.

### 3.45 Hypnosis in the Treatment of Pain

Although known as an effective analgesic treatment in itself, hypnosis lends itself readily as a valuable adjunct to a wide range of therapies, particularly in the treatment of pain (Kroger, 1993). One of the earliest applications of hypnosis was in the field of pain control. The analgesic efficacy of hypnosis has since been repeatedly demonstrated in respect to both somatic and psychological pain, as well as pain arising from a combination of these factors (Stanley, 1993). Pain is not only a somatic problem, the exclusive result of injury or secondary inflammatory factors. It has many roots in the psychic economy of the person, and the therapist has to be prepared to deal with them, so therapy is often directed to the person rather than the symptom (Hilgard, 1993; Milne, 1995).

The analgesic effectiveness of hypnosis in the control of purely somatic pain is demonstrated in surgical cases where hypnosis is the sole anaesthetic, and in other cases such as obstetrics, and pain due to burns and advanced terminal disease (Milne, 1995). With the exception of severe pain due to terminal disease and surgical procedures, hypnotic pain reduction is usually aimed at partial reduction, rather than total removal of pain awareness. The reason for this is primarily one of precaution.

Hartland (1973) notes that total removal or alleviation of pain can be so effective that aetiological progress of the pain causing disease or trauma, may proceed without being noticed and without receiving timely treatment. Hartland goes on to describe a case of obstructed labour which went unnoticed for some considerable time because the patient's ability to control her pain was so complete and her state of relative comfort so

apparent, that nursing and medical staff were not alerted as soon as they should have been.

X/ Scott (1974) mentions a case in which hypnotic post-operative pain relief was so effective that the patient over-exercised to the point of tearing the sutures in his wound. The National Health and Medical Research Council Report (1982) alerts to the possibility that inappropriate symptom removal may disturb the pre-existing equilibrium of the patient and manifest as a displacement in the post-treatment phase. Further, the psychological mechanisms which produce symptoms, may also be serving to protect the integrity of the personality. Given these considerations, it has become established professional practice to forego radical removal of pain symptoms and to adopt a more moderate, reductionist approach. This approach is adopted even in cases where pain is known to be of purely psychological origin, that is, pain which does not mask an organic source (Karle, 1992). These consideration will be incorporated in treatment procedures adopted in this study.

A considerable number of hypnotic techniques have been devised by clinicians to enhance the effectiveness of hypnotic pain reduction. The most simple of these are direct suggestions in the form that the pain will reduce and become more tolerable. Studies have shown (Hilgard, 1993) that pain reduction is better achieved when the patient is given something specific to do to achieve it. These 'locus of control' tasks frequently involve visualization of specific pain lowering images.

One indirect approach (Rose, 1990) involves the patient imagining a freezing out of pain impulses in the affected area. Another similar approach, known as 'glove anaesthesia,' employs sensory focus in which the patient transfers the numbness of an icy

glove, or that produced by it, to the painful area. The patient may also imagine applying an anaesthetic gel to the affected area to reduce or change the nature of the pain.

Alternatively, the pain may be displaced to another, more convenient part of the body which may also be less sensitive, such as a finger (Milne, 1995).

It is possible to dissociate the self from the pain experiencing body, using images of leaving the body in one location, such as the sick-bed or the operating table, and withdrawing to the room next door or to some other non-painful location (Rose, 1990). Some techniques are devised to meet specific individual and pathological needs, such as the vascular manipulation technique employed by Harding (1978) in the treatment of migraine (see details in section 3.5).

Most techniques incorporate both 'ego-strengthening,' which encourages patients to believe and trust in their own ability and control, and 'relaxation' components. Hilgard (1993) points out that while the effectiveness of hypnosis in the treatment of pain correlates to the level of trance, even a non-hypnotisable patient can learn to relax and benefit from pain reduction obtainable through it. Hilgard writes further:

Another important adjunct to the therapy of pain is provided by self-hypnosis.

Recurrent pains, such as migraine headaches or cancer pains, may be controlled within hypnosis, but may occur again when a hypnotist is not present. Treatment is maximally effective if the patient can reinstate the hypnotic experience in the absence of the hypnotist. Some patients, and especially very young children, may find it difficult to undertake self-hypnosis. For them it is possible to prepare a cassette tape directed to the individual patient with the voice of the therapist (p. 241).

The success of these methods in the treatment of pain control has long been established in clinical experience and research studies. It is also well established that the effectiveness of hypnotic pain control can be further enhanced and reinforced through use of post-hypnotic suggestions framed to progressively increase the potency of the technique as the patient continues to practice it (Stanley, 1993).

Stam, McGrath and Brooke (1984) compared the effectiveness of hypnosis with that of relaxation training. The outcome of the study which involved 61 patients with temporomandibular (temple bone) pain, revealed hypnosis and relaxation to have been equally effective, and superior to the placebo control. Whorwell, Prior, and Faragher (1984) compared hypnosis, in the form of general relaxation and ego enhancement suggestions, with supportive psychotherapy in 30 patients with irritable bowel syndrome. Hypnosis was effective in reducing both pain experience and abdominal distension, but supportive psychotherapy produced no significant improvements.

Melzack and Perry (1975) conducted a study to compare the effectiveness of alpha wave feedback with hypnosis in the form of an ego-enhancing tape. Patients (N=24) with established chronic pain syndrome were randomly assigned to one of three conditions: alpha feedback, hypnosis, or a combination of both. Results showed that the combination treatment was the most effective, but hypnosis was found more effective in pain reduction than alpha wave feedback. The results of the above studies demonstrate the effectiveness of hypnotic relaxation training, and the use of tapes in the treatment of chronic, psychosomatic pain. Both of these approaches will be considered as treatment strategies for this study.

Hilgard and Hilgard (1975) studied the effectiveness of hypnosis in the reduction of cold pressor pain and ischaemic pain. The cold pressor pain was experimentally produced by placing one arm of subjects in an icy bath, and the ischaemic pain by the application of a tourniquet. Three basic approaches were used to produce pain relief namely, (a) by directly suggesting a reduction of pain, (b) altering the way the pain was experienced, and (c) by directing attention away from the pain and its source. Actual suggestion of pain relief was found to be an indispensable part of the treatment, as hypnosis by itself did not produce pain reduction. This factor will also be taken into account in the formulation of treatment strategies for this study.

Research findings lead to the conclusion that the effectiveness of hypnosis in the treatment of pain cannot be seriously doubted. Commenting on the findings of their study into the relationship between self-esteem and non-improvement in response to pain treatment, Elton et al. (1993) stated the following:

The benefits of hypnosis in this study extended beyond the relationship variables...

This study confirmed that hypnosis is more than a placebo. Hilgard and Hilgard (1975) have pointed out, hypnosis acts on both the sensory and the suffering component of pain. Hypnosis has been shown to be more effective than behavioural therapy. Its use can be recommended as a method of choice in the treatment of chronic pain (p. 279).

### 3.46 Hypnosis in the Treatment of Headache Pain

Migraine and tension headaches are among the most frequent psychophysiological disorders known as 'psychological factors affecting medical conditions' (Comer, 1995). Although tension headaches are pathologically distinct from migraines, there are some

pain related aspects which are common to both. For example, they share the elements of head pain in terms of chronicity, association with psychological factors, and a generally positive response to non-drug treatment involving hypnotic relaxation. Because of these similarities, it is appropriate to consider the results of recent studies undertaken to investigate the effectiveness of non-drug treatment in tension headaches.

As the name suggests, tension headaches are thought to be due to prolonged muscular contraction in the forehead, scalp, and neck (Lance, 1993). This muscular tension is ascribed to emotional stress, anxiety, and a tense apprehensive attitude. Stress management, incorporating some form of hypnotic relaxation, is one of the most frequently used non-drug treatments for tension headaches (Milne, 1995).

Spinhoven, Linssen, van Dyck, and Zitman (1992) compared the effectiveness of autogenic training with that of self-hypnosis in the treatment of headache pain and psychological stress. The study involved 56 patients suffering from chronic tension headaches. Results at both the completion of the study and at six months follow-up, revealed no significant differences in the efficacy of these two treatments.

Both methods produced significant effects in the reduction of pain and psychological stress, and both of these effects were retained at the time of follow-up. Short and long term pain reductions were accompanied by the patients' significantly increased awareness of their own 'locus of control,' and it was found that patients who attributed their pain reduction to their own efforts, achieved the best long term benefit retention rate.

Zitman et al. (1992) conducted a further study involving 79 tension headache patients. In this experiment the researchers compared the effectiveness of autogenic

training with hypnosis, and with hypnosis disguised under another name (unfortunately, the pseudonym used is not specified in the literature). Although the 'three' treatments were shown to have been equally effective at the conclusion of treatment, testing at six months follow-up showed that hypnosis explicitly presented as hypnosis, was superior to the other modalities in retaining the benefits long term. This result raises some interesting psychological questions about labelling effects.

Blanchard et al. (1991) incorporated a factor of 'locus of control' in their comparison of treatment effectiveness between relaxation training practiced exclusively under the guidance of the trainer without suggestion for home practice, and relaxation training which included regular cue-controlled self practice of the relaxation routine at home. The study comprised 27 patients suffering from chronic tension headaches, plus a control group of 6 headache patients who received no treatment but only monitored their headaches during the 8 week study.

While no change occurred for the control group, both treatment groups were able to achieve significant reductions in headache pain. However, the internal 'locus of control' group achieved a more pronounced trend in improvement. These findings lend further support to the view that self-involvement in pain control, such as may be facilitated through self-hypnotic relaxation or similar self-regulatory practices, enhances self-reliance and treatment results.

Rapoport and Sheftell (1991) comment that perceived inability to do something about a recurring headache problem generally increases anxiety and stress, and exacerbates the headache, but that when patients know how to achieve deep relaxation, they generally feel less panicked. The authors write, "...belief in the ability to control

one's headaches seems beneficial in itself, even if the belief isn't founded on anything more substantial than the researcher's comments" (p. 127).

Van Dyck, Zitman, and Linssen (1991) investigated the efficacy of autogenic training (AT) and 'future oriented hypnotic imagery' (FI) in the treatment of tension headaches. The researchers also explored the extent to which relaxation, hypnotisability, and imagery skills mediate therapy outcome. Fifty five patients completed the study, of these, 28 were randomly assigned to the standardised form of AT therapy (Schultz & Luthe, 1969), and 27 patients to the more individualized therapy involving relaxation and patient generated imagery FI. Each group was randomly assigned to one of two therapists.

Outcome measures used were self-report questionnaires and self-monitoring. Process measures were assessed with assessor ratings and self-monitoring. The main question addressed was whether 'future oriented hypnotic imagery,' with its more individualized approach was the more effective therapy of the two, as stressed by Erickson (1958, 1959). The researchers also sought to discover to what extent these variables correlate with the levels of relaxation and imagery skills of the subject.

Results of the study revealed that although no main effect or significant interaction for treatment conditions were found, subjects reported a significant post-treatment pain reduction of 40%. Significant and clinically relevant correlations of at least .40 emerged only in the future oriented hypnotic imagery condition. In this condition, headaches at post-treatment were found to be significantly and negatively correlated with depth of relaxation during home practice, and with hypnotisability and imagery skills.

Van Dyck et al. (1991) point out that these findings are only partly consistent with the neo-dissociation theory (Hilgard, 1977, 1979), which claims that hypnosis involves more than relaxation and deliberate focus on internal imagery. In this study the influence of relaxation emerged to be more important than assumed under the neo-dissociation theory, and the effect of hypnotisability on pain reduction could only be established in the analysis of subjective headache ratings, and not in headache index scores.

The lack of main effect between standardised relaxation (TA) and individualised future oriented relaxation (FI), indicates that a more individualised hypnotic approach as administered in the FI condition, is not more efficacious in pain reduction than a standardised hypnotic method of relaxation. This finding, however, is contrary to the views expressed by Erickson (1958, 1959).

### 3.5 Hypnosis in the Treatment of Migraine

Although literature on the topic of hypnosis in the treatment of migraine is sparse, experimenters (Anderson, Basker, & Dalton, 1975; Davidson, 1987; Emmerson & Farmer, 1996; Graham, 1975; Harding, 1967, 1978; Milne, 1983, 1995; Olness, MacDonald & Uden, 1987; Spiegel & Spiegel, 1978) have reported favourable results. Olness et al. (1987) conducted a study comparing the effectiveness of self-hypnosis with that of Propranolol (beta-blocker), and with pill placebo. The participants were 28 juvenile migraine patients. Results showed that the group practicing self-hypnosis achieved significant reductions in the frequency of their migraines, whereas the two groups in the propranolol, and in placebo conditions failed to achieve any significant result.

Davidson (1987) reported on treating a series of ten migraine patients with hypnosis. After the initial assessment and the recording of their migraine details in terms of migraine severity and frequency, hypnotic induction proceeded as per the Spiegel and Spiegel (1978) induction profile. After the induction, direct suggestions were given to effect circulatory changes in the head. Each patient was given a pre-recorded tape containing induction suggestions for self-hypnosis, plus the direct suggestions for circulatory changes, as previously administered to the patient. Patients were encouraged to practice with the tape every day in addition to practicing the technique without the tape two to three times daily. Second and subsequent appointments at the clinic were used for feedback on the patients' use of the technique.

The direct suggestions for circulatory changes were also taken from Spiegel and Spiegel (1978). These invited the patient to visualise and feel the icy cold of a large piece of ice slowly descending over the head. The cold was said to produce a numbness inside the head, making it possible for the patient to filter out the pain by focusing on the numbness rather than on the pain. The second part of the Spiegel suggestions invited the patient to imagine wearing wired, battery charged asbestos gloves which cause the hands to become warm. It was then suggested to the patient that the contradictory requirement of making the head cold and the hands warm would neutralise the extreme sensations felt with migraine pain. The patient was then invited to return to a normal state by counting backwards from three to one.

'Biological hypnotisability' was measured according to the Spiegel eye roll on a scale of one to three, with numerical increases indicating corresponding increases in the level of hypnotisability. Migraine frequency outcome was rated on three numerical

levels, one for 'complete remission, 'two for 'significant reduction,' and three for 'no effect.' Severity was rated in similar fashion with one for 'total or near remission, 'two for 'significant reduction,' and three for 'no effect.' Unfortunately, migraine duration was not measured.

Results indicated that of the total number of ten patients, seven patients achieved complete remission of their attacks, and five showed total or near remission in the severity of their attacks. Only one patient failed to show any improvement in either frequency or severity, and this patient had achieved an eye roll score of only one to two on a scale of zero to four. Davidson (1987) concluded that overall results of these serial treatments, suggest a correlation between hypnotisability and treatment outcome. These results can therefore be seen to support the views of Hilgard and Hilgard (1975), and Spiegel and Spiegel (1978), that deeper levels of trance enhance the effectiveness of treatment.

Harding (1978) reported on his use of a treatment method specifically adapted to the pathogenesis of migraine (excluding aura). The method, 'Harding's vascular manipulation technique,' was used on 194 consecutive migraine patients. Harding's standard treatment comprised five sessions. The first was used for notation of patient data and discussion. The Spiegel (1970) induction was administered in the second session. The third session was used to teach self-hypnosis incorporating ego strengthening, positive imagery suggestions, and the achievement of circulatory changes using the vascular manipulation technique described below. The fourth session was used to consolidate gains and to attend to difficulties. The final session was set aside for a time after the anticipated next attack. As improvements gained in the preceding four

sessions were usually maintained, additional appointments were only needed for unusual problems.

Follow-up questionnaires were sent out at 6 months, 2 years, and then every 4 or 5 years; some patients were followed up for 20 years. Follow-up data indicated that the treatment results were generally maintained. Of the total number of 194 patients, 77 (40%) achieved total remission, 87 (45%) achieved at least 25% reduction in severity, frequency or duration of their attacks. Only 30 (15%) failed to respond positively to the treatment. Because of the psychological aspects of migraine, it is important to note that only 4 (2.44%) of the improved patients reported substitute symptomatology. This result was attributed to the systematic use of self-hypnosis incorporating ego strengthening and positive imagery.

Harding demonstrated this vascular manipulation technique at his workshop conducted at the Queensland Branch of the Australian Society for Clinical and Experimental Hypnosis in 1978. The technique required the hypnotised patient to visualise the blood vessels on the surface of the migraine affected side of the brain getting smaller to reduce the flow of blood. This suggestion was given if the patient had on some prior occasions experienced relief from the application of an ice pack to the affected area.

If, in contrast, the patient had obtained relief from the application of heat, such as may be obtained from a hot water bottle, then the patient was to imagine the blood vessels expanding, carrying more blood and nourishment to the site, and carrying away bradykinins, excess serotonin, and other migraine producing substances which had accumulated in response to the vascular 'spasm.'

The reference to 'bradykinins' which are vasodilators, illustrates Harding's view that, contrary to current neurological thinking, the enlargement of the blood vessels during migraine is due to oedema (abnormal accumulation of fluid) in the walls of the vessels. And further, that the lumen or cavity is still obstructed as a consequence of the prodromal spasm, and not, as is now generally understood, that the enlargement is due to dilatation of the blood vessels.

The therapeutic results achieved with Harding's vascular manipulation technique are most encouraging, however, his oedema theory has emerged as flawed. Harding maintained in this theory, that vascular spasms lead to vascular oedema (an abnormal accumulation of fluid) which, in turn, leads subsequently to migraine. The flaw in the oedema theory lies in its inability to explain why, if enlargement is due to oedema and not to dilatation, vasoconstrictive agents like ergotamine are effective in providing relief by the very process of reducing dilatation? In the light of research advances made into migraine related vascular changes, Harding's oedema theory, which was formed in the 1970s, is no longer tenable. Fortunately, acceptance of that theory is not a necessary condition for the application of Harding's effective vascular manipulation technique.

Milne (1983) wrote about his successful treatment of 12 migraine patients over a period of 3 years. He noted that in 9 instances, hypnotherapy was specifically asked for by the patients due to their dissatisfaction with the low level effectiveness of their medications. The 3 different treatment methods employed were aimed at controlling blood flow in the brain.

Method 'A' consisted of hypnotic relaxation, ego strengthening, positive imagery suggestions, and Harding's (1978) vascular manipulation. Method 'B' involved a

combination of hypnotic relaxation, autogenic hand warming, and desensitisation.

Method 'C' comprised hypnotic relaxation, ego strengthening, and positive imagery suggestions. The author points out that the recorded results simply seek to illustrate the outcome of the different treatment approaches used over time, and that no comparative study was ever planned.

Treatment results show that 4 of the 12 patients had achieved complete remission at follow-up periods, which ranged from 6 months to 3 years. Of the 7 patients who received method 'A' treatment, 3 achieved complete remission, 3 achieved 'worthwhile improvement,' and 1 patient failed to respond. The only patient who received method 'B' treatment achieved 'near-complete remission,' and of the 4 patients who were treated with method 'C', 1 achieved complete remission, and 3 failed to respond to the treatment.

Notwithstanding the small number of patients and the lack of strict replication of conditions in these serial treatments, the overall results support the findings of Davidson (1987), Harding (1978), and Spiegel and Spiegel (1978), and show that self-hypnotic intervention based on strong visualisation, has a valuable role to play in the treatment of migraine. Of further interest is the fact that 12 years after the above Milne (1983) treatment results were published, Milne (1995) was able to report again on the follow-up results of this series, stating that "Trial analysis of treatments since indicates that the previous results have been maintained" (p. 31).

The analgesic effectiveness of hypnosis in general pain management, including non-migraine headaches, is extensively documented in medical, psychiatric, psychological, and other professional journals. However, documentation dealing with the analgesic

relationship between hypnosis and migraine management, specifically, is not only sparse, but is wanting in clinically controlled rigour. This lack of rigour has, in many instances, arisen because the cases presented were not intended to be part of a rigorous experimental study. They were drawn from serial case studies involving patients who were treated in the normal course of medical, psychiatric, or psychological practice over several years. The reports of Davidson, (1987), Harding, (1978), and Milne, (1983) are cases in point.

These serial treatments, nevertheless, provide some clinical insight into the analgesic potential of hypnosis in the treatment of migraine. Milne (1983) made the following comment:

The evidence would suggest that hypnotherapy could have quite a valuable role in the treatment of migraine. It may prevent attacks developing among a substantial proportion of patients, and its effectiveness would seem to last for years. In other cases, it can significantly reduce the severity and frequency of the attacks. The treatment programmes are brief, pleasant, and completely free from noxious side effects. Furthermore, by learning self-hypnosis, the subject is no longer a patient, but a person responsible for his or her own welfare (p. 29).

### 3.6 Summary Conclusions and Considerations in the Formulation of Research

#### Strategies

##### 3.61 Summary Conclusions

The foregoing review of literature, dealing with the pathogenesis of migraine and aura, and the analgesic effectiveness of hypnosis in the treatment of pain, has led to the following summary conclusions.

1. Migraine is understood to be the result of neuro-vascular activity causing a dilatation and swelling of extracranial blood vessels accompanied by migraine pain (Evans, 1988; Lance, 1993).
2. Few studies have been conducted to test the efficacy of hypnosis in the treatment of migraine, and there appear to have been no studies which measured the hypnotic treatment effects on the duration of migraine, as well as the usual measures of frequency and severity. Duration variable is of considerable importance, especially from the perspective of the patient as it represents the total number of hours spent in migraine pain or in the disabling symptoms of aura. These are factors which are conveyed neither by the frequency nor the severity variable.
3. Aura symptoms are produced in response to stimulation of the nerve pathways in the mid-brain, causing a constriction of blood vessels and reduction of blood flow of up to 50% in the affected mid-brain area (Comer, 1995; Lewis, 1988; Rose & Gawel, 1981).
4. Previous studies have not been conducted to test the effectiveness of hypnosis or other psychological treatments on aura symptoms. Approximately 20% of migraine patients suffer debilitating aura symptoms (Lance, 1993).
5. The brain's capacity to alter the transmission and perceptual quality of pain, also facilitates its psychological and hypnotic manipulation (Harding, 1978; Hilgard & Hilgard, 1975; Knox et al. 1974; Spinhoven, et al. 1992).
6. Psychological factors trigger migraine. Migraine is one of the 'traditional' psycho-physiological disorders (Comer, 1995; Graham, 1990), and anxiety as well as depression, are part of the migraine profile (Spiegel, 1965).

7. Stress increases the release of adrenalin, which leads to vasoconstriction, and which, in migraine prone individuals, may further lead to aura, prodromes, and migraine (Comer, 1995).

8. Prostaglandins are released as a counter measure to vasoconstriction, leading to vasodilatation, and migraine in migraine prone individuals (Lance, 1993).

9. Serotonin levels are excessively high during vasoconstriction, prodromes and aura, and low during vasodilatation in migraine. Low levels are accompanied by negative affects on mood and pain control, which helps to explain why migraine is frequently accompanied by anxiety, depression, and low motivation (Carlson, 1986; Graham, 1990).

10. The strong presence of psychological factors in migraine pain, suggests, that the use of a psychological therapy such as hypnosis, will be an effective treatment for this pain disorder (Comer, 1995; Davidson, 1987; Finer, 1993; Hilgard, 1993). Hypnosis has the capacity to address pain at sensory and perception levels (Chertok, 1993).

11. Group hypnosis, as well as self-hypnosis, have, in addition to applied hetero-hypnosis, been shown to be effective therapy modalities in the treatment of pain (Braun, 1993; Harding, 1978; Melzack & Perry, 1978).

12. The eye roll technique is an effective aid in the induction procedure, and guided imagery is an effective technique to facilitate deepening and treatment input (Spiegel & Spiegel, 1993; Stanley, 1993).

13. Indirect suggestions are effective for overcoming induction resistance (Erickson & Rossi, 1980). Direct suggestions are effective for treatment input, post-hypnotic compliance, and alerting (Chertok, 1993; Hartland, 1971).

14. Low self-esteem or self-perceived inability to cope with psychological stress, increases anxiety and stress (Rapoport & Sheftell, 1991), and suggests that chronic psychological pain such as migraine, is a behavioural 'escape into pain' response (Elton & Burrows, 1979; Elton et al. 1978).

15. Low self-esteem or self-perceived inability to cope, may, when accompanied by chronic psychological pain, result in resistance to pain improvement. The relinquishing of habitual pain involves a psychological cost factor, where the greater the need to 'escape into pain,' the greater the reluctance to surrender this psychological coping mechanism (Elton et al. 1978).

16. It is likely that, just as increased self-coping skills or internal locus of control can reduce treatment resistance (Elton et al. 1978), so a more depleted sense of coping ability, may increase such resistance (see section 2.15). This might be occasioned through comorbidity of a somatoform disorder (see section 5.14).

17. Health related quality of life of migraine sufferers is below normal in terms of productivity, social role, and psychological well-being. The quality of life of patients suffering comorbidity is significantly further reduced (Osterhaus et al. 1994).

These summarised conclusions, and the factors set out below, combine to facilitate an outline in point form of the research strategies to be employed in this study. Detailed descriptions of the methods adopted and the results achieved, are presented in Chapter 4.

### 3.62 Considerations in the Formulation of Research Strategies

1. In order to minimize focal complexities and the possibility of error in the self-administered treatment of aura, the treatment focus was directed at normalizing its accompanying vasoconstriction, rather than directing it at its varying neurological

scotoma. No previous research has tested the effectiveness of hypnosis, or other psychological therapies, in the treatment of migraine aura. Similarly, the measure of duration, which is of major concern to the migraine patient, is commonly overlooked in studies testing non-drug migraine treatments. This study will undertake both these tasks.

2. Hypnosis is known for its efficacy in the treatment of pain (Carlson, 1986; Comer, 1995; Hilgard, 1975). Light to medium levels of hypnosis have been shown sufficient for non-surgical treatment such as pain reduction in psychosomatic disorders. It is estimated that in excess of 90% of the population can attain these levels of hypnotisability (Erickson & Rossi, 1980; Hilgard, 1968; Milne, 1995). In view of these factors, and in accordance with prevailing clinical conditions and practices, hypnotisability tests for participants will not be administered.

3. A combination of direct and indirect suggestions, and the Spiegel eye roll induction technique, will be employed to maximise the effectiveness of induction and treatment procedures. Hypnotic relaxation, especially when accompanied by guided imagery, is beneficial for the relief of stress and chronic pain (Jackson, 1989; Rose, 1990; Stam et al. 1984). When self-induced, it also increases coping skills and internal locus of control. It regulates the production of insulin, noradrenaline, prostaglandins, adrenaline, and levels of serotonin, and as such, it has the potential to alleviate the vasoconstriction of aura and prodromes.

4. Serotonin is understood to be involved in pain control, aura and migraine, as well as anxiety and depression (Carlson, 1986; Comer, 1995; Lance, 1993; Low, 1987). Self-hypnotic relaxation is included in this study as one of four modalities which combine to constitute one treatment. This daily relaxation procedure is aimed at addressing the

above aspects in aura and migraine profiles, and to reduce their severity, frequency, and their duration.

5. Vasoconstriction of aura and prodromes, as well as the vasodilatation in fully developed migraine, can be alleviated with specific hypnotic imagery following induction (Harding, 1967, 1978). The imagery used in the present study is aimed to facilitate a normalisation of the vascular system, and pain reduction in terms of severity and duration through the imagined application of heat or cold, respectively.

6. Imagery used in the case of aura and prodromes, will suggest that heat is being applied to the affected blood vessels in the mid brain. This 'experienced' warmth is aimed to relax and open the blood vessels, thereby restoring normal blood flow to the affected area. Imagery for migraine, will suggest that cold is being applied to the swollen and dilated blood vessels in order to facilitate a reduction to their normal size (Harding, 1978; Spiegel, 1978).

7. Group hypnosis can be advantageously used for ego-enhancement, increasing self-confidence and self-esteem. It is also of assistance in the training of self-hypnosis (Braun, 1993; Harding, 1978). It can also encourage post-hypnotic responsiveness, and co-operation with the essential requirements of the research program, ensuring adherence to recording procedures, and thus improve the reliability of recorded data (Braun, 1993). Self-hypnosis provides a strong and effective psychological coping mechanism, thereby increasing self-reliance and locus of control (Comer, 1995; Milne, 1995; Stanley, 1993).

8. The transition from group to self-hypnosis, and adherence to specified hypnotic procedures can be further enhanced by means of pre-recorded audio tapes

(Melzack & Perry, 1975). The pre-recorded suggestions also provide the means for standardising the three hypnotic treatment components of relaxation, the imagined application of heat in the case of aura, and the application of cold in the case of migraine (Davidson, 1987).

9. Considerations regarding the prevalence and impact of comorbid somatoform disorders have, for the purpose of this study, been restricted to Somatization Disorder, Undifferentiated Somatoform Disorder, and Somatoform Disorder Not Otherwise Specified. The impact of this comorbidity will be assessed in terms of treatment responsiveness and treatment resistance.

10. Although the prime objective of this research is to test the analgesic effectiveness of hypnosis in the treatment of migraine and aura, other psychological aspects associated with migraine, such as depression, quality of life, and comorbid somatoform disorders, will also be assessed.

## EXPERIMENTAL CHAPTERS

### CHAPTER 4

#### The Clinical Experiment

##### 4.1 Introduction

Migraine affects approximately 1.5 million Australians. The majority of migraine sufferers are in their prime years of career achievement, between 24 and 54 years of age (The Australian Brain Foundation, 1993). The painful and frequent impairment of functional ability suffered by male and female migraine patients, even when on medication, does more than negatively impact on their personal and family life. Studies by Osterhaus et al. (1994) and by Solomon et al. (1993) have shown that the quality of life of migraine patients, after adjustment for comorbidity, was significantly lower than that of the non-afflicted population. Such suffering also detracts from patients' productivity in the work force. These ongoing private, and public sector costs indicate that a self-help treatment, capable of minimising personal suffering and reducing the national cost of migraine of \$264 million per annum, would constitute a welcome and nationally significant benefit. This study sought to determine the extent to which hypnotic treatment can help to facilitate such benefit.

More specifically, the treatment objective of this study was to administer one integrated treatment for migraine and one integrated treatment for aura. However, with the exception of the contra-distinctive migraine and aura vascular manipulation modalities, the treatment streams were identical. The treatment comprised four individual hypnotic procedures or modalities in all. These were: group hypnosis

administered as treatment in the course of training, self-hypnosis for relaxation, self-hypnosis for migraine, and self-hypnosis for aura treatment. Each stream comprised only three modalities to the exclusion of either the migraine or aura treatment. The rationale for the employment of three treatment modalities within each integrated treatment stream, was that this manifold approach would increase the effectiveness of the hypnotic treatment as a whole. The choice of each modality is explained in section 5.16.

The psychophysiological nature of migraine, the psychological nature of hypnosis, and its efficacy in the treatment of pain, as reviewed and summarised in the preceding chapters, provide the background to the variables which were experimentally studied. This chapter focuses on these variables and on the research strategies employed to test and analyse them. The following factors constitute the rationale for the hypotheses of this study:

1. Migraine (including aura) is officially recognised as one of the traditional psychosomatic disorders (Comer, 1995; DSM-III, 1992).
2. Its psychological nature makes it particularly receptive to psychological treatment, as evidence by previous studies (Emmerson & Farmer, 1996; Harding, 1967, 1978).
3. Hypnosis is a psychological treatment known for its efficacy in pain management (Hilgard & Hilgard, 1978).
4. Hypnosis is therefore likely to be an efficacious psychological therapy in the treatment of migraine.

The hypotheses of this study, stated as null-hypotheses, are as follows:

1. There will be no significant pre/post treatment differences in the duration of migraine pain.
2. There will be no significant pre/post treatment differences in the frequency of migraine pain.
3. There will be no significant pre/post treatment differences in the severity of migraine pain.
4. There will be no significant pre/post treatment differences in the duration of aura symptoms.
5. There will be no significant pre/post treatment differences in the frequency of aura symptoms.
6. There will be no significant pre/post treatment differences in the severity of aura symptoms.

The .05 ceiling (alpha) of acceptance for probability of error was set as the rejection criterion for these hypotheses. A Bonferroni adjustment to alpha to compensate for multiple testing resulted in a .008 criterion for all hypotheses. The statistics used for analysis are noted in the results.

#### 4.2 Methods

The researcher was reminded and guided by the views expressed by Sheehan and Perry (1993).

Experimental designs in hypnosis never make every comparison conceivable.

Rather, researchers select methods that appear the most relevant to them, their selection being determined both by the theory preferred and the question being asked on any particular occasion of testing (p. 521).

The research design adopted for this study was a single group time series, comprising a pre-treatment and a post-treatment phase. Each phase was of 12 weeks duration. A one-week interval between phases one and two, was incorporated to allow participants to be trained in the self-administering of hypnotic treatment.

Phase I was used to record daily pre-treatment data for fortnightly collection and measurement. These recording procedures, which covered details concerning migraine, aura, and quality of life factors, were extended for Phase II, to include details of administered treatment. Details of these pre and post-treatment procedures are discussed in sections 4.22 and 4.23.

The single group design was chosen because of the relatively small number of (32) participants commencing the study, which, in the event of marked attrition, would make residual numbers too small for meaningful group comparison.

The time series design is a quasi experimental design capable of indicating causal effects by eliminating all threats to internal validity, with the exception of history. There was no threat of history in this study, and therefore no indication that it would impact on the results.

#### 4.21 Participants

Recruitment of migraine sufferers for participation in this study was undertaken through press advertising, display of posters in supermarkets, pharmacies, medical clinics, and referrals from migraine clinics at the Austin Hospital, and the Royal Melbourne Hospital (see Appendix A). Inquiring migraine sufferers were sent an acknowledgment letter containing further information, (see Appendix B) and a registration form for attending a two-hour information seminar (see Appendix C). The

completed registration form was to be returned if migraine sufferers wished to attend the seminar, and if they met all the selection criteria set out on the form.

The final selection of 32 volunteers was made on the basis of specified selection criteria, and the signing of a participation consent form (see Appendix D). The selection criteria included the requirements that participants be clinically diagnosed migraine sufferers, that they experience at least two migraines per month, and that they would not undergo any other drug-free migraine treatment in the course of this study. Attrition occurred only during the first 12 weeks of the study (prior to treatment) and was due to various domestic and work related changes. Attrition reduced the original number of 32 participants, to the 25 participants who completed the study; of these, 6 were male, and 19 were female. Participants received no financial compensation for their participation in this study. Their ages ranged from 36 years to 68 years. The mean age was 51 years, and the median age was 49 years.

#### 4.22 Instruments

A questionnaire was designed to obtain baseline information (see Appendix E). It contained 25 items; 6 items pertained to the severity, duration, and frequency of migraines and auras, 9 pertained to quality of life factors, 1 referred to stress, and 9 items related to medication and demographic data.

Participants recorded their daily details regarding the severity, duration, and frequency of each migraine and aura, as well as details of their hypnotic treatment on Log Sheet A (see Appendix F). The hypnotic treatment details recorded, provided information regarding the date, the number of times, and the length of time each of the

three self-hypnotic treatment modalities were practiced, as well as the level of hypnosis reached in each of them.

Log Sheet B was used by participants to record daily details regarding their 'quality of life', and stress levels. The 9 quality of life items detailed in this log sheet, included self-esteem, sense of control, task achievement, confidence, and health. A tenth item pertained to stress. The details and definitions of these items are presented in Appendix G.

Audio tapes were used to provide the three standardised hypnotic treatment modalities of relaxation, vascular manipulation for migraine, and vascular manipulation for aura. Side A of the tape (relaxation and migraine) contained instructions for the use of the tape, a hypnotic induction procedure for relaxation, and hypnotic suggestions aimed to reduce the vascular dilatation and swelling symptomatic of migraine pain.

Side B of the tape (relaxation and aura) also contained instructions for the use of the tape, and a similar induction procedure for relaxation. However, on side B of the tape, the hypnotic suggestions subsequent to relaxation, were aimed to reduce the vascular constriction symptomatic of aura. The verbatim script of side A is contained in Appendix H, and the verbatim script of side B is contained in Appendix I. The script used for group induction and training, which was aimed to familiarize participants with hypnosis, and to enhance the success of the treatment, is contained in Appendix J.

The instrument used to determine the prevalence of coexisting somatoform disorders among participants, was a questionnaire specifically designed for this purpose, the 'General Health Questionnaire' (see Appendix K). Its content was a composite of DSM-IV diagnostic criteria for Somatization Disorder, Undifferentiated Somatoform

Disorder, and Somatoform Disorder Not Otherwise Specified. The questionnaire comprised 19 questions, covering 41 symptoms and 3 conditions relating to time factors and functional impairment.

The Dishman and Ickes (1981) Self-Motivation Scale was used to test participants' self-motivation. This scale comprises 21 negative items (not motivated) and 19 positive (motivated) items, each on a 5 point scale. The test has been used by several researchers (Thackwray-Emmerson, 1987), and has a high reliability rating with Cronbach alpha of .91 (Dishman & Ickes, 1981).

The IPAT Personal Assessment Inventory (depression scale) was used to assess the prevalence and level of depression. The IPAT scale comprises 40 items, and takes approximately 15 minutes to complete. In view of the strong correlation of .80 which has been shown to exist between depression and anxiety (Krug & Laughlin, 1976), the test facilitates a scoring method with correction factor; scores may then be converted to STEN norms.

The uncorrected raw score, derived from only 36 items, identifies pure depression, whereas the corrected score, derived from 40 items, includes anxiety factors. A comparison of reliability between corrected and uncorrected scores, shows that the latter have a Cronbach alpha coefficient .94, and a parallel split-half reliability of .96. The reliability of corrected scores is .91, and .94 respectively. In terms of validity, the IPAT Personal Assessment Inventory (depression scale) has a correlation of .88 between the 36 item scale and pure depression (Krug & Laughlin, 1976).

#### 4.23 Procedures

Permission to conduct this research with human subjects was granted by the Ethics Committee of the Victorian University of Technology. Initial contact with migraine patients was made by telephone or correspondence in response to their inquiries about the publicised research program. Information regarding selection criteria and the forthcoming information seminar (see Appendix C) was mailed out to each migraine patient who expressed interest. Migraine patients who met all selection criteria were invited to register and attend the seminar, which was conducted three days prior to the study. A procedural flow chart is presented in Figure 1.

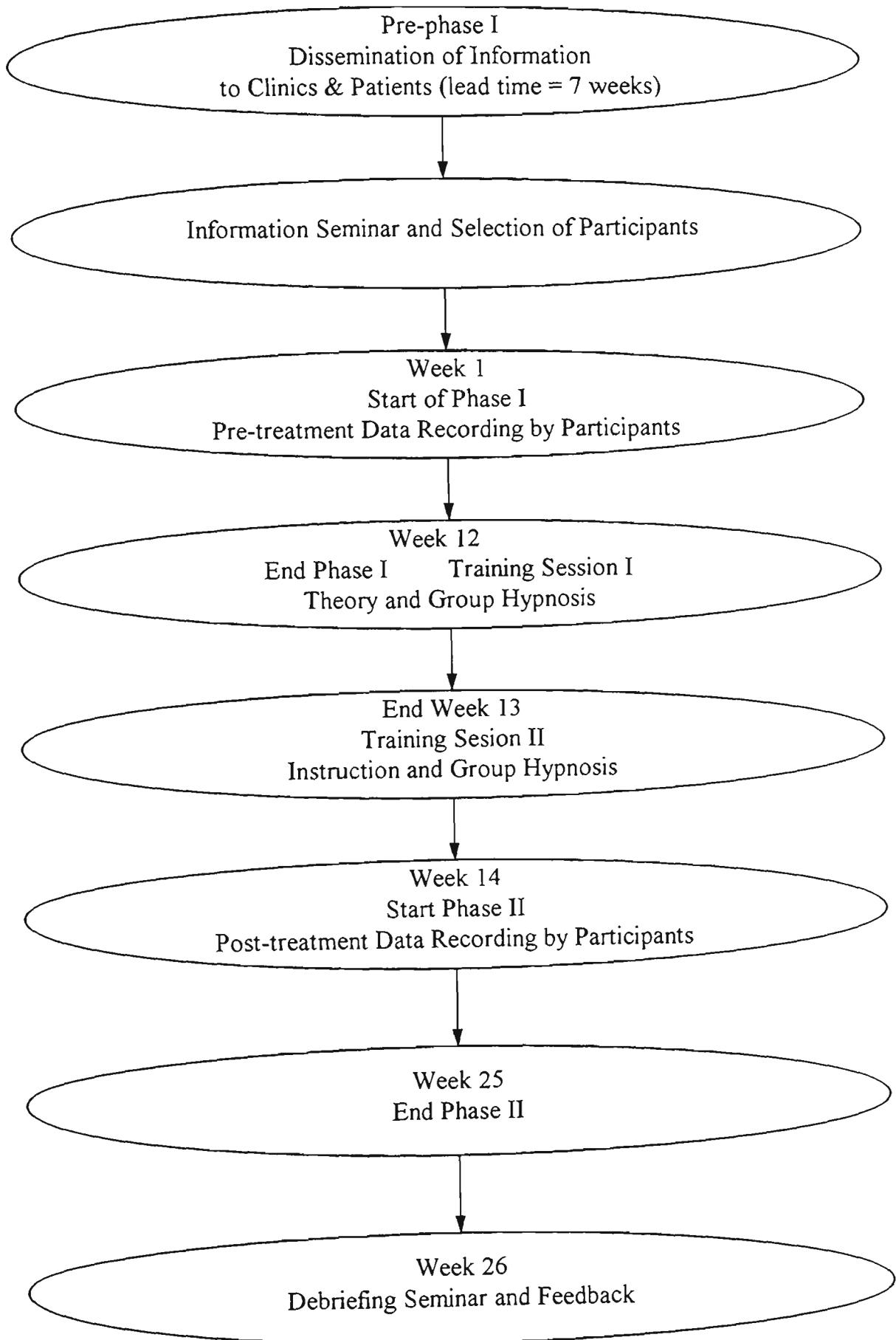


Figure 1. Chronological Flow Chart of Major Procedural Events

### Information Seminar

The information seminar served the purpose of explaining the aim of the research, the nature of the hypnotic treatment, and the requirements of participation in terms of daily record keeping self-hypnosis, and the six months duration of the study. Participants were informed that the success of this research in no way depended on their success to reduce their aura or migraine, but rather, that it depended on the accuracy and reliability of their recorded data. It was therefore scientifically important, that their recorded details were accurate. This emphasis was designed to remove possible performance bias, motivated by participants' desire to meet the 'expectations' of the researcher.

Notwithstanding the correlation between increased effectiveness of hypnotic pain reduction and deeper levels of hypnosis (De Benedittis et al. 1989; Hilgard & Hilgard, 1975), it was decided not to test participants' hypnotisability because (a) this factor was not a variable of interest, and (b) to enhance the generalisability of the results (in a clinical setting, patients are entitled to hypnotic treatment, and usually receive it, irrespective of their hypnotisability).

The relevance of the stated selection criteria was again stressed, and all migraine sufferers who satisfied the criteria and who confirmed their willingness to participate at the end of the information period, were formally accepted, and asked to sign a Consent Form (see Appendix D). This selection or acceptance procedure reduced the possibility of researcher bias. Participants were then briefed on daily recording procedures for Log Sheets A and B (see Appendices F and G), and asked to mail their completed log sheets to the researcher on each of the fortnightly dates specified.

Participants were also encouraged to telephone the researcher whenever they needed assistance or clarification. The researcher's mobile telephone line was kept open seven days a week, throughout the six months study. The seminar concluded with the pre-treatment administration of the IPAT Personal Assessment Inventory (depression scale), and the baseline questionnaire (see Appendix E). This baseline information provided a measure of comparison in the monitoring of participants' data during the pre-treatment phase.

#### Pre-treatment Data Collection

The researcher contacted each participant on an average of once every 9 days during the pre-treatment phase, and once every 7 to 8 days during the treatment phase. These monitoring contacts were found to be valuable in terms of data verification, and clarification when required. This ongoing 'connectedness' may also have contributed to keeping the attrition rate below 23%.

Throughout these contacts, care was taken by the researcher not to personalise the communications or influence participants' performance, thus maintaining the boundaries of scientific objectivity. All treatment instructions and hypnotic suggestions were standardised on audio tape or administered to the group as a whole, to reduce the possibility of extraneous influences.

#### First Training Session

The end of the 12 week pre-treatment phase was followed by an interval of 1 week. During this time, which was kept free of data recording, participants attended 2 training sessions, each one of 3 hours duration, in preparation for the 12 week treatment

phase (see procedural flow-chart, Figure 1). Each of the 2 training sessions comprised theoretical and practical components.

In the first training session, participants received broad-based theoretical instruction on the vascular systems involved in the two distinct processes of vasoconstriction in aura and the early phase of migraine (Sachs, 1985; Milne, 1995), and the vasodilatation of full migraine (Lance, 1993). This information was deliberately kept short and simple in order not to complicate participants' hypnotic focus or cause fear.

Information was given about the efficacy of hypnosis in the treatment of pain reduction, and as a form of relaxation. Practice of relaxation was explained as one of the four treatment modalities in this study, and participants were told that the prescribed daily practice of this self-induced hypnotic relaxation was likely to contribute to a reduction in the frequency of auras and migraines. Since this information would normally be given in a clinical setting by therapists using hypnosis with migraine sufferers, it was considered important to provide it in this study, in order to maintain clinical generalisability of any significant results.

These theoretical instructions were followed by an introductory group practice of the eye roll technique (Spiegel, 1973), and the muscular relaxation component of the relaxation section contained on side A of participants' treatment tape (see Appendix H). Participants were then progressively alerted by counting backwards from five to one. The purpose of this 'introductory practice', was to relax participants, and to facilitate greater responsiveness to suggestions (Stanley, 1993) given in the course of the next induction at the end of this training session.

After a 15 minute break for refreshments, participants were encouraged to ask questions. A discussion of approximately 15 minutes followed, indicating a positive response to the concept of hypnotic treatment. Participants' positive reactions were reinforced with the suggestion that the practice of their self-hypnotic treatment was likely to be successful.

This 'self-fulfilling prophesy' aspect of the group treatment, may be seen to introduce the possibility of a placebo effect. However, the decision to include such potentially 'self-fulfilling' psychological suggestions, was a considered one.

Notwithstanding the need to avoid the possibility of confounding variables which cloud the attribution of treatment effects, the inclusion of these suggestions is legitimate and justifiable on the following grounds. Hypnosis is essentially a psychological therapy, and as such, any treatment effects produced in response to psychological beliefs which were induced under hypnosis, are the effects of hypnosis. And in this respect, hypnotically induced 'self-fulfilling prophesies' can be seen to be distinct from purely psychological placebo effects.

Hypnotically induced beliefs are therefore always part of the hypnotic treatment, and not extraneous or confounding influences on the treatment and its effects. The factor which prompted the final decision to incorporate these suggestions into the treatment, was that these suggestions were likely to enhance participants' confidence in the treatment, and in their own capacity to use it successfully (Elton et al. 1993). It was expected, that these beliefs would in turn, enhance the effectiveness of the hypnotic treatment as a whole.

The next part of this training session comprised group hypnosis using the full version of hypnotic relaxation and treatment input (see Appendix J). Participants were then given their standardised treatment tapes and asked to practice the relaxation component every day. Home practice by participants of the aura and migraine sections was to be deferred until after their next training session. It was recommended however, that participants listen to those sections once or twice, to determine whether any points required clarification.

### Second Training Session

The second training session was conducted one week later. It was opened with a reminder to participants, that the success of this research in no way depended on their success in reducing their auras or migraines, although their success was likely and desirable for their own benefit. It was also reiterated, that the accuracy of their daily records was of great scientific importance. Participants were then invited to talk about their relaxation experiences of the past week, and to ask clarifying questions.

This discussion period was followed with a short summary of vascular responses in aura and migraine, as discussed in the previous training session. The summary led to the introduction of the topic of vascular manipulation as an effective hypnotic treatment for migraine (Harding, 1978), and as an effective treatment for aura. This was followed with a session of group hypnosis, using the eye roll technique and muscular relaxation as suggested on side A of the treatment tape (see Appendix H). As in the first training session, the main purpose of this short induction, was to facilitate, in the next session of group hypnosis, a deeper level of trance, and greater receptivity to treatment suggestions.

After a short interval, the use of the taped aura and migraine sections, as well as the required recording methods on log sheets, were discussed in detail. Participants were encouraged to telephone the researcher in the event of any queries. The Dishman and Ickes (1981) Motivation Test was then administered, requiring approximately 20 minutes to complete. On completion of the test, the researcher conducted the final session of group hypnosis for treatment purposes. This session comprised the full version of group hypnosis with treatment input relating to participants' treatment success. Post-hypnotic suggestions were given for increased effectiveness of practice over time, and for compliance with the requirements of accurate data recording (see Appendix J). The session concluded with participants providing written information regarding bilateral or unilateral aspects of their migraines. This information is summarised in section 4.32.

#### Debriefing Seminar

Following a 12 week treatment phase, a debriefing seminar was conducted. Participants were asked for written comments on their participation and treatment experiences in general, and more specifically, on perceived differences in terms of their medication and the impact of the treatment on other areas in their life. At this stage, the researcher was able to convey to participants their individual percentage scores in reducing the duration, frequency, and severity of their auras or migraines, or both.

This segment was followed by a second administration of the IPAT Personal Assessment Inventory (depression scale) for pre/post-treatment comparison. The debriefing seminar concluded with a final session of group hypnosis in response to participants' request. The hypnotic input in this session facilitated relaxation through tranquil forest imagery as an alternative to the beach imagery practiced in the course of

treatment. Post-hypnotic suggestions in this session, were aimed to encourage participants' continued success in their self-administered hypnotic treatment.

#### 4.24 Limitations

The limitations of this study pertain to (a) the reliability threat generally associated with self-reporting methods, (b) the threat to external validity associated with research conducted by only one researcher, and (c) the threat to external validity associated with a non-random sample. Regarding the first limitation, it is believed that the use of standardised log sheets, the repeated emphasis by the researcher on the importance of accurate recording, and the use of post-hypnotic suggestions to reinforce compliance with this request, have minimized this threat.

Regarding the second limitation, that of only one researcher undertaking the research in this study, there is no reason to believe that the abilities of this researcher vary in any marked or significant way from the abilities of other researchers in the field. Regarding the third limitation, although the sample was not randomly selected, all persons who were interested in participating, and who met the criteria for entry, were accepted for this study. While imperfect, practical considerations prevent the possibility of any true random selection from a population of migraine sufferers.

#### 4.25 Data Analysis

Four statistical procedures were utilized to test the null hypotheses pertaining to both migraine and aura. Due to the proposed use of multiple t-tests in the analyses, alpha inflation became a statistical concern. To minimise the threat of a type 1 error, a Bonferroni adjustment was applied. This adjustment effectively reduced the acceptable alpha to 0.008 for all t-test comparisons on migraine and aura.

In response to information derived from the examination of data, the modified Kolmogorov-Smornov test (Hair, Anderson, Tatham, and Black, 1995) was employed to assess the normality of distribution of dependent variables. Three variables (duration and frequency of migraine in phases 1 and 2, and duration of aura phase 2) were found to be significantly (positively) skewed. Their distributions were normalised using logarithmic transformations (a transformation for positively skewed variables), thereby satisfying the t-test assumption of normality (Hair, Anderson, Tatham, and Black, 1995).

Although the variable of duration aura phase 1, was not significantly skewed, it was statistically appropriate to transform it for comparison with aura duration phase 2 (Aron & Aron, 1994). Details are presented in Tables 1 and 2 respectively. A total of six dependent, two tailed t-tests were then employed with hypothesised dependent variables to determine the statistical significance of any pre and post treatment differences.

Duration, frequency, and severity, were the hypothesised dependent variables of both migraine and aura. Comparisons of duration and frequency were based on group totals, whereas all comparisons of severity were based on group means. The hypnotic treatment, comprising group hypnosis, hypnotic relaxation, and vascular manipulation 'A' (vasoconstriction), was the independent variable for migraine, whereas the treatment comprising group hypnosis, hypnotic relaxation, and vascular manipulation 'B' (vasodilatation), was the independent variable for aura.

Quality of life, depression, motivation, stress, and comorbid somatoform disorders, were the non-hypothesised dependent variables. The statistical procedures employed to analyse hypothesised variables, were also adopted to analyse non-hypothesised variables.

In addition however, an analysis of variance was conducted to test for treatment differences between comorbid and non-comorbid somatoform participants.

### 4.3 Results

The research data are presented in five parts. Section 4.31 presents the formulation and transformation of variables. Section 4.32 provides the demographic data. Section 4.33 presents the results of the hypothesised variables for migraine, and section 4.34 presents the hypothesised variables for aura. Results for non-hypothesised variables are set out in sections 4.35. through 4.35.4.

#### 4.31 Variable Formulation and Transformation

The role of any treatment directed to reduce migraine pain, can be seen to be threefold, pain reduction in terms of (a) the extent to which it is felt, that is, its severity, (b) the total length of time for which it continues, its duration, and (c) in terms of its recurrence, its frequency.

Similarly, any treatment directed to reduce aura symptoms, can be seen to involve the same three measures. It is a matter of the extent to which aura symptoms induce suffering (severity); the length of time they have to be endured (duration), and the rate of their recurrence (frequency). The dependent variables for both migraine and aura, were therefore severity, duration, and frequency.

The data for these dependent variables were obtained from participants' Log Sheets A and B (see Appendices F and G) which were recorded on a daily basis and mailed to the researcher on specified fortnightly dates for computer entry. Duration and frequency scores from each participant, were individually totalled each week by the researcher to provide an ongoing overview of individual responsiveness. Participants'

individual severity scores were converted by the researcher to individual weekly means. However, at the completion of this study, all individually recorded data were combined within each measure and within their respective pre-treatment or post-treatment phase. This yielded aggregate group totals or group means for each phase, and facilitated time series graphs indicating pre and post-treatment trends.

All data analyses in this study, are therefore based on pre and post-treatment group scores. This also applies to the non-hypothesised variables examined in this study. These include variables commonly associated with the migraine profile, namely depression, quality of life, stress, and motivation.

Depression data were obtained with the use of the IPAT depression test, which was administered at the information seminar prior to the commencement of Phase I, and again, after treatment, at the debriefing seminar. Motivation levels were determined with the Dishman motivation test, which was administered prior to treatment. Quality of life data, obtained from Log Sheet B, comprised nine factors of quality of life (see details and definitions in Appendix G). The weekly means of participants' daily scores for each factor, were computed and recorded by the researcher. The same procedure was adopted for the separate domain of stress.

Comorbid somatoform disorders was examined as a variable not previously researched in association with migraine. This variable was examined in relation to depression and quality of life. The rationale and implications of this analysis are discussed in section 5.14.

Table 1 presents details of the Kolmogorov-Smirnov assessment of the distribution of dependent variables. Table 2 presents the results after the employment of logarithms

to restore normality of distribution to those variable which were significantly (positively) skewed. The correction was achieved in each case. Although these transformations introduced a measure of proportional change or elasticity into the relationships of variables, the interpretation of these measures was not changed.

Table 1

Normality of Distribution: Kolmogorov-Smirnov Test

Variable	Kurtosis	Skewness	K-S Test	Sig. p
<u>Migraine</u>				
Dur.1*	6.43	2.50	1.4992	.0223
Dur.2*	11.83	3.27	1.5293	.0186
Fre.1*	6.15	2.51	1.5174	.0200
Fre.2*	4.67	2.28	1.5761	.0139
Sev.1	-.70	-.17	.9256	.3584
Sev.2	-.89	-.25	.5303	.9412
<u>Aura</u>				
Dur.1	2.37	1.42	.6107	.8499
Dur.2*	12.13	3.45	1.3776	.0449
Fre.1	5.89	2.10	.8444	.4738
Fre.2	11.25	3.28	1.2074	.1083
Sev.1	-1.30	-.25	.5403	.9322
Sev.2	1.00	1.32	.7094	.5451
<u>Depr.</u>				
Dep.1	-.55	-.66	.8067	.5333
Dep.2	-1.13	.17	.6934	.7222
<u>Q.o.L.</u>				
Qua.1	-.62	.03	.5625	.9098
Qua.2	.36	-.67	1.0209	.2483

Abbreviations

Dur = duration, Fre = frequency, Sev = severity, Depr = depression, Q.o.L = quality of life. Numbers 1 and 2 denote phases 1 and 2. \* denotes positively skewed variables. There were no negatively skewed variables.

Table 2

Results of Logarithmic Transformations showing normality has been achieved for Positively Skewed Variables

Variable	Kurtosis	Skewness	K-S Test	sig. p
<u>Migraine</u>				
Dur.1	1.88	.806	.7099	.6947
Dur.2	.970	-.218	.6143	.8447
<u>Frequency</u>				
Freq.1	.623	.987	.7937	.5544
Freq.2	.447	.093	.6688	.7624
<u>Aura</u>				
Dur.1**	-.689	-.399	.4743	.9781
Dur.2	1.557	1.083	.6122	.8477

\*\* Aura Dur. 1 was not significantly skewed, but it is statistically appropriate to transform this variable so it could be tested with aura Dur. 2 which had to be transformed (Aron & Aron, 1994).

Abbreviations Dur = duration, Fre = frequency.

#### 4.32 Demographic Data

Participants comprised clinically diagnosed migraine patients drawn from the population at large. All 32 volunteers who met the selection criteria (see Appendix C) were accepted. Seven of these (21.8%), were lost through attrition. Of the 25 participants who completed the study, 6 were males and 19 were females. Their ages ranged from 36 years to 68 years, the mean age was 51 years, and the median age 49 years. The migraines suffered by all participants were chronic and had become established by early adulthood.

Participants provided information which showed that 15 (60%), suffered bilateral migraines, 7 (28%) suffered unilateral migraines involving only the left hemisphere of their brain, and 3 (12%) suffered unilateral migraines involving only the right hemisphere.

This indicates that from the combined total of bilateral and unilateral migraines, the left hemisphere of the brain was involved in 88% of migraines, and the right hemisphere in only 72% of all attacks. Thirteen (52%) of the 25 participants experienced aura, that is, classic as opposed to common migraine (see section 4.34). Thirteen participants also suffered comorbid somatoform disorders (see section 4.35.4). No significant correlation was found between aura and comorbid somatoform disorders.

#### 4.33 Duration, Frequency, and Severity of Migraine

Dependent two-tailed t-tests were employed after Kolmogorov-Smirnov and logarithmic adjustments to test each of the three null hypotheses relating to migraine, with time-series graphing used to validate consistent change over time. Significant pre/post treatment effects were found for all migraine hypotheses analysed. Measures of duration and frequency are representative of group totals, whereas the measure of severity represents the group mean. Observations pertaining to means and standard deviations are presented in Table 3. The progressive reductions obtained the duration, frequency, and severity of migraine are presented in Figures 2, 3, and 4 respectively.

##### Hypothesis 1

There will be no significant pre/post treatment difference in the duration of migraine. Results showed post-treatment duration was significantly shorter than pre-treatment duration,  $t(24) = 5.74, p < 0.0005$ . The first null hypothesis was thus rejected.

##### Hypothesis 2

There will be no significant pre/post treatment difference in the frequency of migraine. Results showed a post-treatment frequency was significantly lower than pre-

treatment frequency,  $t(24) = 3.86$ ,  $p < 0.001$ . The second null hypothesis was also rejected.

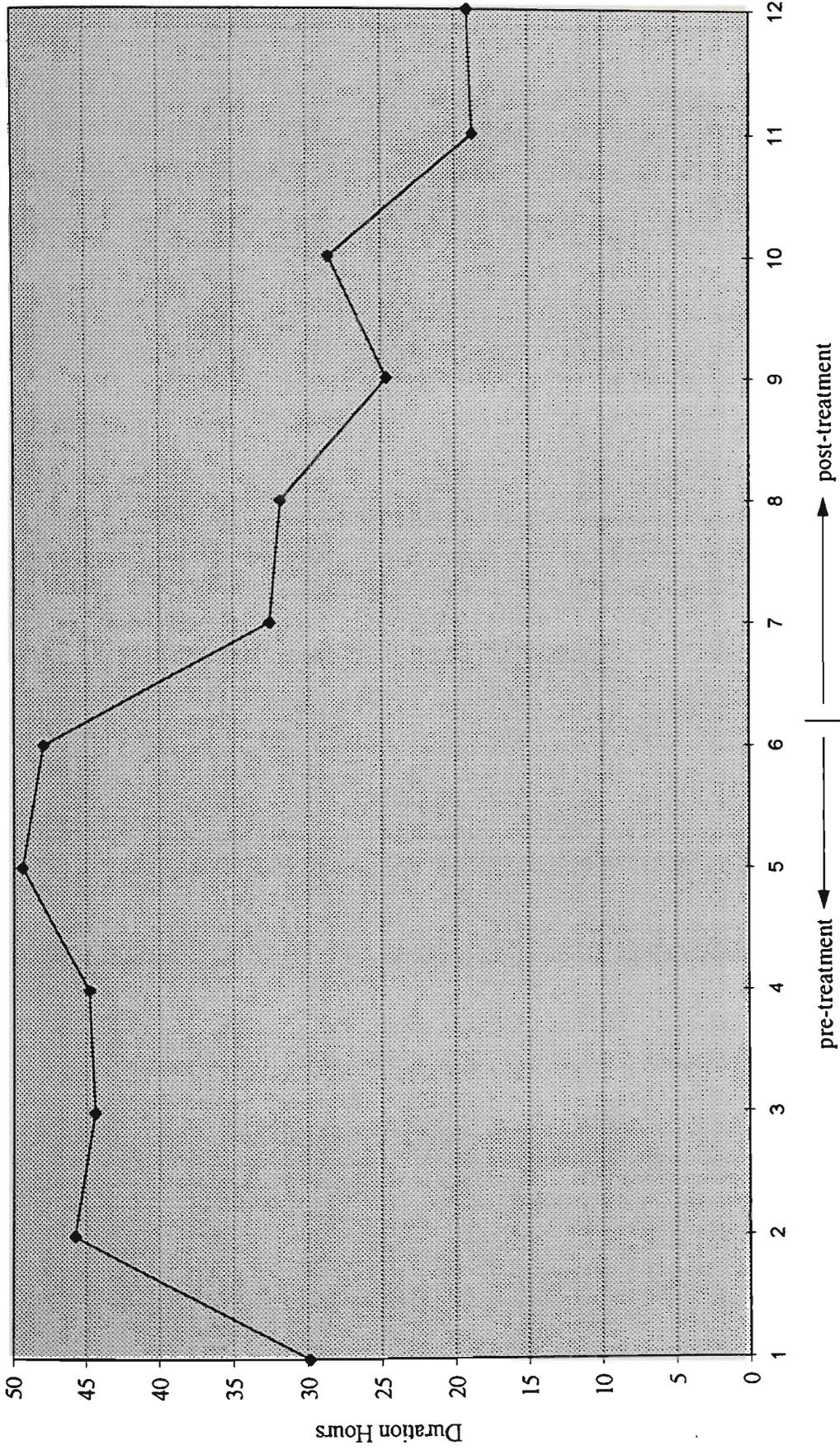
### Hypothesis 3

There will be no significant pre/post treatment difference in the severity of migraine. Results showed post-treatment severity was significantly less than pre-treatment severity,  $t(24) = 5.71$ ,  $p < 0.0005$ . The third null hypothesis was therefore also rejected.

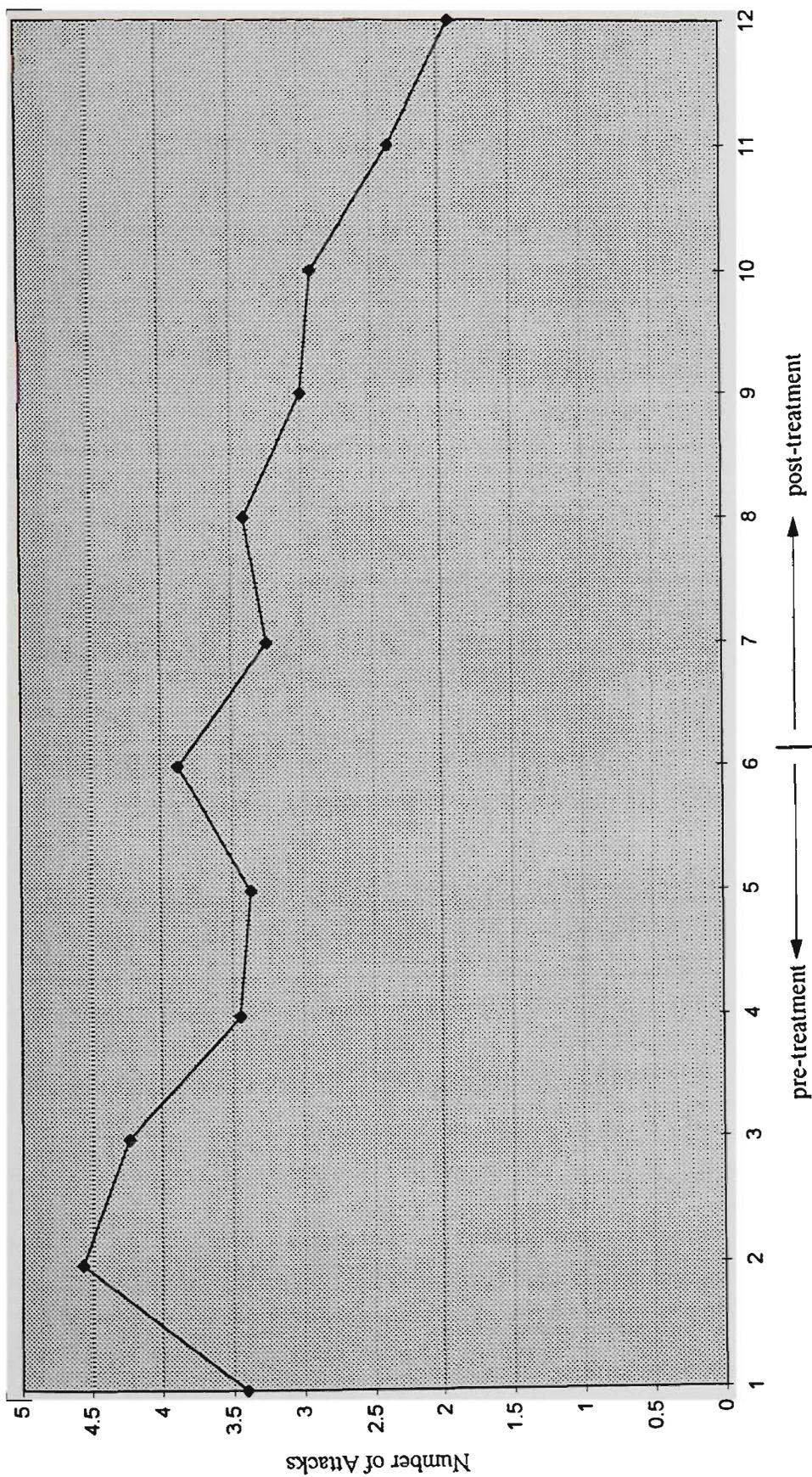
Table 3

### Migraine Means and Standard Deviations Phases 1 (pre-treatment) and 2 (post-treatment) (N=25)

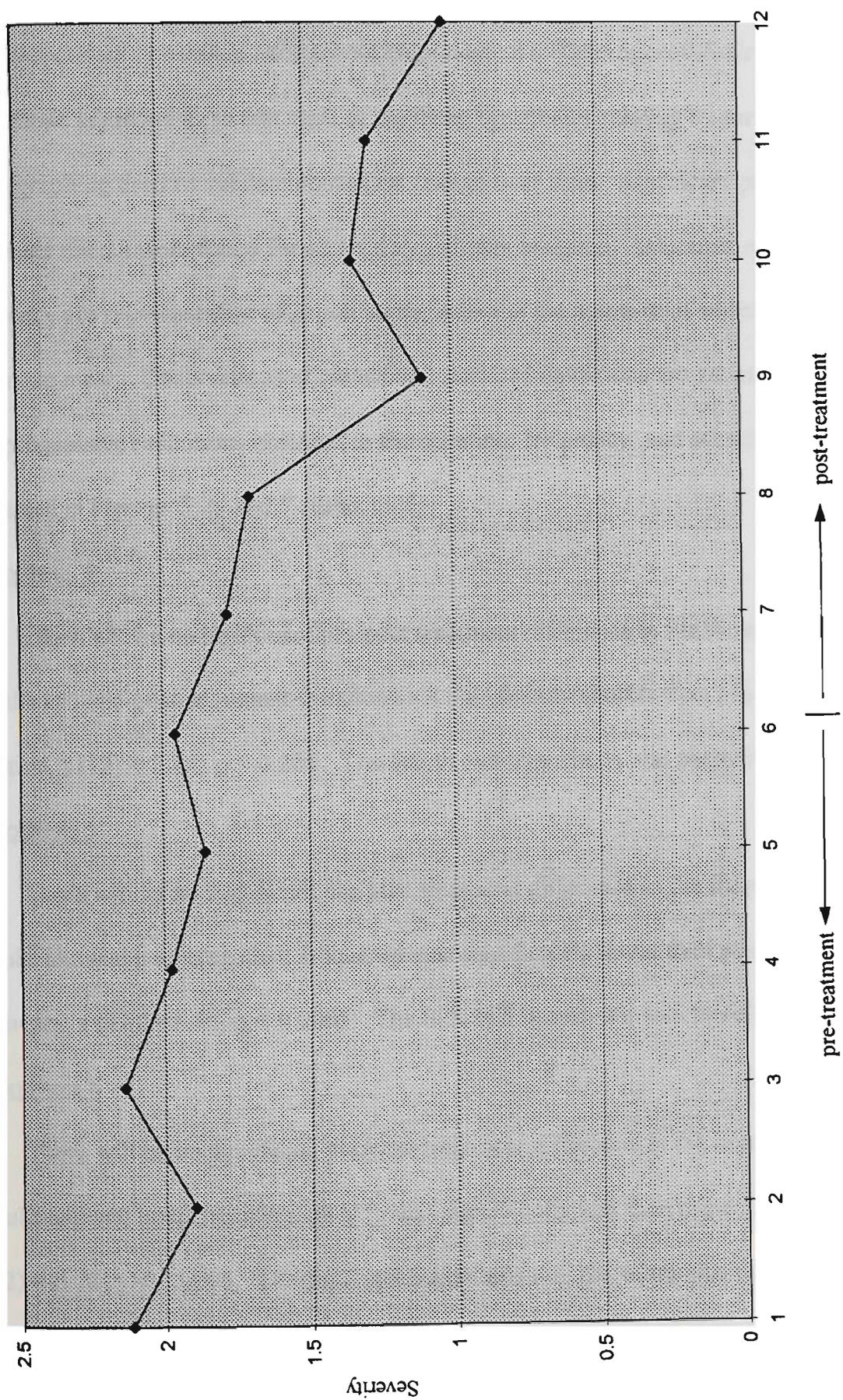
Variable	Mean	Std. Deviation
Duration 1	54.0067	68.3927
Duration 2	25.9267	40.3896
Frequency 1	3.8133	4.6486
Frequency 2	2.8067	3.4088
Severity 1	1.9933	.5193
Severity 2	1.3700	.5824



**Figure 2.** Migraine duration  
Group means of number of migraine hours per consecutive fortnight (N=25)



**Figure 3.** Migraine frequency  
Group means of number of migraine attacks per consecutive fortnight (N=25)



**Figure 4.** Migraine severity  
Group means per consecutive fortnight (N=25)  
Self-rated on a scale of 1-3

#### 4.34 Duration, Frequency, and Severity of Aura

Dependent two-tailed t-tests were employed after Kolmogorov-Smirnov and logarithmic adjustments to test each of the three hypotheses relating to aura, with time-series graphing used to validate consistent change over time. Significant pre/post treatment effects were found for all aura hypotheses analysed. Measures of duration and frequency are representative of group totals, whereas the measure of severity represents the group mean. Observations of means and standard deviations are set out in Table 4. The progressive reductions obtained in the duration, frequency, and severity of aura, are presented in Figures 5, 6, and 7 respectively.

##### Hypothesis 4

There will be no significant pre/post treatment difference in the duration of aura. Results showed post-treatment duration was significantly shorter than pre-treatment duration,  $t(12) = 3.66$ ,  $p < 0.008$ . The fourth null hypothesis was therefore rejected.

##### Hypothesis 5

There will be no significant pre/post treatment difference in the frequency of aura. Results showed post/treatment frequency was significantly lower than post-treatment frequency,  $t(12) = 5.35$ ,  $p < 0.0005$ . The fifth null hypothesis was therefore rejected.

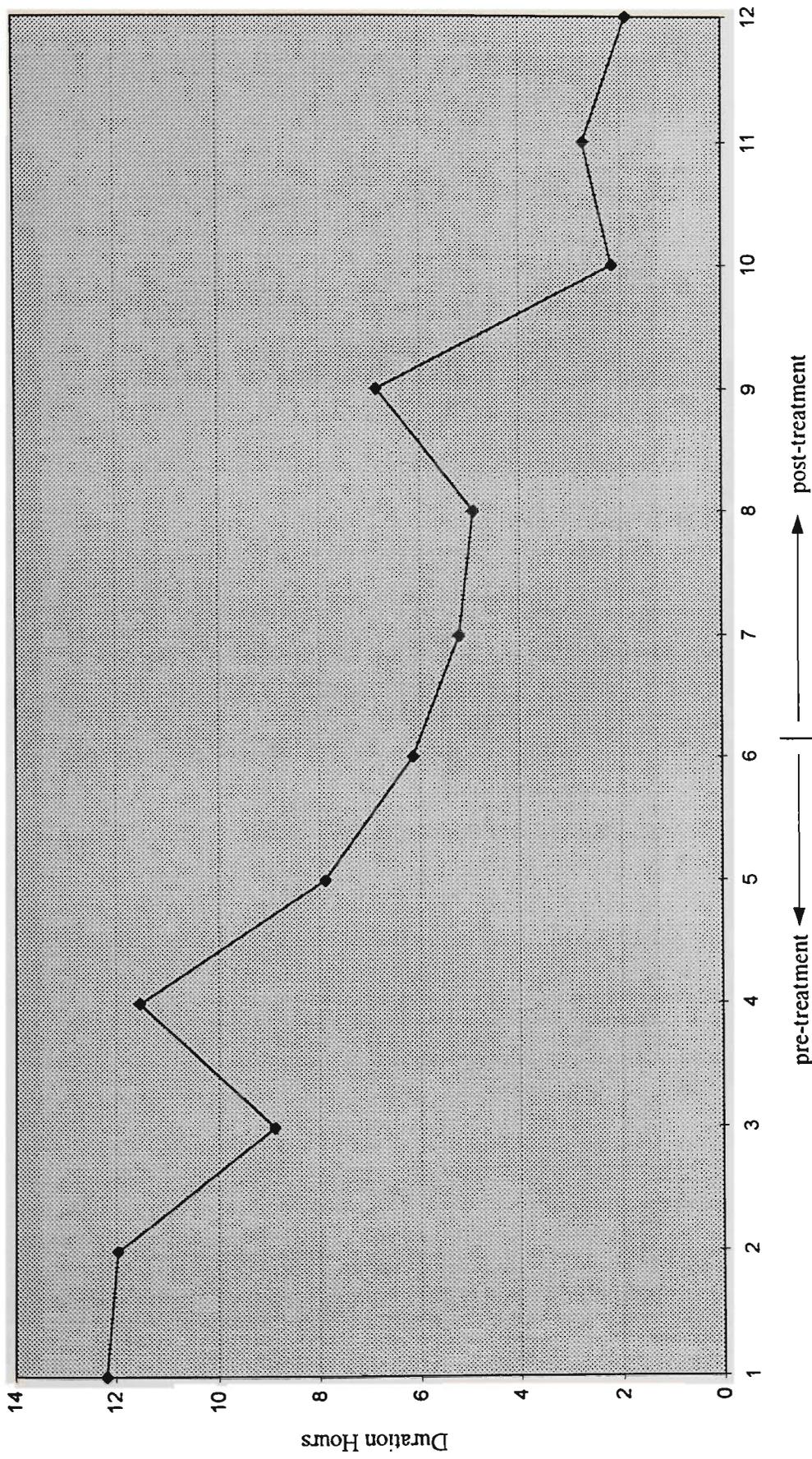
##### Hypothesis 6

There will be no significant pre/post treatment difference in the severity of aura. Results showed post/treatment severity was significantly less than pre-treatment severity,  $t(12) = 6.78$ ,  $p < 0.0005$ . The sixth null hypothesis was therefore also rejected.

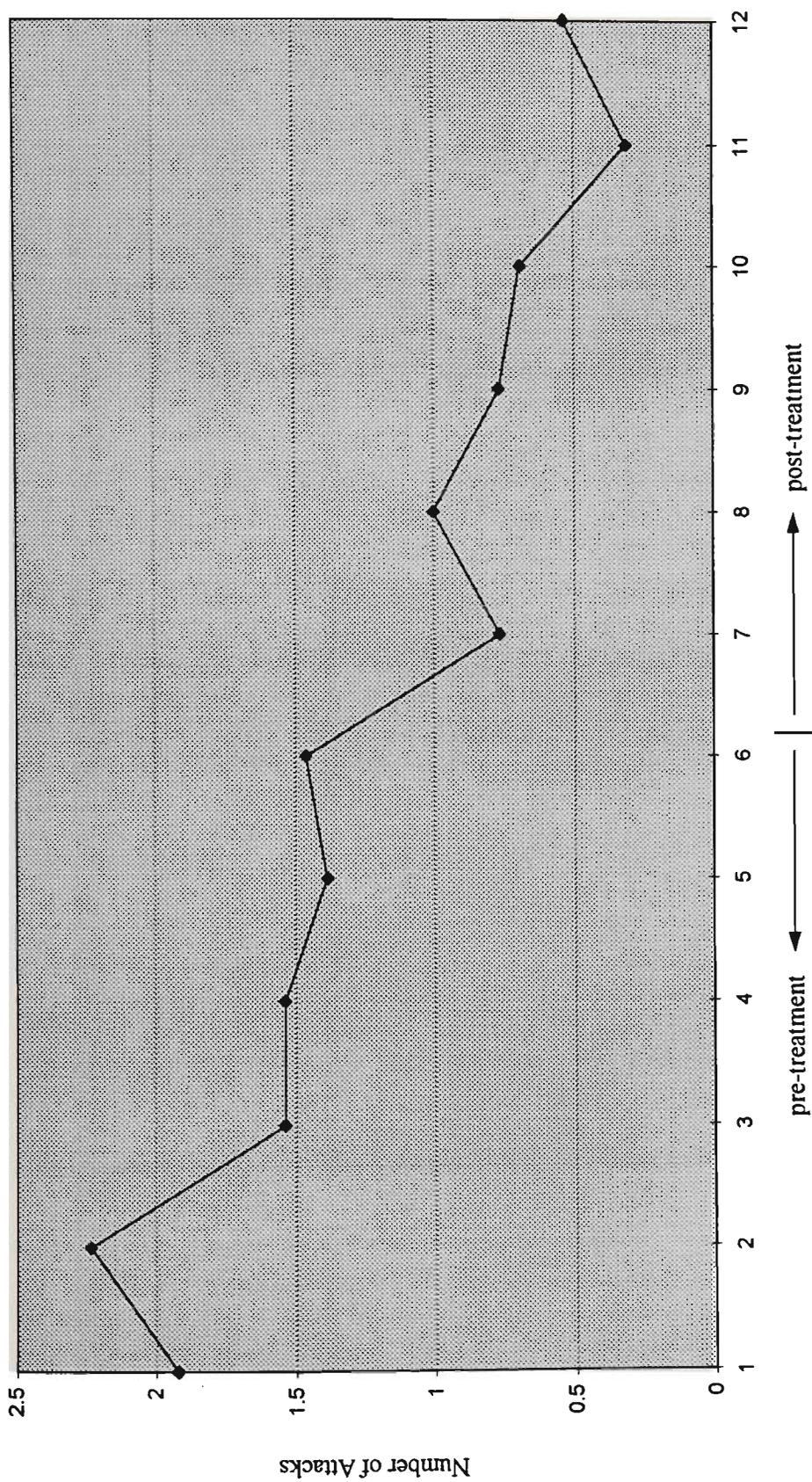
Table 4

Aura Means and Standard Deviations Phases 1 (pre-treatment) and 2 (post-treatment)  
(n=13)

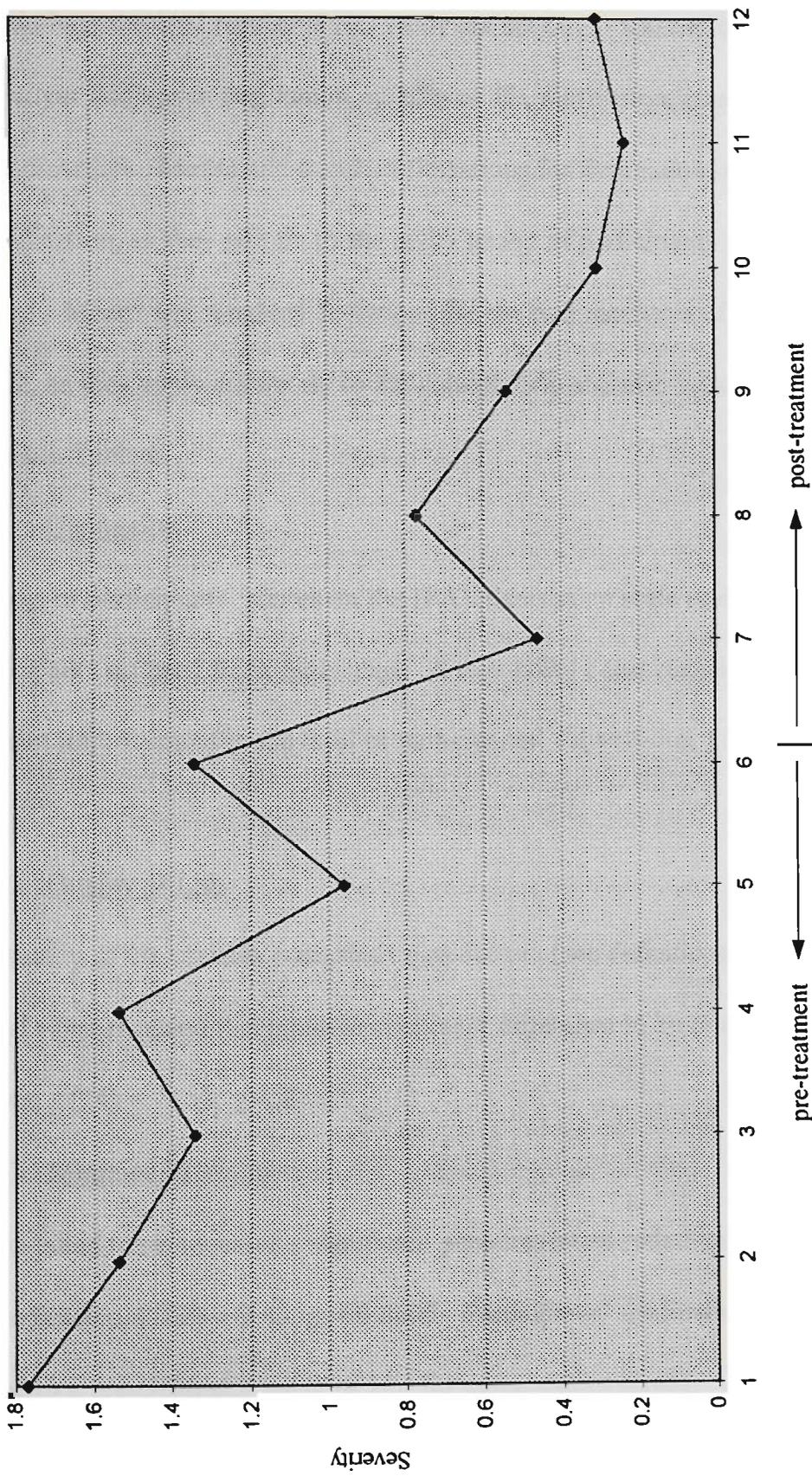
Variable	Mean	Std. Deviation
Duration 1	9.7692	9.0228
Duration 2	3.9647	10.2372
Frequency 1	1.6795	1.3272
Frequency 2	.6795	1.4377
Severity 1	1.4167	.5813
Severity 2	.4359	.5370



**Figure 5.** Aura duration  
Group means of migraine hours per consecutive fortnight (N=13)



**Figure 6.** Aura frequency  
Group means of number of migraine attacks per consecutive fortnight (N=13)



**Figure 7.** Aura severity  
Group means per consecutive fortnight (N=13)  
Self-rated on a scale of 1-3

#### 4.35 Non-hypothesised Results

The non-hypothesised results of this study are set out in subsections 4.35.1 to 4.35.5, and cover findings in depression, quality of life, motivation, stress, and comorbid somatoform disorders. Means and standard deviations for depression, quality of life, and migraine medication, as they pertain to the entire group of participants ( $N=25$ ), are set out in Table 5. Means and standard deviations for the two measures of depression, and quality of life, as they apply separately to the somatoform and non-somatoform groups, are presented in Table 6.

##### 4.35.1 Depression

Analysis of participants' scores on the IPAT depression scale revealed that 22 of the 25 participants suffered from depression. A two-tailed t-test revealed a significant post-treatment improvement in the level of participants' depressions,  $t(24) = 2.22$ ,  $p = 0.036$ .

##### 4.35.2 Quality of Life

The quality of life variable comprised nine factors (see definitions in Appendix G.). A two-tailed t-test revealed this pre/post-treatment difference to be one of significant improvement,  $t(24) = -3.53$ ,  $p < 0.002$ .

##### 4.35.3 Migraine Medication

A two-tailed t-test revealed a significant post-treatment reduction of participants' migraine medication,  $t(12) = 9.43$ ,  $p < 0.0005$ . Participants' medication levels were standardised to a pre-treatment score of 100 for comparative clarity.

Table 5

Means and Standard Deviations for Depression, Quality of Life, and Migraine Medication for Phases 1 (pre-treatment) and 2 (post-treatment) (N25)

Variable	Mean	Std. Deviation
Depress. C 1	6.6800	2.2494
Depress. C 2	5.8800	2.2605
Qual. Life 1	3.2308	.5166
Qual. Life 2	3.4733	.4956
Migr. Med. 1	100.0000	.0000
Migr. Med. 2	51.5385	18.5280

Abbreviations

Depress. C = depression score corrected to accommodate anxiety components.

4.35.4 Motivation and Stress

Neither motivation nor stress proved to be significant factors when correlated with dependent measures. Any significant differences below 0.05 were lost with Bonferroni adjustments.

4.35.5 Comorbid Somatoform Disorders

Fifty two percent of participants ( $n=13$ ) were found to suffer from comorbid somatoform disorder. Factors which lead to the analysis of this variable are discussed in section 5.14. Two independent t-tests were employed to compare quality of life, and depression results obtained from the somatoform group with those obtained from the non-somatoform group.

Although there was no significant difference between these two groups in their quality of life at pre-treatment, the analysis of post-treatment results showed the quality

of life of somatoform group was significantly lower than that of the non-somatoform group  $t(23) = 2.41, p = 0.024$ . The pre-treatment depression level of the somatoform group was significantly higher than that of the non-somatoform group,  $t(23) = -3.76, p = 0.001$ , and also significantly higher at post-treatment  $t(23) = 4.70, p = 0.0005$ . The means and standard deviations pertaining to these measures are set out in Table 6.

Table 6

Somatoform Group (n=13) and Non-somatoform Group (n=12): Means and Standard Deviations For Depression and Quality of Life for Phases 1 (pre-treatment) and 2 (post-treatment)

Variable	Mean	Std. Dev.	Sig. p
Depress. C 1			
Somatoform	2.8205	.220	.001
Non-somatoform	2.2388	.509	
Depress. C 2			
Somatoform	2.7006	.314	.0005
Non-somatoform	2.0254	.402	
Qual. Life 1			
Somatoform	1.7403	.133	.062
Non-somatoform	1.8477	.141	
Qual. Life 2			
Somatoform	1.8009	.155	.024
Non-somatoform	1.9216	.082	

#### Abbreviations

Depress. C = depression score corrected to accommodate anxiety components. 1 and 2 = phases 1 and 2

## CHAPTER 5

### Discussion

#### 5.1 Summary

According to DSM-IV diagnostic criteria, migraine can be seen to qualify for the classification of either a 'Disorder of Psychological Factors Affecting a Medical Condition' (p. 675), or a 'Pain Disorder Association with both Psychological Factors and a General Medical Condition. It should be noted that the psychological factors of a pain disorder may be of a general nature, such as a reaction to psychosocial stressors (p. 458). Given the frequent association of stress with migraine, (Catalano & Hardin, 1996), it is likely that psychological factors play a major role, in the antecedent conditions of migraine.

Acceptance of this view leads to the further conclusion, that vascular change which accompanies migraine pain and aura scotoma, is only one of several necessary pathogenic, but not sufficient conditions in the causal chain of migraine and aura. The same can be said of other neurophysiological changes or biochemical factors which accompany migraine and aura.

Literature indicates that medical scientists researching causal, or aetiological, factors of migraine, have confined their inquiries to the pathogenesis of migraine. Despite many years of research into this area the pathogenesis of migraine is still not fully understood, and in many ways, remains open to speculation (Lance, 1993). This stalemate is, at least in part, ascribable to the absence of sustained inquiry into the psychological conditions preceding migraine.

Just as the expertise of medical scientists does not commonly extend into psychological inquiry, so the scientific expertise of psychologists does not usually extend into medical inquiry. It follows, that research aimed to determine the cause of migraine, in which both psychological and physiological conditions are implicated, could benefit from a co-operative effort, involving researchers from both sciences. Such joint application of expertise and pooling of research equipment, including electronic scanning devices, can also facilitate a monitoring of vascular and other physiological changes in response to hypnotic suggestions for pain alleviation. In doing so, light may well be shed on the question of how hypnosis achieves its analgesic effectiveness.

Despite the aetiological uncertainties surrounding migraine and aura, it is generally accepted that migraine is a disorder of function, and not one of organic disease (Sachs, 1985). It is a heterogeneous, psychologically influenced and significantly debilitating chronic disorder suffered by approximately 12% of the Australian population during their potentially most productive years. Of the 25 participants who completed this study, 13 (52%) also suffered the debilitating neurological symptoms of aura.

Motivated to address the absence of research into the efficacy of hypnotherapy in the treatment of aura symptoms, the treatment program adopted in this study was designed to comprise two distinct treatment streams, one was aimed to reduce migraine pain, while the other was focused on reducing aura symptoms.

Research into the efficacy of pharmacotherapy has shown that neither migraine nor aura is consistently responsive to drug treatment (Australian Brain Foundation, 1993; Herzberg, 1994; Plosker and Mc Tavish, 1994). The four-modality treatment employed in this study, (three modalities for the treatment of aura, and three modalities for the

treatment of migraine), have produced statistically significant treatment responses in relation to the duration, frequency, and severity of both migraine and aura. These results have demonstrated the effectiveness of hypnosis in the treatment of both migraine and aura.

The range of these positive treatment responses is further augmented by the significant post-treatment improvements achieved in relation to depression, medication, and quality of life. Significant differences were also found between the comorbid somatoform group and the non-somatoform group. These differences pertained to quality of life, and depression.

The experiments conducted in this study, and their ensuing results (reported in Chapter 4) enable a range of important conclusions to be made. These are grouped into three major areas of relevance. The first pertains to the responsiveness of migraine and aura to the treatment of hypnotherapy in terms of duration, frequency, and severity. The second pertains to treatment-related benefits in terms of depression, medication, and quality of life. And the third deals with the prevalence and potential impact of comorbid somatoform disorders. The conclusions are discussed under their respective headings in the course of this chapter.

Two issues merit special consideration. These pertain to the question of intervention through extraneous influences, and the capacity of each constituent part of the two treatment streams (the four-treatment modality) to contribute to the effectiveness of the treatment as a whole. These issues are considered in section 5.15 and in sections 5.16. through 5.16.4 respectively.

It is also pointed out, that the results of this study are not meant to imply that the effect achieved, is only achievable with the treatment approach adopted in this study, or that the hypnotic induction of relaxation is necessarily more effective than other relaxation procedures. Comparisons of this nature did not form part of this study, and therefore, no such comparative claims are made.

The further qualification is given that the results achieved for both aura and migraine, cannot, with absolute certainty, be attributed to the treatment given. As ever, statistical analysis in the field of empirical science remains a matter of probability. In this study, the high level of statistical significance achieved, shows that the highest probability of error in aura results is less than 0.008, and that for migraine is 0.001.

#### 5.11 The Treatment Responsiveness of Migraine

The first null hypothesis concerned the treatment response of migraine in terms of duration. Duration is of particular importance from the point of view of the migraine patient, since duration, more so than the frequency of migraine, represents the actual time spent in migraine pain, and in the debilitating neurological spectra of aura. The mean total duration of migraine per participant for the 12-week post-treatment phase, was 155.54 hours, compared with 260.28 hours for the pre-treatment phase. This reduction of 104.74 hours (40.25%) at the conclusion of treatment, is statistically significant,  $p < 0.0005$ .

A factor of interest regarding the results achieved for duration, is the connection between duration of migraine and use of analgesic medication. Sachs, (1985) claims that the duration of migraine becomes prolonged by using analgesic drugs, and that patients

have the option to choose reduced pain for a longer period by taking medication, or to endure without medication, the full severity of pain for its shorter, natural duration.

Participants were able, in the course of the treatment phase, to decrease the dosage of their usual migraine medication. As such, and in keeping with Professor Sachs' view on prolonged medicated pain, it could be the case, that the achieved decrease in medication, also contributed to the decrease in the duration of their migraine. To avoid the possibility of subconscious compliance responses, participants were not told of this connection. However, as no controlling or testing mechanisms for this factor were used in this study, the possibility of such a contribution having occurred, remains a possibility. This point is further discussed in section 5.13.

The treatment responses of migraine in terms of frequency and severity, were also significantly reduced. The group's frequency mean of 22.88 migraine attacks for the 12-week pre-treatment phase, was reduced to 16.8 attacks at post-treatment ( $p = 0.001$ ), while severity was reduced from its pre-treatment mean score of 1.98, to a mean of 1.35 at the end of the treatment period ( $p = 0.0005$ ). Figures 2, 3, and 4 depict the progressive reductions of duration, frequency, and severity of migraine respectively.

These treatment responses by participants clearly demonstrate the effectiveness of hypnosis in the treatment of migraine. The reduction in frequency, in particular, demonstrates the capacity of hypnotic treatment to prevent the onset of migraine symptoms. Research has shown, that with the exception of continuous daily medication, only psychologically based treatments, such as hypnosis, appear to have a particularly strong capacity for reducing the frequency of migraine, as well as the duration and severity (Harding, 1978; Milne, 1983; Schaefer et al. 1979). The results of this study

confirm the effectiveness of self-empowering psychological strategies such as self-hypnosis, in the treatment of migraine. They also demonstrate the efficacy of this approach in the treatment of aura.

Further, results achieved in the reductions of duration and severity attest to the efficacy of hypnosis to inhibit sensitivity to pain. This raises the question of what actually occurs in the course of this inhibitory process, how does hypnosis reduce the severity and duration of migraine pain? For example, are these reductions accomplished by facilitating actual neuro-vascular or bio-chemical changes, such as a return of abnormally dilated blood vessels to their normal state, or are they simply due to an inhibited awareness of the 'pain in progress,' as suggested by Hilgard and Hilgard (1975)?

As research methods available to this study precluded the possibility of testing vascular responses by means of positron emission or other electronic imaging techniques, the answer to this important and long standing question continues to await future research. However, considering that major operations, such as caesarean sections, are performed with hypnotic analgesia only, it is likely that hypnotic pain control goes beyond merely inhibiting or changing the perception of pain. It may be, that in these deeper levels of hypnotic pain control, there is no hidden 'pain in progress' (Hilgard, 1975) because the pain relay mechanisms have temporarily ceased relaying pain impulses to the cortex.

The reality of hypnotically reduced physiological sensitivity to pain (as opposed to mere psychological perception of pain) has been demonstrated by Prior, Colgan, and Whorwell (1990). These researchers employed objective, physiological measures to

conduct a study with 15 irritable-bowel-syndrome patients. The physiological measures showed that hypnosis reduced rectal sensitivity in these patients.

The levels of hypnosis reported by participants in the present study showed that the results for migraine and for aura, were generally achieved in only a light state of hypnosis. Although six participants reported to have attained medium levels on several occasions, only two participants had been able to reach deep levels, and this occurred only on rare occasions. This outcome supports findings that for clinical purposes, a light to medium level of hypnosis is generally adequate for successful treatment (Rose, 1990; Stanley, 1993).

#### 5.12 The Treatment Responsiveness of Aura

The results discussed in relation to aura represent the treatment responsiveness of the 13 participants who suffered aura symptoms. This constitutes 52% of the migraine sample of this study, and exceeds the 20% estimate of the Australian Brain Foundation (1993).

A projection of figures derived from the Russell and Olesen (1996) nosographic (non-treatment related) analysis of aura, supports the higher aura prevalence found in this study. The researchers found that 232 people out of their 'general population' sample of 4,000 people, suffered auras. Although the actual migraine population within their general population sample was not identified, it may be estimated on the basis of the migraine prevalence estimate of 12% (see section 1.14). Application of this ratio to a general population of 4,000 shows the likely migraine population of their general population sample to have been 480. The 232 aura sufferers in the Russell and Olesen (1996) analysis, may thus be taken to represent a prevalence rate of 48.3%, which is in

keeping with the prevalence of 52% found in the migraine population of this study. A general prevalence rate in excess of 20% is also suggested by figures presented in the Migraine Foundation of Australia Report (1997). This Report states, that 64% of migraine patients surveyed, reported symptoms of visual disturbances as part of their migraine.

As in the case of migraine, the results achieved in the treatment of aura, clearly demonstrate the efficacy of hypnosis. The progressive reductions achieved in the measures of duration ( $p < 0.008$ ), frequency ( $p < 0.0005$ ), and severity ( $p < 0.0005$ ) of aura, are depicted in Figures 5, 6, and 7, respectively.

Unlike migraine however, the vascular system involved in producing aura symptoms, is located in the midbrain and involves vascular constriction. Like muscular tension in other parts of the body, constricted blood vessels respond well to the counter measure of hypnotic relaxation. This treatment modality was practiced independently on a daily basis, as well as in conjunction with both vascular manipulation treatments. It is therefore reasonable to infer that these multiple relaxation procedures played a prominent role in contributing to the significance of the results achieved.

The fourth null hypothesis in this study concerned the duration of aura. The mean aura duration total of 56.30 hours per aura participant for the 12-week pre-treatment phase, was significantly reduced to a mean of 23.78 hours at the conclusion of treatment. The treatment responses in frequency and severity, as expressed by null hypotheses 5 and 6, also showed statistically significant reductions. The pre-treatment frequency mean of 10 aura attacks per aura participant, was reduced to 4 attacks at the conclusion of

treatment, and severity was reduced from a pre-treatment group mean of 1.93 (on a scale from 1 to 3) to a post-treatment mean of 0.95.

Seven of the 13 aura sufferers, achieved complete remission by the end of the treatment period. Five others achieved reductions in duration, and frequency, ranging between 50% and 94%. Only one participant failed to achieve a significant improvement in relation to aura. This participant suffered from comorbid somatoform disorders (this topic is discussed in section 5.14), and achieved a reduction of only 15% in aura severity.

The results achieved by 12 of the 13 aura participants were impressive in relation to all three aura variables. The best reduction was achieved in frequency (59.59%), followed by duration (57.77%), and severity (50.78%). This effectively means that for every 10 pre-treatment auras, participants at post-treatment experienced fewer than 5 attacks. From a clinical perspective, these reductions represent a combined post-treatment benefit which reduces 10 hours of debilitating aura symptoms to only 4.2 hours, at half the former severity.

The question which presents itself now is whether the reduction or elimination of an aura attack can, or is likely to, prevent the associated onset of a migraine attack. If so, there is the further question of how such prevention can be assessed and quantified. In cases where total elimination (remission) of aura has been achieved, there is no meaningful way of assessing the degree to which migraines have been prevented as a consequence of the elimination of aura. It seems probable, however, that the elimination of preconditions for migraine, such as auras, plays a contributory role in its prevention, and in the reduction of its frequency.

Further, if it is accepted that aura manifests most commonly in association with migraine symptoms, then by extension, treated auras with fewer subsequent migraines, could be taken to imply that their treatment has had a migraine preventative affect. The rate of aura without headache has been shown to be low (Lance, 1993). This was further substantiated by Russell and Olesen (1996) in their nosographic analysis of aura. The researchers found that migraine followed aura in 93% of cases, they occurred simultaneously in 4% of cases, and migraine preceded aura in only 3% of cases. Only 3% of aura patients suffered exclusively from aura without migraine.

Similarly, during the pre-treatment phase of this study, only 1 of the 13 aura sufferers reported 2 auras without subsequent migraine. During the post-treatment phase however, 6 (46%) of the 13 aura sufferers reported a total of 9 auras without subsequent migraines. This shows that, of the significantly reduced number of post-treatment auras, the number of auras which did not lead to migraines was increased by 78.78%. On this basis it seems reasonable to conclude, that some migraines were prevented through the use of hypnotic treatment for aura. Whether, or not, the administered migraine treatment, could have confounded these 'preventions' is not clear. It is therefore desirable, in the future, to test the migraine preventative capacity of aura treatment independently.

Statistical verification of such preventative capacity is achievable by employing a two-phase time series design, administering only aura treatment, and recording the measures of duration, frequency, and severity of both auras and migraines suffered before and after aura treatment. Subsequent statistical analyses should reveal the extent

of changes, if any, in migraine and aura measures, and if hypnotic aura treatment can by itself, prevent the onset of migraines.

### 5.13 Treatment Related Benefits

#### 5.13.1 Medication

The significant post-treatment reduction achieved in migraine medication further demonstrates the effectiveness of the integrated four-modality treatment. Participants' medication prior to treatment was standardised to a value of 100, thereby allowing participants' post-treatment score to reveal the residual medication in use. Fourteen of the 25 participants who completed the study, reduced the quantity of their migraine medication by more than 50%, and 1 participant no longer used any migraine medication at all.

#### 5.13.2 Depression

Migraine sufferers are four times more likely to suffer depression and are twice as likely to suffer from anxiety disorders (Australian Brain Foundation, 1993). This view is supported by the finding of this study, which showed that 22 of the 25 participants who completed the study suffered from depression, as measured on the IPAT depression scale. Further, it is generally recognised that depression and anxiety are part of the migraine profile (Lance, 1993; Sachs, 1985). Krug and Laughlan (1976) found a strong correlation of ( $r = .80$ ) between depression and anxiety. Rose (1990) stated that 50% of all depressed patients suffer from a chronic pain disorder, including that of migraine and other headaches.

These factors indicated that the use of the full scale corrected score of the IPAT depression scale was appropriate for this study, to accommodate anxiety factors

commonly associated with depression. The post-treatment results, which showed a significant reduction ( $p < 0.0005$ ) in participants' depression levels, lends support to the view expressed by Spiegel (1965), that the practice of hypnotic relaxation regulates the production of serotonin, and contributes to an alleviation of depression. Abnormally low levels of serotonin are known to be involved in depression, low pain control, and vasodilatation, whereas abnormally high levels are associated with vasoconstriction, such as is experienced in aura and other migraine prodromes (Lance, 1993; Rose & Gawel, 1981).

The post-treatment improvement achieved in relation to depression, may also be related to participants' increased sense of control over their own migraine pain, and increased self esteem commonly associated with the exercise of such control (Elton et al. 1993). If left untreated, depression and migraine can establish a vicious circle. Patients can become depressed at their perceived lack of control over migraine pain (and other pain), and experience migraines through awareness of their depression (Lewis, 1988).

### 5.13.3 Quality of Life

The quality of life variable in this study comprised nine factors commonly associated with physical and emotional well being. Physical factors included energy levels and general health; emotional factors included sense of control, future outlook, self-esteem, achievement, and relationships (details and definitions are presented in Appendix G).

Although the impact of migraine on quality of life factors has not been extensively documented, it is generally accepted that "Migraine has a unique, significant quality of life burden" (Osterhaus et al. 1994, p. 337). In contrast to many other chronic

conditions, migraine has a comparatively acute physical manifestation. Its debilitating effects in general, have been shown to have a strongly detrimental impact on patients' physical and social functioning, as well as on their mental health.

The functionality status of migraine patients in these three categories, has been found to be worse than that of patients with other chronic conditions (Solomon et al. 1993). While these documented debilities of migraine sufferers demonstrate the urgent need for more effective migraine and aura treatments, the significant post-treatment improvement achieved by participants in this measure ( $p < 0.002$ ) demonstrate the effectiveness of hypnosis in this treatment role.

#### 5.14 Prevalence and Impact of Comorbid Somatoform Disorders

A comparison of raw scores in the course of the treatment phase, revealed that a number of participants were consistently less responsive to treatment in two measures. These were the measures of depression and quality of life. The discovery of this trend prompted consideration of the possibility of some mutually shared, intervening condition which reduced the treatment responsiveness of these participants as a whole.

Although subsequent statistical analysis showed that the group as a whole ( $N=25$ ), had achieved significant improvements in all measures, the consistently lower treatment responses of the above mentioned participants ( $n=13$ ), nevertheless warranted a closer investigation. Clinical considerations, based on the psychosomatic nature of migraine and the commonly associated concept of secondary gains, suggested that this group's lower responsiveness in these measures, may be related to a form of treatment resistance.

Literature on the topic of secondary gains abounds with enumerated psychological 'advantages' patients derive from clinging to pain, by resisting cures (Adler, 1927;

Horney, 1950; Reik, 1941). More recently, authors speak of the 'escape into pain' syndrome as being simply one of several maladaptive coping mechanisms adopted by individuals in times of stress (Elton et al. 1993; Montgomery & Evans, 1987; Rapoport & Sheftell, 1991; Sachs, 1985). However, as these considerations may be said to apply to all individuals suffering from psychosomatic disorders, or to migraine patients in general, the further question arose of why one group of participating migraine patients should be more treatment resistant than the other?

One answer presented itself in the form of factors which compound or increase the need for psychological coping mechanisms. This may understandably occur under conditions of coexisting psychosomatic or somatoform disorders. In order to determine whether the 'less responsive' participants suffered from such comorbid disorders, the 'General Health' questionnaire was administered to all participants at the conclusion of the treatment phase. This questionnaire, which was especially compiled for this purpose, comprised a composite of DSM-IV (1994) diagnostic criteria for somatoform disorders (see Appendix K).

The questionnaire results showed that 13 (52%) of the 25 participants met the DSM-IV diagnostic criteria for somatic disorder. Subsequent statistical comparisons revealed the group of participants suffering from comorbid somatoform disorders, to have been the 'less responsive' group. For example, although the quality of life for these two groups was not significantly different prior to treatment, the treatment responsiveness (improvement) of the comorbid somatoform group was significantly lower than that of the non-somatoform group,  $p = 0.024$ . This significantly lower responsiveness to treatment, may be taken to constitute a form of treatment resistance, a

reluctance to surrender a tried coping mechanism, albeit in the form of a maladaptive 'escape into pain'.

A comparison of IPAT depression scores revealed, that participants with coexisting somatoform disorders were significantly more depressed at pre-treatment ( $p = 0.001$ ), and at post-treatment testing ( $p = 0.0005$ ). The score range on the IPAT depression scale is 1 to 10, with 10 representing the most severe depression level. Fifteen (60%) of the 25 participants who completed the study, had a depression score greater than 7, of these, 11 (73%) suffered from coexisting or comorbid somatoform disorders. Further, out of a total of 6 participants with a score in excess of 9, 5 (83%) belonged to the comorbid somatoform disorder group.

These depression findings suggest that comorbidity exacerbates the prevalence and severity of depression among migraine patients, and that negative emotional states not only influence, but also increase the potential for psychosomatic dysfunction. As comorbidity compounds debilities, self-esteem and self-perceived coping abilities are further diminished (Osterhaus et al. 1994). It is therefore likely that these patients feel all the more compelled to cling to the only coping mechanism they have come to rely on, namely their escape into pain. This results in non-improvement and a continuation of the vicious circle (Elton et al. 1978) referred to above.

It is also likely, that in the absence of a functional alternative, the adopted dysfunctional alternative becomes established and reinforced through secondary gains, and more deeply entrenched (Rapoport & Sheftell, 1991; Schaefer et al. 1979). It follows, therefore, that comorbidity not only increases debilities, but in the absence of self-perceived coping skills, leads to a compounding of treatment resistance. It is, after

all, a resistance to a treatment which requires the relinquishing of a conditioned habit, of a 'way out' with secondary gains.

It is generally accepted that the clinical purpose of overcoming treatment resistance is to enhance the effectiveness of the treatment administered, and to promote its success. Catalano and Hardin (1994) define treatment resistance as "...all those behaviors in a system that are obstacles to success" (p 90).

This raises the question of which treatment approach might be beneficially employed to overcome, or reduce, resistance arising from comorbid somatoform disorders. From a clinical perspective, the general answer is that any attempt to overcome such resistance, has to address, in the first instance, the psychological factors which contribute to the somatoform disorder (Comer, 1995).

Because of the self-generated origin of resistance, it is unlikely, that self-regulatory approaches by themselves, can succeed in this task. For clinical purposes therefore, the psychological treatment for this task, remains a matter of professional choice.

#### 5.15 The Question of Extraneous Intervention

It is appropriate in any research, to address the question whether the effects achieved are likely to be due to some intervening variables extraneous to the treatment administered. To this end, the researcher asked all participants at the conclusion of the study whether any had undergone some other non-drug treatment in the course of the six months research period, or had changed their normal dietary habits for longer than one week. The response indicated that none of the participants had been involved in these activities.

The researcher also ascertained that participants neither changed the type of their medication during this period nor increased their normal dosage. As previously mentioned, a correlation is believed to exist between the use of migraine medication to reduce the severity of pain, and prolonged duration (Sachs, 1985). As participants were able to reduce their medication subsequent to treatment, it is possible that this reduction in medication also contributed to the reduction of the duration of migraine. If this is the case, the reduction in duration could still be considered a response (indirect) to the treatment. In view of these, and the above considerations, the likelihood of the results achieved in this study being due to, or markedly affected by extraneous factors, is considered minimal.

#### 5.16 The Four Constituent Treatment Modalities: A Theoretical Appraisal

The treatment in this study, which was administered and tested in two integrated treatment streams, comprised four constituent treatment modalities in all, with three modalities in each stream. The first modality was group hypnosis used for training and treatment suggestions. The second modality was hypnotic relaxation used daily as a relaxation routine, and also as the induction procedure prior to the use of each vascular manipulation modality. The third modality was vascular manipulation A (directed to reduce the frequency, duration, and severity of migraine). The fourth modality was vascular manipulation B (directed to reduce the frequency, duration, and severity of aura).

This research was conducted in a manner and with a view to facilitate easy replication of the treatment by clinical practitioners with patients. Although most clinical considerations are theoretically based, they are of necessity, pragmatic in treatment

orientation. The adoption of treatment procedures depend largely on their demonstrated effectiveness. It is therefore important from a clinical perspective, that each major treatment procedure used, has the capacity to contribute to the effectiveness of the treatment as a whole, and that the capacity or 'functional relevance' of each procedure or modality, can be theoretically demonstrated.

Accordingly, such demonstration is the aim of this section. Although this study did not seek to measure the effectiveness of each modality by itself, its individual functional relevance may be derived and postulated on the basis of past research results, and where such research has not yet been undertaken (as in the case of aura), it may be theoretically postulated in terms of its potential effectiveness (Day, 1961; Wisdom, 1952).

Although it is important to test the individual effectiveness of each treatment modality used, this study initiated a tri-modal approach, and as such, this study sought to determine the effectiveness of their combined application. Future research may well be directed to determine the impact of each individual modality.

#### 5.16.1 Hypnotic Relaxation

The two treatment modalities of vascular manipulation A and B were applied only in response to the manifestation of aura or migraine symptoms. Hypnotic relaxation however, was practiced independently on a daily basis, irrespective of the presence of migraine or aura. Its capacity to contribute to the effectiveness of pain treatment is well documented (Graham, 1990; Sarbin & Slagle, 1993; Tasto & Hinkle, 1973; van Dyck et al. 1991).

Self-induced hypnotic relaxation has two major functions. The first of these is a physiological one, producing a slowing down of systemic functions, counteracting

symptoms of stress (Montgomery & Evans, 1987; Warner & Lance, 1975), and inhibiting the release of vasopressor agents such as adrenaline and noradrenaline, commonly associated with migraine prodromes and aura (Lance, 1993).

As self-hypnotic relaxation inhibits the physiological preconditions associated with migraine and aura, its capacity to contribute to the effectiveness of their treatment is also demonstrated in physiological terms. Similarly, when practiced during migraine prodromes or aura, these same inhibitory physiological responses, may also inhibit or reduce the severity and duration of these symptoms. Further, the severity of migraine is understood to be positively correlated and synchronised with arterial pulsation (Lance, 1993; Petty, 1987), and as arterial pulsation can be reduced through relaxation, it is likely that the severity of migraine is similarly responsive.

The second major function of self-induced hypnotic relaxation as a treatment process, is a psychological one. The self-induced, noticeable physiological changes demonstrate to practitioners the effectiveness of their personal intervention, thereby leading to an increase in the awareness of self-empowerment, self-esteem, and psychophysiological well being (Milne, 1995; Stanley, 1993).

Further, given the psychosomatic nature of migraine, and its demonstrated association with low self-esteem (Elton, et al. 1993) and low coping skills (Frank, 1972), it is reasonable to conclude that the psychological enhancement of these factors, as derived from self-induced hypnotic relaxation, can facilitate a reduction in the measures of frequency, duration, and severity, of both migraine and aura. These psychological and physiological functions of hypnotic relaxation thus combine to demonstrate the

functional relevance of self-induced hypnotic relaxation in the treatment of both migraine and aura.

#### 5.16.2 Group Hypnosis

Although group hypnosis was administered to participants during the week preceding the commencement of the treatment phase, the suggestions given, were post-hypnotic and addressed issues relating to participants' future practices of treatment and recording procedures. As such, they are operationally a part of the treatment. Similarly, reservations that suggested belief in treatment success may have a confounding effect, do not apply, since these beliefs were all hypnotically induced, and were therefore integral parts of the hypnotic treatment.

Studies comparing differences in treatment responses between hypnotised groups and individually hypnotised persons, have stressed the similarities between group responses and those elicited in individual treatment sessions (Shone, 1982; Stanley, 1993). Behavioural responses exhibited by participants during the group hypnosis sessions, such as inhibited behavioural arousal, slow rate of respiration, and hypodynamia of the skeletal muscular system, indicated that participants were in a state of hypnosis and therefore, were likely to be receptive to the treatment suggestions given by the researcher (Hess, 1949; Langen, 1993). In view of these considerations, it is reasonable to assume that group hypnosis is of functional relevance to the success of both migraine and aura treatments.

#### 5.16.3 Vascular Manipulation A

Vascular manipulation for migraine pain, which involves focus on constricting dilated and swollen extra-cranial blood vessels, has been shown to be an effective

treatment modality in its own right (Harding, 1967, 1978; Milne, 1983). For example, the positive results of Harding's vascular manipulation treatment for migraine, were achieved without hypnotic relaxation either as an independent component, or as part of the induction procedure. The induction procedure, practiced by Harding in these treatments, involved the use of Spiegel's (1970) eye-roll technique without the addition of a progressive relaxation component. The demonstrated effectiveness of vascular manipulation justifies the conclusion that the vascular manipulation A modality has functional relevance in the treatment of migraine.

#### 5.16.4 Vascular Manipulation B

The final question to be considered in this context, is the role of vascular manipulation B in the treatment of aura. A review of literature has not revealed that any research into the effectiveness of hypnosis in the treatment of aura was conducted prior to this study. The formulation of a suitable treatment modality for aura was therefore necessary. The choice of a vascular manipulation treatment was made because the neurological symptoms of aura are connected with localised changes in the vascular system.

However, given that the vascular symptomatology of aura is the opposite to that of migraine proper (that is, aura involves a constriction of blood vessels as opposed to a dilatation), it follows that its vascular manipulation treatment has to focus on dilating the affected blood vessels back to their normal size rather than constricting them, as in the case of migraine. Further, in contrast to the extra-cranial location of the blood vessels involved in migraine, those involved in aura are located deeper inside the head, in the midbrain, thus requiring a correspondingly different focus on the part of the participant.

Notwithstanding the connection between migraine and aura, their contra-distinctive physiological and clinical features clearly identify these disorders as separate syndromes, each requiring its own uniquely distinctive treatment. It is erroneous to assume, as has been the case in the past, particularly in terms of pharmacotherapy, that the treatment of migraine also constitutes treatment of aura.

The potential capacity of vascular manipulation B to contribute to the effectiveness of the treatment of aura may be derived from the following. The effectiveness of hypnotic manipulation of the vascular system involved in migraine has previously been demonstrated (Harding, 1967, 1978). An appropriate adaptation of this manipulation technique for the vascular system involved in aura is therefore also likely to be effective.

Although no quantitative claim can be made about the extent to which each individual treatment modality contributed to the effectiveness of the migraine or aura treatment, the functional relevance of each modality has been theoretically demonstrated. Further, from a methodological, and a clinical perspective, the strong treatment effects achieved in all six hypothesised measures indicate that their conjoint employment in an integrated treatment approach enhanced the effectiveness of the treatment in both the migraine and aura streams.

## 5.2 Implications and Recommendations

### 5.21 Implications

Several important implications have emerged as a consequence of this research. The strategies employed in this study have facilitated original contributions in six areas of research involving hypnosis in the treatment of migraine and aura; these are set out below in paragraphs one to eight. Included in this number, are four previously untested

areas in the hypnotic treatment of the migraine syndrome. These pertain to the treatment of aura, the integrated use of three hypnotic modalities as one treatment, and the prevalence and treatment responsiveness of comorbid somatoform migraine patients.

1. The pharmacological treatment for aura has, to date, been the same as that for migraine. The symptoms of aura, however, are physiologically and clinically distinct from the symptoms of migraine. This distinctiveness of aura suggests the need for a treatment which addresses its uniquely different symptomatology. The responsiveness of aura to the hypnotic treatment used in this study, suggests that clinical advantages can be derived for patients from such separate treatment. Other behavioural treatments, specifically for the treatment of aura, may be tested in future studies.

2. There is the further consideration, that the elimination of prodromal conditions for migraine, such as aura, is likely to be instrumental in preventing the development of migraine symptoms, thereby reducing the frequency of migraine.

3. The measure of duration of migraine has, in most migraine studies of the past, been overlooked. Instead, researchers have commonly employed the measure of frequency. An exception to this may be the Headache Index developed by Blanchard and Andrasik (1985), and used by Blanchard, Nicholson, Taylor, Steffek, and Appelbaum (1991). This index has 'duration' imbedded in it. The results of this study demonstrate that the variable of duration is an essential measure for migraine studies testing the effectiveness of a particular treatment. While duration is a measure of the time spent in migraine pain, frequency is a measure of the number of times it occurs within specified time spans. The distinction is clear, and the inclusion of both measures is therefore necessary for a complete measurement of migraine. The same considerations apply to aura.

4. The achievement in this study of six hypothesised main effects, plus six non-hypothesised effects (including three comorbid somatoform effects) demonstrates that the employment of an integrated treatment approach, comprising multiple hypnotic treatment modalities, constitutes a highly efficacious treatment for both migraine and aura.

These results also suggest that this integrated treatment approach, enhanced the overall effectiveness of the treatment.

5. The assessment and analysis of psychological comorbidity in this study has revealed the following four results: (a) psychological comorbidity in the form of somatoform disorders has a strong prevalence (52%) within the migraine population, (b) comorbid somatoform migraine patients are more significantly depressed than non-comorbid migraine patients, (c) the quality of life of comorbid somatoform migraine patients is significantly lower than that of their non-comorbid somatoform counterparts, and (d) the treatment responsiveness of comorbid somatoform migraine patients is significantly lower than that of migraine patients without this comorbidity. This lower treatment responsiveness may be ascribable to a form of treatment resistance.

6. Apart from the comorbidity research undertaken in this study, research on the prevalence and impact of psychological comorbidity on treatment responsiveness and resistance, as well as its impact on depression and quality of life factors, appears not to have been undertaken or documented, yet these associations are of obvious psychological and clinical importance, and merit further investigation.

7. The results achieved in this study support the view that migraine treatment involving self-help skills such as self-hypnosis, is very effective. Practiced self-hypnosis

enhances coping skills, leading to improved self-esteem, confidence, and a diminishing of dependence on dysfunctional coping mechanisms such as an escape into chronic pain and drug use (Spinhoven et al. 1992).

8. One of the major clinical benefits to be drawn from the treatment strategies employed in this study is that the treatment procedure can be readily adapted and taught to patients in a clinical setting. For example, a series of two training sessions preceded by one briefing session, should enable the patient to commence home practice. Ideally, as was the case in this study, the patient can also be issued with an audio tape containing the appropriate suggestions. Additional consultations can be arranged to monitor progress. The advantages of home treatment from the perspective of patient convenience and cost, are obvious. Case studies have shown home practiced hypnotherapy to be very effective (Blanchard et al. 1992; Davidson, 1987; Milne, 1983).

The objectives of this study, which were to test the effectiveness of hypnosis in the treatment of migraine and aura, have now been achieved. The results of this inquiry demonstrate that the question, 'is hypnosis an efficacious treatment for these disorders,' merits a clear, affirmative answer in relation to each symptomatology.

#### 5.22 Recommendations

The subjective experience of chronic migraine pain and aura symptoms do not occur in isolation, they occur rather, within the context of a wide range of psychophysiological conditions. These factors need to be taken into account in the search for a better understanding of migraine and aura, and for the improvement of their respective treatments.

No single model of research or treatment modality for the relief of chronic pain commands universal acceptance. Nevertheless, in principle, research studies directed toward a better understanding of factors contributing to the manifestation of pain, meet with few contentions. This, together with the search for improved treatment modalities, reflect the most common aims in pain research.

The overall aim of this research has been to investigate these important issues as they pertain to chronic migraine pain and aura symptomatology. The results and implications of this study, which have been discussed in preceding sections, lead to the following recommendations for future research.

1. That further testing be undertaken of the treatment responsiveness of aura as a clinically distinct part of the migraine syndrome.

2. That hypnotic aura treatment be tested for its capacity to prevent migraine.

A methodology for this purpose is discussed in section 5.12

3. That further studies be conducted using vascular manipulation B as a modality for the treatment of aura.

4. That the measure of duration be tested in addition to the measures of frequency and severity in all migraine and aura research designed to test the efficacy of any treatment.

5. That a survey be conducted of migraine clinics to determine the prevalence of comorbid somatoform disorders among migraine sufferers.

6. That the impact of comorbid somatoform disorders on quality of life factors and depression, be further investigated.

7. That research be conducted to test the correlation between psychological comorbidity and treatment resistance.

8. That further research be conducted to verify the efficacy of hypnosis in the treatment of migraine and aura, with particular emphasis on the use of integrated, multiple treatment modalities.

9. That in relation to summary comments in section 5.1, research into psychophysiological causal conditions of migraine and aura be jointly conducted by researchers from the professions of medicine and psychology.

10. That the physiological effects, if any, of hypnosis on the neurological vascular system, be jointly investigated by researchers from the professions of medicine and psychology.

## GLOSSARY OF SELECTED MEDICAL AND CHEMICAL TERMS

**Acetylcholine** neurotransmitter at synapses in the central, sympathetic, and parasympathetic nervous system.

**Adrenalin** trade name for epinephrine, a vasoconstrictor agent.

**Amines** biochemical compounds such as neurotransmitters; also organic compounds found in food.

**Amino acid** organic compound forming the chief constituents of protein.

**Analeptic** restorative drug such as caffeine.

**Ataxia** irregularity or deficit of muscular action.

**Bradykinin** a pain causing agent when used in combination with serotonin, may also contribute to vasodilatation.

**Capillaries** small blood vessels.

**Catecholamine** any of a group of amines, including dopamine, epinephrine, and norepinephrine.

**Carotid artery** principle artery of the neck.

**Cortex** outer layer of an organ or structure, as opposed to its inner structure.

**Duramater** the outer covering of the brain.

**Endocrine** pertaining to internal secretions, such as hormones.

**Endothelial cell** pertaining to the lining of the cavities of the heart.

**Enkephalins** natural opioids, also known as endorphins.

**Epinephrine** hormone, a strong vasoconstrictor, increases heart rate and blood pressure.

**Fibrosis** formation of fibrous tissue containing cysts and resulting in vascular constriction.

**Gamma aminobutyric acid (GABA)**, substance released by interneurons, reduces neuron responses to pain messages.

**Hemianopsia** deficit in the visual field, such as areas of depressed vision, as experienced during some auras.

**Hemiplegia** paralysis of one side of the body.

**Histamine** an amine involved in capillary dilation and contraction of muscle tissue.

**Hypothalamus** deals with messages from the autonomic nervous system, and is involved in the control of emotions, metabolism, and pain perception.

**Hypoxia** deficient oxygen reaching the blood.

**Interneurons** various functions, those that are part of the pain control system situated in the brain stem, control the release of enkephalins, GABA, and 'substance p'.

**Ischaemia** inadequate blood supply to one or several parts of the body, such as to the cortex and/or brain stem.

**Locus ceruleus** mid brain nerve fibres involved in the release of noradrenalin to alter transmission of pain impulses.

**Meningeal arteries** situated in the three membrane covering the brain and spinal cord: dura mater, arachnoid, and pia mater.

**Monoamine** molecule containing one amino group such as serotonin.

**Neurotransmitters** facilitate chemical transmission at nerve cell synapses, triggering biological responses.

**Noradrenalin** neurotransmitter, stimulates vasoconstriction.

**Occipital** pertaining to the back part of the head.

**Oedema** pertaining to an abnormal accumulation of fluid.

**Oscilloscope** a ray tube instrument displaying visual representation of electrical variations.

**Osmophobia** sensitivity to smell.

**Parasthetic** positive tactile hallucinations, such as in hands, tongue, or mouth, as opposed to negative anaesthesia.

**Parietal** pertaining to the walls of a cavity or parietal bone.

**Periaqueductal grey matter** nerve fibres from the mid brain, involved in the release of serotonin.

**Periarterial** pertaining to the outer covering of an artery.

**Periosteum** the connective tissue covering all bones, it has pain receptors and is particularly sensitive over the brow.

**Phonophobia** sensitivity to sound.

**Photophobia** sensitivity to light.

**Polypeptides** peptides which contain more than two amino acids.

**Pons** one of three main parts of the cerebral cortex, connects nerves, affects blood supply to the cortex, and can cause the release noradrenaline.

**Prodrome** early symptom, indicating the onset of a disease or a disorder such as migraine.

**Prostaglandins** hydroxy fatty acids, involved in the aggregation of blood platelets, and the dilatation of blood vessels associated with migraine.

**Scotoma** visual disturbances of luminous spectra, such as bright flashes of light, and zigzag lines, as experienced during some auras.

**Serotonin** neurotransmitter strongly implicated as a contributory cause of migraine, affects the blood supply to the cortex.

**'Substance p'** the tentative name for what is thought to be a peptide involved in the transmission of pain signals.

**Thalamus** consists of nerve cells involved in relaying pain impulses.

**Temporal** pertaining to the temple.

**Thrombophlebitis** the inflammation of a vein.

**Trigeminal nerve** transmits pain sensations arising from blood vessels in the scalp.

**Tryptophan** amino acid found in milk and turkey, and converted by the brain into serotonin.

**Vasoconstriction** a decrease in the calibre of blood vessels.

**Vasodilatation** an increase in the calibre of blood vessels.

**Viscera** any large interior organ, especially pertaining to the abdomen.

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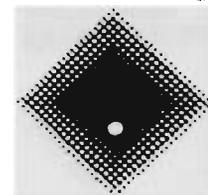
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# MIGRAINE SUFFERERS

**VICTORIA  
UNIVERSITY**



OF  
TECHNOLOGY

## Free Treatment and Training **VOLUNTEERS WANTED**

to participate in  
**DRUG FREE RESEARCH**

conducted by  
**Gitta Trexler**

B.A., M.A., (PHIL), B.LITT., M.A. (PSYCH) Member of the International Society of Hypnosis

Research into the effectiveness of hypnotherapy in the treatment and prevention of  
**MIGRAINE** is being conducted by Ms **GITTA TREXLER** at the  
**Victoria University of Technology, from February to September, 1996.**

All treatment is *free of charge*, non-invasive, drug free and self-administered by participants after having been trained in self-hypnosis.

Migraine sufferers who meet all the selection criteria set out below,  
and who are interested in participating in the study,  
may obtain further information by sending a stamped, self-addressed envelope to:  
**MS G. TREXLER, Department of Psychology, VUT, P O Box 692, Heathmont, 3135**

### **Selection Criteria:**

Volunteer subjects must be:

- over 18 years of age and give informed consent in writing
- fluent in the English language
- diagnosed migraine patients **and must also:**
- have experienced migraines in the past twelve months
- currently experience at least two migraines per month
- refrain from using any drug free treatment other than the prescribed hypnosis during their participation
- practice the prescribed hypnotherapy to reduce severity, duration and frequency of their migraines
- suffer from no psychological disorder for which hypnotherapy is contra-indicated

**VICTORIA  
UNIVERSITY**



OF  
TECHNOLOGY

## Appendix B



Dear Volunteer,

Thank you for your interest in this research which seeks to investigate the extent to which, if any, self-hypnosis can reduce or eliminate migraine pain. The effectiveness of hypnosis in the treatment of pain is well known in medical and associated professions. For example, hypnotherapy has been used successfully to reduce pain in childbirth, arthritic conditions, and tension headaches etc.

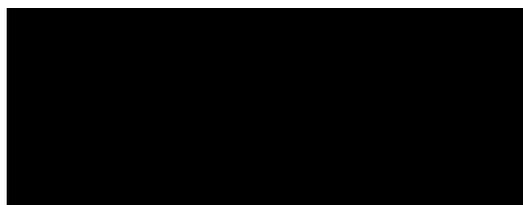
The aim of this study is not only to determine if hypnosis can be used with similar effectiveness in the treatment of migraine pain, but by using **self-hypnosis**, this study also seeks to place the therapy and its results firmly within the control of the migraineur.

If you meet all of the selection criteria set out on the attached Registration Form and you are interested in participating in this study, please complete and mail the Registration Form at your earliest convenience. **It will then be necessary for you to attend the Information Seminar on February 20th. 1996, 11.00 am to 1.00 pm, Seminar Room, 1st. floor, Hawthorn Medical Centre, 140-142 Barkers Road Hawthorn.** Receipt of your registration will reserve a place for you at the seminar. Participation is free of charge. Should you subsequently need to cancel, please leave a message to that affect on 9819 9877 so that someone else may take your place.

Formal selection of participants will be made at the Seminar after details of the study have been explained and questions answered. In the meantime however you might find it helpful to know that all participants will receive free treatment and training in self-hypnosis prior to the start of the study. All subsequent treatment will be self-administered by participants in their own home. Procedures and results will be monitored on a regular basis by Gitta Trexler throughout the research period. The study requires preparedness by volunteers to participate for a period up to 6 months.

I hope you will be able to participate in this research, and I look forward to meeting you at the Seminar.

Your sincerely,



Gitta Trexler



Appendix D

Victoria University of Technology

Sample Consent Form for Subjects Involved in Experiments

CERTIFICATION BY SUBJECT

I,
of
certify that I have the legal ability to give valid consent and that I am voluntarily giving my consent to participate in the experiment entitled:

Effectiveness of Hypnosis
in the Treatment of Migraine and Aura

being conducted at Victoria University of Technology by:

Gitta Trexler

I certify that the objectives of the experiment, together with any risks to me associated with the procedures listed hereunder to be carried out in the experiment, have been fully explained to me by:

Gitta Trexler

and that I freely consent to participation involving the use on me of these procedures.

Procedures:

Group Hypnosis, and as per audio tape suggestions, self-hypnosis for relaxation (daily), and for migraine and aura symptoms (on presentation).

I certify that I have had the opportunity to have any questions answered and that I understand that I can withdraw from this experiment at any time and that this withdrawal will not jeopardise my in any way.

I have been informed that the confidentiality of the information I provide will be safeguarded.

Signed:..... }

Witness other than the experimenter: }

Date: .....

.....}

Any queries or complaints about your participation in this project may be directed to the experimenter, or to the Secretary, Human Research Ethics Committee, Victoria University of Technology, PO Box 144428 MMC, Melbourne, 3000 (telephone no: 03-9688 4710).

My migraine condition was diagnosed .....

..... by .....in 19 \_\_\_\_\_

Signature: \_\_\_\_\_

## Appendix E

**BASELINE INFORMATION**

Please Print

Name in full: \_\_\_\_\_ Phone: \_\_\_\_\_

Address: \_\_\_\_\_ Code: \_\_\_\_\_

Sex: \_\_\_\_\_ Age: \_\_\_\_\_

**MIGRAINES/AURAS****FREQUENCY.**

How many Migraine days do you have per month? \_\_\_\_\_

How many Auras do you have per month? \_\_\_\_\_

**SEVERITY.**

How severe are your Migraines on average? \_\_\_\_\_

1 = light, 2 = mod., 3 = severe

How severe are your Auras on average? \_\_\_\_\_

1 = light, 2 = mod., 3 = severe

**DURATION.**

How long do your Migraines last on average? \_\_\_\_\_ hrs.

How long do your Auras last on average? \_\_\_\_\_ hrs.

**MEDICATION**

Name/s of migraine medication taken on migraine days:

1. \_\_\_\_\_ Total daily dosage: \_\_\_\_\_ mg.

2. \_\_\_\_\_ Total daily dosage: \_\_\_\_\_ mg.

What side effects are there if any? \_\_\_\_\_

Do you take any of the above migraine medication each day even when you don't have a migraine? YES \_\_\_ NO \_\_\_

What other medication do you take regularly? \_\_\_\_\_

For what purpose? \_\_\_\_\_

What side effects are there if any? \_\_\_\_\_

## Appendix E

## Baseline Information

Quality of Life and Stress

Please answer questions 1 to 9 by using a 1 to 5 scale, where 1 represents 'poor' and 5 represents 'excellent', with numbers 2, 3, and 4 representing graded levels in between. The same scale applies to question 10, but here 1 represents 'low stress' and 5 represents 'high stress' with graded levels in between. Please choose the number which most closely represents the way you feel about the question asked.

1. My health \_\_\_\_\_

2. My emotional security \_\_\_\_\_

3. My energy \_\_\_\_\_

4. My attitude to the future \_\_\_\_\_

5. My self esteem \_\_\_\_\_

6. My confidence \_\_\_\_\_

7. My sense of control \_\_\_\_\_

8. My task achievement level \_\_\_\_\_

9. My relationship satisfaction \_\_\_\_\_

10. My stress level \_\_\_\_\_

Comments:.....

.....

## Appendix F

## LOG SHEET (A)

**Instruction Note.**

Please use a separate sheet for each experienced Migraine.

IF your Migraine is preceded by prodromal symptoms or Aura, please record those together with your Migraine on the same sheet.

IF you only experience prodromal symptoms or Aura without a subsequent Migraine, please record those on a separate sheet.

IF your week has been free of prodromal symptoms, aura and Migraines, you still need to fill in Log Sheets (A) and (B) to answer all the other questions. Please mail your log sheets every second Friday on the dates specified.

NAME..... SIGN..... TEL.....

PHASE: I or II.

WEEK ENDING...../...../ 96

Migraine/Aura

Date	Migr./Aura	Start Time		Finish Time		Duration		Improvement		Max Severity L1 M2 H3
		hr.	min.	hr.	min.	hr.	min.	Start time	hr.	
	Migr.									
	Aura									

Self-Hypnosis

Date	Type of self- hypnosis	Time 1st practice		No. of times done	Total Duration		Max. Level hypnosis L1 M2 H3
		hr.	min.		hrs.	mins.	
	Relax						
	V.M. (A)*						
	V.M. (B)						

\* (A) = Hypnosis for Migraine. (B) = Hypnosis for Aura

Appendix G

LOG SHEET (B)

Instruction Note.

Log sheet (B) needs to be filled in by you every day of the week, irrespective of whether you have a Migraine/Aura or not.

Please mail your log sheets every second Friday.

NAME..... SIGN..... TEL.....

PHASE I or II.

WEEK ENDING...../...../96

Please answer questions 1 to 9 by using a 1 to 5 scale, where 1 represents 'poor' and 5 represents 'excellent', with numbers 2, 3, and 4 representing graded levels in-between. Please choose the number which most closely represents the way you feel on that particular day about the question asked.

Description	Sun.	Mon.	Tues.	Wed.	Thur.	Frid.	Sat.
1. My health							
2. My emotional security							
3. My energy							
4. My attitude to the future							
5. My self esteem							
6. My confidence							
7. My sense of control							
8. My task achievement level							
9. My relationship satisfaction							
10. My stress level							

Please record your stress level by using a 1 to 5 scale, where 1 represents 'low stress' and 5 represents 'high stress', with numbers 2, 3, and 4 representing stress levels in-between.

Comments:.....

## Appendix G

Definitions of Quality of Life Terms  
(as presented and discussed with participants)

**Health**, general overall health as it is today.

**Emotional Security**, feeling reasonably safe and relaxed about relationships, events, and developments of importance.

**Energy**, physical and mental energy.

**Attitude to Future**, emotional outlook toward the likely quality of future developments and life events.

**Self-esteem**, self-image of the person I am, and self-worth.

**Confidence**, belief in my coping and managing skills.

**Sense of Control**, feeling of being adequately in control of my commitments and the way I react to them.

**Achievement Level**, the degree of meeting set tasks and goals.

**Relationship Satisfaction**, this includes all relationships, partners, family members, friends, colleagues, acquaintances.

**Stress**, anxiety and concern regarding private life or career.

## Appendix H

### Relaxation and Migraine Treatment

Dear Volunteer, this is side A of your hypnotherapy tape. It is for your use only so please keep it safe. It is scientifically important that you use the sections on this tape strictly for their unique and individual purpose. So please make sure you do not inadvertently listen to the wrong side or section. This is side A, it contains two sets of hypnotherapeutic suggestions, namely (a) your relaxation routine which you are asked to practice at least once every day, and (b) the hypnotherapy to be used only when you have a migraine. There are no restrictions on the number of times you can use the relaxation session in any one day, and there are no restrictions on the number of times you can use the migraine section in any one day so long as you have migraine pain.

#### Relaxation Section A

Please make yourself comfortable now in readiness for your relaxation period..... You begin by turning your eyes upward as though you are looking at the crown of your head... that's right, eyes right up, and keeping your eyes in that position, gently close your eyelids now.... that's right.... still keeping your eyes upward under your closed eyelids take in a gentle breath and hold it to the count of four, 1... 2... 3... and 4, exhale now slowly and deeply, and as you do, tension and stress are leaving your body... yes let it go.... let it all go.... That's better now, see you are feeling much more relaxed already... things can be so simple,... you are doing very well, letting go of stress, of anxiety and of fear... Your breathing has adjusted itself to an easy rhythm, this happens quite

## Appendix H

automatically when you allow yourself to relax... no need to think about it any more.

And now allow your eyes to return to their normal comfortable position under your closed eyelids, if they haven't done so already....there, that's very good.

You can now allow your forehead to relax, you do this by allowing your eyebrows to droop.... that's good, and now you can release the tension in your jaw, by opening your mouth a little..... good, and now, let go of the tension in the shoulders by allowing them to droop as well... yes that feels good and easy... so very very easy... And now, allow the tension to leave your chest and your stomach by tightening up those muscles first, and then letting go.... more and more relaxed and at ease, well done... more and more relaxed and feeling good.

And now for a special treat and to release any last, left-over tension, visualize yourself lying on a sheltered beach.... See yourself on this beautiful secluded beach...

It is a warm and sunny day, the sky is blue, and there is a gentle breeze about.... feel it caressing your cheeks and shoulders.... It is very quiet.... so quiet so serene.... feel the peace and tranquillity that surrounds you ... allow yourself to enjoy this peaceful experience....This is a special beach for you, it may be familiar to you, or perhaps you are creating it in your mind's eye, so it looks and sounds and feels just the way you want it to.... This is your special place where you feel at peace.... without stress and worries ... where you feel so safe and so so calm... and so very very relaxed.... Your mind enjoys this calm and beautiful state of relaxation just as much as your body does, it is highly beneficial for your emotional and physical well being....

## Appendix H

You already know you will want to revisit this special place of yours every day, and when you do, your entire system can work with ease and renew its energies.... Well done,.... so beneficial.... so deeply relaxed.... so safe.... so much at ease.... Continue to relax and enjoy your relaxation for a while longer before you either alert yourself, or if you have a migraine now, you will want to go on to the migraine section.... if that is so, you can simply stay relaxed and ignore the alerting suggestions until after your migraine section, then you will alert yourself very readily... you will also feel good about your successful reduction of migraines.

If you were free of migraine pain at the start of this tape, you will feel ready to become alert now and conclude your daily relaxation routine.... staying very comfortable and maintaining your sense of comfort,... you will nevertheless become progressively more and more alert as I count backwards from ten to one, becoming progressively more alert with each count, opening your eyes at two, and feeling fully alert and refreshed at the count of one. Counting backwards now... 10 beginning to alert.. 9 becoming more alert.. 8 and more alert.. 7 feeling fine.. 6 and 5.. more and more alert.. 4 and 3.. feeling refreshed and well.. 2 eyes open.. open wide.. good,.. and now 1, take a good breath in.. and breath out.. there, feeling good and fully alert with renewed energy (length of relaxation with alerting section: 10 mins.)

### Migraine Section A

This is your migraine section, it contains hypnotherapy suggestions to be used when you have a migraine. Your focusing in response to these suggestions will reduce the severity, duration, and frequency of your migraine headaches. To assist your focus

## Appendix H

and relaxation, you will want to repeat your eye roll now, ... opening your eyes.. turning them upwards as though you were looking up at the crown of your head....and leaving your eyes in that position gently close your eyelids now, that's good...now take a breath in....hold it to the count of 1...2...3...and 4, slowly exhale now and as you do, feel any left over tension leave your body....let this tension slip away... let it all go... good, that does feel better doesn't it... It feels good also to know that you have the skill to free your whole system of stress in this way any time you want to.... Allow your eyes now to return to their normal comfortable position if they haven't done so already,... that's good... feeling relaxed and more and more at ease, knowing you are helping yourself...knowing you are getting better and better at helping yourself... you are becoming more and more successful at reducing your migraines.... you are becoming more and more successful at controlling your migraine pain... and this is good to know, it gives you strength.

Focus now on the site of your migraine pain, let the throbbing blood vessels guide you to it initially... You can reduce this painful dilatation and swelling,... you can cool these affected blood vessels so they return to their normal size.... and as they get smaller and smaller and they return to their normal size, so the pain becomes less and less until you too, feel back to normal again.... Enjoy this knowledge for a while as you remain relaxed and at ease.

As you narrow your focus now, you can slowly visualize a helmet to fit your head comfortably, ... see it very clearly... its colour is a cool silvery blue, and it is very light in weight... as you focus still further in, you notice

## Appendix H

its special feature on the inside... it has specially fitted freeze coils in those parts of the helmet which coincide with your migraine sites. The coils and their protective cover remain flexible even when frozen so that when you put the helmet on, the cool coils can easily and snugly follow the contours of your head and cover those parts where your migraine... As you look closer now you also see the ON/OFF switch and the temperature control dial on the outside of your helmet, and all are within your easy reach. See the five temperature settings: ON, OFF, COOL, VERY COOL, and COLD. You control the ON/OFF switch, and adjust the temperatures in accordance with your migraine needs and comfort.

It is possible now for you to set all worries aside.... allowing yourself true peace of mind.... there is nothing to disturb you now.... you are finding it easier and easier to just let go and to listen to my voice.... knowing that you are being safely guided to take control.... to ease the dilatation and the swelling of your affected blood vessels, and to let the pain ease away.

Having familiarized yourself with your very special cooling helmet you can now gently put it on your head.... it is light and fits very comfortably... you make sure it fits snugly on the migraine sites so that the contact between the protective cover of the freeze coils and the swollen blood vessels is direct... that's right, you can now turn the switch to ON, and set the temperature control to COOL.... aah, now you allow yourself to relax even further, you feel the comforting cool against those throbbing blood vessels.... You begin to feel a soothing effect already... you welcome this soothing coolness...and so you allow the freeze coils behind their protective cover to go on cooling these affected blood vessels near the skull on the migraine sites of your head...

## Appendix H

let those affected blood vessels cool...let them cool gently ...let the swelling go down and their diameter return to normal... and let the pain go at the same time ...let the pain go as the blood vessels cool and return to their normal size.

You find it very easy now to stay focused in this manner... to control the temperature as you think necessary and to enjoy the increasing comfort as the cooling is having its desired effect and the pain eases... You feel so much better as the cool reduces both the swelling and the diameter of the affected blood vessels... allowing these blood vessels to return to their normal size inside and out.... Stay focused on this comfort and positive effect.... focus exclusively now on this comforting coolness and the easing of the pain... and now, visualise again and again, how the freeze coils gently take away the swelling of the blood-vessels on the migraine sites of your head... see and feel how this allows their size to return to normal and the pain to go away....

In a little while when you feel much better, and you feel ready to return to full alertness you will want to turn the switch outside your helmet to the OFF position and gently take your helmet off, and although your head will feel perfectly normal in all respects after you have alerted yourself, a sense of gentle comfort will remain with you for the rest of the day.... In a little while.... now when you are ready, you will easily and fully alert yourself by counting backwards from ten to one in exactly the same way as suggested in the alerting instructions at the end of your daily relaxation session. And when you do, you will have a true sense of achievement, well being, and personal control. (length of migraine section = 15 mins.).

## Appendix I

## Relaxation and Aura Treatment

This is side B of your tape, it contains hypnotherapy suggestions to be used when you have aura symptoms. You may use this tape whenever you feel the need to do so, there are no restrictions on the number of times you can use it in any one day. Your focusing in response to the suggestions in this section will help to relax you and reduce or eliminate these aura symptoms, it could also prevent the onset of a full migraine.

Relaxation B

Make yourself comfortable now.... take your time.... you are probably looking forward to the time when your aura symptoms will ease, knowing as you do, that your aura symptoms will ease very soon. Begin your self-hypnotic procedure with your eye roll technique, turning your eyes upwards as though you were looking at the crown of your head... turn your eyes up as far as you can.... that's good and now, leaving your eyes in that position gently close your eyelids.... good, and still keeping your eyes in the up position, you can now take a big breath in.... good, hold it for 1....2....3....and 4, and now exhale slowly.... very slowly and deeply....and as you do, feel the tension in your body drain away....there, that feels better already.... Your breathing can return to its own easy rhythm now, there is no need for you to think about it any more.... and your eyes can return to their normal position behind your closed eyelids..... all is well.

And now, you can proceed into greater comfort and deeper relaxation by releasing the tension in your forehead... just allowing your eyebrows to droop a little increases your easy relaxation... good,... and now, you may want to release the tension in your jaw by opening your mouth a little... good.... you can also readjust your position to

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make yourself as comfortable as possible...that's good now. Continue to feel comfortable and at ease... Your mind is relaxed along with your body... and you feel secure in this experience knowing that you are being safely guided to take greater control over your aura symptoms and associated migraine...

Your aura symptoms are related to a temporary narrowing of certain veins and arteries inside your head, this causes the blood to be shunted away from those constricted areas. You can reverse this process successfully by directing your focus in accordance with the suggestions given on this tape... As usual, you will find the suggestions and imagery very comforting.

It is possible now for you to set all worries aside, allowing yourself true peace of mind,.... there is nothing to disturb you now... your are finding it easier and easier to relax, to just let go... listen to my voice... more and more relaxed... deeper and deeper relaxed.... feeling secure... feeling in control to restore the constricted blood vessels to their normal comfortable and relaxed state.... You can concentrate your focus now... you can visualize your aura helmet, its colour is a warm red and it fits your head very comfortably, see that it is quite different from the one used for migraine treatment... notice its special feature on the inside, designed to treat aura symptoms only.... It is fitted with warming coils, the exact opposite to your migraine helmet because this your aura helmet has a very different task to perform.... See the soft warming coils positioned to cover your entire scalp area, finishing at your hairline all around, the coils are soft and flexible with a protective shield so they can quite safely and comfortably follow the contours of your head giving off just the right amount of warmth you want... Look closer now and see the control panel... there is the ON/OFF switch and the temperature

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control dial, both are situated within your easy reach on the outside of the helmet. The temperature range is MODERATE.. WARM.. and VERY WARM.... you can always choose and adjust the temperature in accordance with you aura needs and comfort...

Now that you are familiar with your special aura helmet you can put it on.... You notice that it is light and fits very comfortably all around... you are now ready to turn the switch to ON and set the temperature to WARM.... You may allow yourself to relax even more now as you feel the comforting warmth... and soon... very soon... an easing of your aura symptoms..... you can feel that comforting warmth... you can feel it radiating from the coils in your aura helmet... and now you can feel this warmth right close to the affected blood vessels.... it feels soothing and reassuring.... it feels comfortable and easy.... let those affected blood vessels open up to this comforting warmth... let them ease and open up... see them opening up slowly and gently... very gently... relax... relax... enjoy this warmth you feel... let that warmth continue to comfort the affected blood vessels... you maintain your focus so easily... and you go on directing the warmth toward those blood vessels.... let them continue to open up... gently... gently... until they have returned to their normal size... until the blood is circulating easily inside them... and you feel so much better... feel the symptoms ease.

You find it so easy to stay focused in this way.... feeling comfortable and so deeply relaxed.... feeling again and again, the radiating warmth... the blood vessels opening up... gently... gently... the increasing comfort... the easing of your aura symptoms... they are easing... leaving you with a real sense of well being... You have achieved a good deal already... you can feel pleased... you are very successful in reducing your aura symptoms.

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In a little while when you feel ready, you will want to become fully alert, and when you do, you will turn the switch outside your helmet to the OFF position and gently remove your red aura helmet. After you have alerted yourself, your head will feel perfectly normal in all respects, and a sense of well being and gentle comfort will remain with you for the rest of the day..... Very soon now, you may alert yourself very easily by counting backwards from 10 to 1... becoming more and more alert with each count... 10... 9... beginning to alert... 8.. more alert.. counting backwards and alerting.. alerting and feeling good... well done. Eyes open at the count of 2.. good. Feeling fully alert and refreshed now at the count of 1. Well done, feeling pleased and feeling good. (length of side B = 18 minutes).

## Appendix J

### Group Induction (training session)

#### Introduction

Before we proceed to our first practice session of hypnosis, I would like to repeat briefly, the most relevant points of hypnosis within the context of its practical application to relaxation, aura and migraine. Hypnosis is a focused way of communicating with your brain. The imagery you focus on, is communicated to your brain by way of signals, and your brain responds in accordance with the signals it receives. If, for example, you perceive yourself in danger, your brain responds to those signals by releasing certain chemicals which increase your heartbeat, blood pressure, and muscular tension. All these changes occur to prepare you to deal with the perceived danger. It is known as the 'fight or flight syndrome.'

If on the other hand, you perceive calming images, your brain responds by releasing chemicals which have relaxing effects, allowing your whole system to function with ease, and reducing your heart rate, blood pressure, and muscular tension. The good news for our purpose is, that the brain does not differentiate between messages or signals based on reality and those based on imagination; dreams and nightmares are prime examples in point. If for example, you have a bad nightmare and perceive yourself in danger, your brain will release sufficient 'flight or fight' chemicals, such as adrenalin, that you will most likely wake up in fright with severe palpitations.

Hypnosis makes use of this natural communication method, which enables us to exercise some control, to communicate by choice, those images and signals, to which our

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brain responds in a relaxing and comforting way. It is this natural way of communicating with our brain, which also enables you to control your migraines.

Your migraine pain is related to a dilatation and swelling of the blood vessels between the skull and the scalp, and your aura involves a constriction of blood vessels somewhat deeper inside your head. You will be able to reduce the frequency, severity, and duration of your migraines and auras by focusing on the arteries, and visualising them returning to their normal size. Your treatment tapes will guide you, and your success will become more evident to you each time you practice your self-hypnosis treatment. Your daily relaxation practice will reduce the frequency of your auras and migraines, and your prompt focus on the imagery suggested for migraine and that suggested for aura, will reduce the severity and duration of each.

### Group Hypnosis

Please make yourself comfortable now in readiness for your first of many enjoyable relaxation sessions. This easy procedure is identical with the relaxation procedure on side A of your tape, this practice and experience now, is part of your training to acquaint you with the procedure and to demonstrate how straight forward it is. You begin by turning your eyes upwards as far as they will go, as though you are looking at the crown of your head.... that's right, eyes right up, and keeping your eyes in that position, gently close your eyelids now.... that's right.... still keeping your eyes upward under your closed eyelids take in a gentle breath and hold it to the count of four, 1... 2... 3... and 4, exhale now slowly and deeply, and as you do, tension and stress are leaving your body... yes let it go.... let it all go....

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That's better now, see you are feeling much more relaxed already... things can be so simple,... you are doing very well, letting go of stress, of anxiety and of fear... Your breathing has adjusted itself to an easy rhythm, this happens quite automatically when you allow yourself to relax... no need to think about it any more. And now allow your eyes to return to their normal comfortable position under your closed eyelids, if they haven't done so already....there, that's very good.

And now, you can allow your forehead to relax, you do this by allowing your eyebrows to droop.... that's good, and now you can release the tension in your jaw, by opening your mouth a little..... good, and now, let go of the tension in the shoulders by allowing them to droop as well... yes that feels good and easy... so very very easy... And now, allow the tension to leave your chest and your stomach by tightening up those muscles first, and then letting go.... more and more relaxed and at ease, well done... more and more relaxed and feeling good.

And now for a special treat and to release any last, left-over tension, visualize yourself lying on a sheltered beach.... See yourself on this beautiful secluded beach... It is a warm and sunny day, the sky is blue, and there is a gentle breeze about.... feel it caressing your cheeks and shoulders.... It is very quiet.... so quiet so serene.... feel the peace and tranquillity that surrounds you ... allow yourself to enjoy this peaceful experience.... This is a special beach for you, it may be familiar to you, or perhaps you are creating it in your mind's eye, so it looks and sounds and feels just the way you want it to.... This is your special place where you feel at peace.... without stress and worries ... where you feel so safe and so so calm... and so very very relaxed.... Your body mind

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enjoy this calm state of relaxation .... this deep relaxation is very beneficial for your emotional and physical well being....

You will want to revisit this special place of yours every day.... relax like this every day... and when you do, your entire system can work with ease and renew its energies.... allow yourself to enjoy this deep relaxation.... Well done....so beneficial.... so deeply relaxed.... so safe.... so much at ease.... stay with your focus and enjoy your relaxation for a while longer until I speak to you again, and when I do, you will hear my voice very clearly.....

Now, as you feel so deeply relaxed.... and more and more at ease within yourself.... and more and more at ease about using the hypnotherapy on your tape.... and keeping to the suggestions in it.... you find it is all so very easy.... so very natural.... hypnosis is so natural and so very relaxing.... deeply relaxing.... it comes to you with great ease.... doing very well already.... your success in reducing your migraines and auras is assured.... yes, your success in reducing your symptoms is assured.... Your daily relaxation practice will be comforting and beneficial... use the migraine and aura sections as required... so simple... so easy to be successful in reducing your migraine and aura symptoms... so important to record accurate data on your log sheets and mail them as asked.... your success is assured... your success increases every time you practice your hypnotherapy.... should you ever be unable to use your tape, you can activate the procedures from memory, and use them very effectively. But you will always prefer to use your tape if possible.

Maintaining your sense of comfort and of success, you become progressively alert as I count backwards from ten to one. You become progressively more alert with

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each count.. opening your eyes at two.. and feeling fully alert and confident about your success in reducing your migraine and auras by the count of one. Counting backwards now... 10 beginning to alert.. 9 becoming more alert.. 8 and more alert.. 7 feeling fine.. 6 and 5.. more and more alert.. 4 and 3.. feeling refreshed and confident.. 2 eyes open.. open wide.. good,.. and now 1, feeling good.. fully alert with renewed energy and confidence. (length of group hypnosis session = 18 mins.)

## Appendix K

## General Health Questionnaire

Name: (please print) \_\_\_\_\_

No. \_\_\_\_\_

Have you suffered many physical complaints over a period of years before you were 30 years old, which needed medical treatment or significantly detracted from your social, occupational, or other important areas of functioning?

NO \_\_\_\_\_ YES \_\_\_\_\_ How many complaints? \_\_\_\_\_

Is this still the case? NO \_\_\_\_\_ YES \_\_\_\_\_ How many complaints? \_\_\_\_\_

Have you experienced the following symptoms separately from Migraine attacks?

1. Pain in many different areas of your body eg. in your head, back, abdomen, joints, chest, extremities, rectum, or pain during menstruation, during intercourse, or during urination?

NO \_\_\_\_\_ YES \_\_\_\_\_ In how many places? \_\_\_\_\_

2. Nausea, bloating, vomiting, intolerance to different foods, or diarrhoea?

NO \_\_\_\_\_ YES \_\_\_\_\_ How many? \_\_\_\_\_

3. Sexual or reproductive difficulties other than pain, eg. sexual indifference, erectile or ejaculatory dysfunction, irregular menses, excessive menstrual bleeding, or vomiting throughout pregnancy?

NO \_\_\_\_\_ YES \_\_\_\_\_ How many? \_\_\_\_\_

4. Impaired co-ordination or balance, paralysis or localized weakness, difficulty in swallowing, lump in throat, urinary retention, loss of touch or pain sensation, hallucinations, double vision or blindness, deafness, seizures, amnesia, loss of voice, or loss of consciousness other than fainting?

NO \_\_\_\_\_ YES \_\_\_\_\_ How many? \_\_\_\_\_

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General Health Questionnaire

5. Have you ever had any of the following symptoms lasting 6 months or longer: Fatigue, loss of appetite, gastro or urinary upsets?

NO \_\_\_\_\_ YES \_\_\_\_\_ How many? \_\_\_\_\_

6. Have you ever had a combination of these symptoms in Question 5. above, which lasted more than 1 month?

NO \_\_\_\_\_ YES \_\_\_\_\_ How many? \_\_\_\_\_

7. Were any of the above 6 groups of symptoms due to known medical conditions (other than migraine) or due to medication?

NO \_\_\_\_\_ YES \_\_\_\_\_ If yes, please give details below.

Please state each symptom due to known (non-migraine) medical conditions:

\_\_\_\_\_  
\_\_\_\_\_  
\_\_\_\_\_  
\_\_\_\_\_

8. Please state any symptoms due to (non-migraine) medication:

\_\_\_\_\_  
\_\_\_\_\_  
\_\_\_\_\_  
\_\_\_\_\_

Thank you!