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THE EFFICACY OF HYPNOTIC AND NONHYPNOTIC RESPONSE-BASED
IMAGERY TRAINING FOR THE SELF-MANAGEMENT OF RECURRENT HEADACHE

ROBERT P. NOLAN

SUPERVISED BY NICHOLAS P. SPANOS, PH.D.

A Dissertation Submitted to the Graduate Faculty
of Carleton University in Partial Fulfillment of the Requirements
for the Degree

DOCTOR OF PHILOSOPHY (PSYCHOLOGY)

PSYCHOLOGY DEPARTMENT, CARLETON UNIVERSITY

OTTAWA, ONTARIO, CANADA

MARCH 3, 1989

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The undersigned hereby recommend to
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in partial fulfilment of the requirements
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March, 1989

ROBERT P. NOLAN

The Efficacy of Hypnotic and Nonhypnotic Response-Based
Imagery Training for the Self-Management of Recurrent Headache
(Under the direction of NICHOLAS P. SPANOS)

This dissertation research is comprised of two experiments that investigated the use of Hypnotic and Nonhypnotic Response-based Imagery training for recurrent headache. Experiment 1 included subjects who presented symptoms of chronic migraine headache (CMH) or chronic mixed migraine/tension headache. The efficacy of Imagery training was assessed over 2-week intervals, at Baseline, Post-treatment, and 3 successive Follow-up periods. Headache activity across Trials was compared among Hypnotic and Nonhypnotic Imagery subjects and Monitoring Control subjects. Experiment 2 was conducted with subjects who reported symptoms of chronic tension headache (CTH). Four treatment conditions (Hypnotic Imagery/ Nonhypnotic Imagery/Placebo/Control) were studied across 2-week intervals at Baseline, Post-treatment, and 8-week Follow-up.

The principal findings of experiments 1 and 2 were that hypnotic and nonhypnotic response-based imagery training was equally efficacious in reducing headache activity over Trials. This finding was not attributable to alterations in the pattern of medication consumption over Trials. Reduced headache activity also appeared to be independent of actual changes in frontalis EMG and cardiovascular functioning. Placebo and Monitoring Control groups failed to significantly reduce headache activity. Treatment-

induced headache reduction was studied in relation to Baseline measures of affective distress, as well as Baseline and Follow-up illness schemas of recurrent headache as a health problem. Headache reduction covaried with illness attributions only when these were obtained at Follow-up. Finally, treatment-induced headache reduction was not significantly associated with hypnotizability, or the trait propensity to engage in vivid imagery. This finding was consistent with the Social Psychological formulation of hypnotic analgesia, and it was contrary to the Neodissociation account.

Acknowledgements

The mythological student, setting forth from his/her commonday hut or castle, is carried away, or else voluntarily proceeds, to the threshold of adventure. There he/she encounters a shadow presence that guards the passage.... Beyond the threshold, then, the student journeys through a world of unfamiliar yet strangely intimate forces, some of which are severely threatening (tests), some of which give magical aid (helpers). Upon arriving at the nadir of the mythological round, he/she undergoes a supreme ordeal...to gain a supreme reward.

Adapted from:

Campbell, J. (1968). The hero with a thousand faces.

(Second Edition), Princeton, NJ: Princeton University Press.

To participate in the mythological student's adventure has been a privilege. To Nick Spanos, thank you for accepting me into your laboratory, and for your commitment to our research projects. I have always appreciated your generosity in being readily available to discuss the projects, analyses, and papers. I will do well to emulate your example as a scientist, writer, and teacher. Barbara Nolan is a Summa Cum Laude soon-to-be Psychologist who has been my editor, colleague, friend, and wife, since the beginning of my graduate career. Thank you for your expert feedback, unfailing support, and the countless hours that you gave in reviewing this manuscript. I look forward to completing a lifetime of post-doctoral ventures with you. This dissertation research could not have been completed without the assistance of Amber Hayward and Heather Scott. Thank you for your help in running subjects, coding data, and conducting analyses. Thanks are also due to Max Gwynn and Hans de Groot, who always have been willing to provide assistance with statistical analyses. The Psychology Department of the Ottawa General Hospital was very generous in giving me time to

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1

The Efficacy of Hypnotic and Nonhypnotic Response-based
Imagery Training for the Self-Management of Recurrent Headache

Introduction

The use of cognitive strategies as a primary form of treatment for recurrent headache has only become established in this decade. To date, little empirical research (that utilized adequate experimental controls) has been conducted to assess the efficacy of specific cognitive strategies. Relevant empirically-based findings have been primarily derived from two sources. The first source has been clinical outcome studies that employed general cognitive/cognitive-behavioral treatments--e.g. stress inoculation training that involved positive self-statements, cognitive re-appraisal, and abbreviated relaxation (Turk, Meichenbaum, & Genest, 1983). Experimental pain studies have provided the second source of evidence for the pain-attenuating effects of cognitive strategies.

This dissertation research assessed the efficacy of a specific cognitive strategy, response-based imagery (Lang, 1977; 1979), in facilitating the self-management of recurrent headache. This intervention taught subjects to become imaginatively involved in attending to sensory and behavioral response dimensions of pain-attenuating imagery. The treatment protocol included hypnotic and nonhypnotic training modalities.

Response-based imagery training was examined in two experiments in order to replicate and extend supportive findings from its previous application with migraineurs (Brown, 1984). The

present study included subjects who presented symptoms of chronic migraine headache (CMH), chronic tension headache (CTH), or Mixed CMH/CTH headache. A primary interest was to assess whether treatment-induced reductions in headache activity would be observed with these headache groups. A second investigative interest concerned whether treatment-induced reductions in headache activity significantly covaried with pre- and post-treatment illness attributions regarding recurrent headache as a "health problem" per se. Finally, it was of interest to assess the relation between treatment-induced headache reduction, hypnotizability, and the trait propensity to engage in vivid imagery.

Demographics of Recurrent Headache

Recurrent headache is likely the most common form of benign, episodic pain. Epidemiologic studies of the general population in North America and Northern Europe have demonstrated that 62 to 67% of those sampled experienced at least one headache per month (Goldstein & Chen, 1982; Leviton, 1978). From these surveys, 9.8 to 16.7% of males and 20.8 to 26.6% of females identified their headache symptoms as severe in nature. Blanchard and Andrasik (1985) have estimated that a potential headache problem exists in 7.5 to 15% of men and 15 to 30% of women, due to the frequency or perceived severity of headache activity. The incidence of new cases per annum of severe, recurrent headache is approximately 1% of the general population (Goldstein & Chen, 1982).

Interestingly, university students report recurrent headaches

at a frequency that is at the upper limit of activity estimated for the general population. Controlled studies have found that headaches occurring once or twice per week are prevalent in approximately 26% of males and 31% of females (Andrasik, Holroyd, & Abell, 1979; Antanasio & Andrasik, 1987). These investigations have also found that about 8% of undergraduate males and 14% of undergraduate females experience headaches three to four times per week.

A clear association has not been discovered between recurrent headache and most potential risk factors (Goldstein & Chen, 1982; Leviton, 1978). For example, there has been a failure to observe a significant relationship between recurrent headaches and socioeconomic status, intelligence, educational background, and marital status. Investigations into the relationship between recurrent headache and genetic factors (e.g. positive family history, or incidence in monozygotic vs. dizygotic twins) or hormonal factors (e.g. menarche, or menstruation) have yielded contradictory results.

The sex ratio trend of headache prevalence across the life span has not been investigated in controlled longitudinal or cross-sectional studies. Nevertheless, clinical reports typically describe this disorder as beginning in the second or third decade of life, and diminishing in frequency and severity in later life (Blanchard & Andrasik, 1985; Ziegler, 1984). Sex differences in the prevalence of recurrent headaches do not exist in pre-pubescent children, yet a higher prevalence of headaches among females is

evident in adolescent and adult populations. Headache activity is more frequently reported by females, and this is particularly the case when headaches are self-defined as severe or disabling in nature (Goldstein & Chen, 1982; Leviton, 1978). Related research has indicated that approximately 75% of persons who are reported as seeking treatment for recurrent headache are female (Holroyd, 1986). This extraordinarily high proportion of female-to-male patients does not reflect the sex ratio trend in headache prevalence that was reported in the epidemiologic studies referenced above. The high proportion of female headache patients may be attributable to any of the following: (a) different health care utilization patterns in response to headache symptoms, among males and females; (b) a predisposition among physicians to use differential diagnoses for headache-related symptoms presented by males; or (c) a bias to interpret non-specific, stress-related symptoms as essentially a headache disorder, when presented by females.

Diagnostic Classification of Recurrent Headache

The most influential classification schema for headache diagnosis was developed in 1962 by the American Medical Association (Ad Hoc Committee on Classification of Headache, 1962). Fifteen distinct headache types were formulated, with Vascular Headache of Migraine Type (Classification 1) and Muscle-Contraction Headache (Classification 2) being the most common.

The Ad Hoc Committee's schema of vascular headaches is

subdivided into five categories. Of these, the most prevalent are Classic and Common Migraine, with approximately 85% being diagnosed as the later (Williamson, 1981). The features of Classic Migraine include the following: throbbing pain that is unilateral at onset; pain location in the temporal, orbital, supraorbital, or occipital areas of the head; headache frequency ranging between two to three per week to less than one per year. Related symptoms can include nausea, emesis, anorexia, photophobia, constipation or diarrhea, as well as prodromes such as scotoma, flashing lights, fortification spectra, vertigo, and parasthesia of the face or hands. Common Migraine includes the same features as Classic Migraine, with the exception that no prodromes are present and the pain is less often unilateral.

Muscle-Contraction Headache was defined by the Ad Hoc Committee as a bilateral, bandlike ache that was also commonly manifested in the suboccipital area. It was associated with sustained contraction of the skeletal muscles, and as such, it was interpreted as a reaction to life stress (Ad Hoc Committee, 1962).

The Ad Hoc Committee's classification schema has been considered inadequate on conceptual and empirical grounds. For example, Dalessio (1984) suggested that the 15 hypothesized headache classifications are not organized into a manageable schema, and consequently amount to a series of disparate syndromes of limited clinical utility. Similarly, Martin (1985) argued that the Ad Hoc Committee failed to classify headache diagnoses on the

basis of the alleged criteria of identifying diagnosis-specific pain mechanisms. In fact, some headache groups were diagnosed primarily on the basis of presenting symptomology, as opposed to an empirically substantiated pain mechanism--e.g. Classification 1 (Vascular Headache of Migraine Type) and 2 (Muscle-Contraction Headache). Moreover, Classification 5 (Headache of Delusional, Conversion, or Hypochondriacal States) was diagnosed with neither an hypothesized pain mechanism nor a specified symptomology (Martin, 1985).

The need for a new headache classification schema has been widely recognized. A new "provisional taxonomy" of chronic pain diagnoses was recently presented by the International Association for the Study of Pain (IASP: Merskey, 1986). This taxonomy offers many advantages over the Ad Hoc Committee's schema in regard to its potential utility as an aid to research. Most noteworthy is the fact that the IASP utilized the following multiaxial system: Axis 1, region or site of pain; Axis 2, systems involved with the pain (e.g. nervous system, musculoskeletal system, etc.); Axis 3, temporal characteristics of the pain episode; Axis 4, the patient's perception of pain severity and chronicity; and Axis 5, proposed etiology for pain. Each axis is not required to be coded in making a diagnosis. In addition, characteristics coded under one axis need not be presumed to be causally linked to phenomena reported on other axes (Merskey, 1986). This feature is particularly valuable insofar as the IASP's multiple, independent axes rely more directly

upon specified symptomology, without necessarily implying etiological mechanisms that may be empirically unfounded.

In spite of the potential advantages of the IASP taxonomy, its schematic organization of headache classifications amplifies the theoretical shortcomings found in the original Ad Hoc Committee's classification. For example, the IASP taxonomy virtually disregards an abundance of empirical evidence indicating that chronic muscle contraction and migraine headaches are not differentiated by distinct psychophysiologic profiles (e.g. Andrasik, Blanchard, Arena, Saunders, & Barron, 1982; Kroner, 1983; Ziegler, Hassanein, & Hassenein, 1972). This taxonomic plan also misrepresents the prevailing, empirically-based, opinion that CTH is not distinctly associated with psychological stress (Bakal, 1982; Haynes, Cuevas, & Gannon, 1982; Philips, 1978; Pikoff, 1984). The IASP still defines CTH in terms of bilateral pain that is "associated with muscle tension, anxiety and 'depression'" (Merskey, 1986, p. S58). This definition is conceptually unclear, insofar as it does not clarify how pain, muscle tension, and anxiety are associated. More importantly, a pejorative interpretation of CTH follows from its subclassification into a category of headaches with an hypothesized psychological (i.e. non-physiological) etiology. Included in this subclassification are Headaches of Delusional or Hallucinatory Pain, as well as Hysterical or Hypochondriacal Pain. This diagnostic grouping undermines the advantages of the multiaxial system because it

presupposes the validity of inferred psychodynamic processes-- independent of a specified set of presenting symptoms or an empirically substantiated etiology.

In the final analysis, the IASP taxonomy is self-defined as provisional in nature. The onus is obviously upon the IASP to better specify what diagnostic guidelines are formulated from empirically-derived findings, clinical observations, and psychodynamically-based inference. It also remains the responsibility of investigators to develop alternate diagnostic schemas that will validly and reliably discriminate between various headache phenomena.

Psychophysiology of Recurrent Headache

It was noted in the previous section that the diagnostic representations of recurrent headache developed by the Ad Hoc Committee and IASP are not substantiated by evidence drawn from investigations into CTH and CMH. This section will present a detailed summary of research findings regarding the hypothesized distinction between CTH and CMH. Symptom features, etiology, and precipitants of CMH and CTH will be reviewed.

Symptom Features

One of the primary difficulties in conducting headache research is in determining appropriate screening criteria that will ensure homogenous headache symptoms within diagnostic groups. The current practice, as recommended by Blanchard and Andrasik (1985) is to diagnose CMH, CTH, and Combined CMH/CTH according to criteria listed in Table 1.

Table 1

Diagnostic Criteria for Recurrent Headache (Blanchard & Andrasik, 1985)

CMH	CTH	Combined CMH/CTH
1 headache per month and 3 of the following:	3 headaches per week and 2 of the following:	Presence of both of the following:
I. Unilateral onset of CMH	I. Bilateral onset of CTH, beginning in occipital, suboccipital, or back of the neck	I(a). Subject identifies two distinct types of headache I(b). Subject meets criterion for CMH & CTH
II. Pain is throbbing or pulsating	II. Pain is a continuing "dull ache"	
III. Independent diagnosis of CMH by physician	III. Independent diagnosis of CTH by physician	
- - - - -	- - - - -	- - - - -
IV. CMH accompanied by nausea and vomiting - photophobia during headache	IV. Sensation of tightness or external pressure on head, and/or "cap" or "band" around head	
V. One or more 1st degree relatives diagnosed with CMH		
VI. Headache preceded by visual changes, hemiparesthesias, transient hemiparesis, or speech difficulty		

It is well established that there is significant overlap of symptom features across diagnostic groups, as well as considerable within-group variability on presenting symptomology (Bakal & Kagonov, 1977; Blanchard & Andrasik, 1985; Kroner, 1983; Takeshima & Takahashi, 1988; Zeigler et al., 1972). This fact was particularly well illustrated by Kroner's (1983) attempt to derive distinct headache syndromes through factor analytic techniques. She also assessed the concordance between medically diagnosed and empirically derived classifications.

Kroner administered a comprehensive questionnaire on headache symptoms to 302 patients who were medically diagnosed as having either CMH ($n = 86$), CTH ($n = 85$), Combined CMH/CTH ($n = 65$), or Headache-free status ($n = 66$). A principal component analysis with orthogonal rotation was performed on 52 items from the 90-item questionnaire. The variables assessed comprised the following symptom features: quantitative properties of headache activity (e.g. duration and intensity); qualitative features (e.g. localization of pain, and various pain sensations); presence of muscular symptoms related to headaches (e.g. sensitivity to pressure); relevant neurological and autonomic phenomena preceding or accompanying headaches (e.g. scotoma and vertigo); possible somatic disturbances (e.g. allergies and infections); as well as exposure to precipitating social factors.

A two-factor solution (representing a migraine and a tension headache syndrome) was obtained from Kroner's analysis, and subsequent confirmatory analyses supported this solution. In

keeping with the objective of obtaining a simple factor structure, thereby differentiating distinct headache syndromes, factors 1 and 2 were virtually uncorrelated ($r = 0.05$). The proportion of total symptom variance accounted for by the two factors was very small. Factors 1 (migraine) and 2 (tension) accounted for only 7.4% and 6.7% of the total variance, respectively.

Next, Kroner derived diagnostic-specific rating scales from the screening questionnaire. Symptom features with factor loadings ranging between .38 and .64 were selected to form 9-item migraine and tension headache scales. The migraine scale included items reflecting autonomic and sensory disturbance (e.g. nausea, unilateral pain, less than 3 headaches per week). Muscular symptoms comprised the tension headache scale (e.g. sensitivity to pressure in neck or shoulders, tense neck muscles, more than three headaches per week). Kroner then analyzed the concordance between derived scale values with medically diagnosed migraine and tension headache syndromes. The findings were statistically significant: migraine-- $\chi^2_2 = 70.23$ ($df = 3$), $p = 0.01$; tension-- $\chi^2_2 = 28.84$ ($df = 3$), $p = 0.01$. A discriminant function analysis assessed the degree to which medical diagnoses could be predicted from the two empirically derived headache scales. Kroner (1983) reported that on average 47.2% of medically diagnosed patients were correctly classified. The correspondance between empirically derived and medically diagnosed groups was as follows: migraine, 55.0%; tension, 52.6%; combined, 39.1%; headache-free status, 36.4%.

There are two important issues to be gleaned from the research into symptoms of recurrent headache. First, in the words of Blanchard and Andrasik (1985, p. 13) there are no "pathognomonic symptoms" for CMH, CTH, or Combined CMH/CTH. Kroner's (1983) reported level of 52 to 55% agreement on empirically vs. medically diagnosed migraine and tension headache is comparable to the range of concordance reported elsewhere (Blanchard & Andrasik, 1985). This range is typically found to be statistically significant. Nevertheless, it is clinically unsatisfactory that only slightly more than 50% of medically diagnosed migraine and tension headache patients manifest empirically distinct symptom clusters.

The second issue concerns the limited utility of traditional symptom profiles for recurrent headache. Blanchard and Andrasik (1985) claim that traditional diagnostic criteria are valuable in predicting differential rates of response to some forms of treatment. They report that relaxation training is more effective for subjects with CTH, as compared to subjects with Combined CMH/CTH. They further note that diagnostic reliability increases significantly when extensive (60 to 90-minute) clinical interviews are used in place of succinct, empirically-based questionnaires. These assertions call attention to the fact that the utility of a diagnostic schema is determined by criteria pertaining to both reliability and validity. While the headache diagnoses appear to be reliable, the validity of the traditional symptom profiles

remains questionable. The contention that treatment outcome is predicted by the major headache diagnoses presupposes that this schema reflects qualitatively distinct (vascular vs. muscle tension) phenomena. The argument by Blanchard and Andrasik is premised on the unlikely supposition that a specific component in relaxation training acts on a pathophysiologic mechanism that is unique to CTH vs. Combined CMH/CTH patients. This inference disregards previously noted evidence that there is considerable overlapping symptomology between, and variable symptoms within, CTH and CMH diagnostic groups. In short, the empirical evidence argues against using traditional diagnostic profiles to differentiate between recurrent headache phenomena. Alternate representations of headache symptoms have been designed on the basis of quantitative vs. qualitative distinctions regarding headache severity (e.g. Bakal, 1982; Featherstone, 1984; Takeshima & Takahashi, 1988). Nevertheless, no current schematic classification of recurrent headache symptoms has received more than qualified support.

Etiology

Owing to independent research findings from the late 1930's to 1950's, CMH and CTH were considered to have system-specific etiologies, that were manifest in distinct symptom features (Andrasik, Blanchard, Arena, Saunders, & Barron, 1982; Pikoff, 1984). The traditional view of CMH as primarily a vascular phenomenon, and CTH a myogenic phenomenon, was generally accepted until the early 1980's.

The Ad Hoc Committee (1962) inferred that the tension/muscle contraction headache was symptomatic of a temporary or sustained reaction to life stress. The specific pain mechanism was psychosomatic in the classical sense. That is, the persistence of bilateral, bandlike pain was considered a primary manifestation of sustained contraction of skeletal muscles in the face, neck, or shoulders. The muscular contraction was in turn attributed to the individual's reaction to life stress. In labelling this phenomenon as a Muscle Contraction Headache the Ad Hoc Committee recognized that the inferred psychophysiologic etiology also led to the "ambiguous and unsatisfactory terms of 'tension', 'psychogenic', and 'nervous' headache" (Ad Hoc Committee, 1962, p. 718). The diagnostic label of Muscle Contraction was preferred on the basis of the assumed validity of the muscular etiology. The IASP taxonomy (Merskey, 1986) essentially supports this etiologic profile.

The classification schemas developed by the Ad Hoc Committee (1962) and IASP (Merskey, 1986) support the hypothesis of a stress-related vascular etiology for migraine. This notion implies that vasoconstriction of intra- and extracranial arteries leads to cerebral ischemia, that in turn causes sensory and neural disturbances (e.g. visual scotomata, nausea, etc.). During the migraine attack there are paroxysmal vascular changes that can be observed as corresponding to a unilateral site of pain--e.g. the cutaneous branches of the external carotid artery (Saper, 1986). The eventual onset of migraine pain is attributed to a vasodilation

phase. It is still commonly believed that the vasodilation is accompanied by (a) the release of some pain-related vasoactive substance, such as platelet serotonin, as well as by (b) the dysfunction of the brain stem pain modulating system (Holroyd, 1986). While the actual pain mechanism for migraine headache has not been identified recent research indicates that vascular symptoms are secondary to a neuronal disturbance. For example, Saper (1986) favorably reviewed current investigations into the hypothesis that migraine disturbance begins within the hypothalamus, and extends to the midbrain via the mammillothalamic tract. Specifically, the range of migraine pain and related vascular symptoms are hypothetically linked to the periaqueductal gray region of the midbrain and the locus ceruleus of the pons and medullary system.

Research over the past decade has presented a substantial amount of data that contradict the etiological accounts of the Ad Hoc Committee and IASP regarding CMH and CTH. Numerous reviews of research have concluded that the relationship between recurrent headache activity and either muscle tension (frontalis, trapezius, temporalis, and/or neck) or cerebral blood flow is by no means direct or clear. There is considerable empirical support for the following conclusions. First, CTH subjects do not exhibit significant elevations in muscle tension during headache vs. non-headache periods (Haynes, Cuevas, & Gannon, 1982; Pikoff, 1984). Second, CTH subjects do not show significantly higher EMG levels

during baseline biofeedback procedures or exposure to stress-inducing conditions, in comparison to migraine and/or controls (Bakal, 1982; Haynes, Cuevas, & Gannon, 1982; Philips, 1978; Pikoff, 1984). Third, the elevation of muscle tension does not covary directly with subjective ratings of pain (Philips, 1978). Fourth, the cerebral blood flow to the sensorimotor cortex does not directly correspond with focal cerebral symptoms ascribed to that area, and headache onset is not significantly associated with increased cerebral blood flow (Olesen, Tfelt-Hansen, Henriksen, & Larsen, 1981; Olesen, Lauritzen, Tfelt-Hansen, Henriksen, & Larsen, 1982).

A study by Andrasik, Blanchard, Arena, Saunders, & Barron, (1982) was particularly thorough in its investigation of potential etiological factors for recurrent headache. Subjects who met diagnostic and screening criteria comprised one of three headache groups--tension ($n = 62$), migraine ($n = 39$), or combined CMH/CTH ($n = 37$). These 138 subjects were matched with a control group ($n = 57$) on age, sex, income, marital status, and educational level. All subjects were exposed to a biofeedback training procedure where they were tested on a series of self-control abilities (relaxing the body, hand warming, and relaxing the forehead), as well as their response to stressors (mental arithmetic, pleasant imagery, stressful imagery, and cold pressor). Throughout this procedure six physiological measures were taken: frontalis and forearm EMG, cephalic vasomotor response, heart rate, skin resistance level, and hand temperature.

The data analysis conducted by Andrasik et al. initially examined group differences on baseline physiological data by separate One-Way analyses of variance (ANOVAs). All ANOVAs were nonsignificant. The next analyses turned to the self-control performance trials and stress response trials. Each of the physiological measures served as the dependent variable in separate Group (CMH/CTH/Mixed/Control) X Trials (Self-control trials/Stress-response trials) split plot ANOVAs. There were no clinically meaningful patterns in these data. For the self-control and stress-response analyses, significant main effects for the Trials factor were often the only significant finding. In cases where a Group X Trials interaction was reported, post hoc procedures often could not identify the source of the interaction (e.g. frontalis EMG in stress response testing, and skin resistance in self-control testing). Other Group X Trials interactions were not attributable to clinically meaningful differences. For example, a Group X Trials interaction was found on the cephalic vasomotor response in the self-control testing. Simple main effects and post hoc tests revealed that during the warm hands condition, the CMH group dilated proportionately more than the combined tension/migraine group, but not more than controls or the CTH groups. No significant main effects for diagnostic group were found in either the self-control performance or stress response data.

Andrasik et al. also conducted a discriminant function analysis to assess the predictive ability of the physiological measures and experimental conditions in classifying diagnoses. Accurate classification was evidenced in only 40.9% of the cases (significance level was not reported), and this finding was not interpreted as being clinically meaningful. It was concluded that the absence of systematic psychophysical differences between diagnostic groups strongly pointed out "the futility of seeking a unidimensional, physiological-response-based etiology to chronic pain problems" (Andrasik, Blanchard, Arena, Saunders, & Barron, 1982, p. 425).

Precipitants

The popular view that CMH and CTH are precipitated by maladaptive reactions to life stress (Ad Hoc Committee, 1962; Merskey, 1986), reflects an early sensory paradigm that dichotomized pain experience into two components: pain sensation and normal/abnormal reaction (Melzack, 1983). The Ad Hoc Committee officially placed CMH and CTH in a subclassification, under the rubric of "temporary or sustained difficulties in life adjustment" (Ad Hoc Committee, 1962, p. 727). Other diagnoses in this subclassification included headache of delusional, conversion, or hypochondriacal states. As noted earlier, the IASP's provisional taxonomy (Merskey, 1986) echoed the Ad Hoc Committee's profile of CMH and CTH. Two areas of theoretical-investigative emphasis were associated with these psychogenic headaches. Since these phenomena

were viewed as being precipitated by psychological (vs. physiological) factors, individuals with CMH and CTH were considered to have a headache-prone personality that was comprised of distinct dysfunctional traits. In addition, the depiction of CMH and CTH as disorders that were precipitated by life stress, reflected the belief that these headache phenomena were best understood as maladaptive stress-reactions.

A wide range of personality traits have been purported to precipitate CMH and CTH. Individuals with CMH have been characterized as compulsive, rigid, and perfectionistic, while those presenting with CTH were allegedly unhappy, burdened with psychological problems, lacking in self-insight, and consequently depressed and hostile (Diamond & Dalessio, 1978; Edmeads, 1980). Theories attributing recurrent headache to dysfunctional personality traits have been validly critiqued on theoretical and methodological grounds. Philips (1977) was one of the first to argue that trait assessments of predisposing causal agents are confounded by the consequence of pain chronicity. Elevated levels of anxiety or compulsiveness in recurrent headache groups can be attributed to the degree to which pain chronicity has had an impact on the individual's daily life. Additionally, empirical studies have failed to discriminate between diagnostic groups of headache patients on the basis of personality traits (Andrasik, Blanchard, Arena, Teders, Rodichok, 1982; Sternbach, Dalessio, Kunzel, & Bowman, 1980; Rappaport, McAnulty, Waggoner, & Brantley, 1987).

These investigators instead reported a significant relationship between psychopathology and frequency of headache activity, regardless of diagnosis. More recently, Dieter and Swerdlow (1988) demonstrated that items purporting to assess psychopathology on the MMPI were confounded with actual headache symptoms. Once these overlapping headache symptom/psychopathology items were removed from the patient's HS, D, and HY scales, mean ratings on the conversion "V" profile were no longer clinically elevated. Research endeavors aimed at linking personality traits with specific headache diagnoses were curtailed significantly over this decade, due to the recognition of inherent theoretical and methodologic confounds.

The demonstration of the stress-response perspective as a valid theory of recurrent headache activity (intensity, frequency, duration, etc.) requires the demonstration of at least three empirical points. First, it must be shown that CMH or CTH is in fact a response to identified stressors. Second, it must be demonstrated that CMH and CTH subjects exhibit a uniform and sustained reaction that is distinct from non-headache controls, or from other diagnostic groups. Third, the issue of a causal relationship between stress and recurrent headache must be established through prospective (vs. retrospective) experimental designs, and these must assess the subjects' response to natural stressors. This third issue entails the difficult task of controlling for influences that bias the subjects' report of stress

reactivity--most particularly, the length of chronicity of the subject's recurrent headache activity.

The two initial empirical issues noted above (regarding recurrent headache activity in CTH and CMH subjects as a distinct stress response) have only begun to be assessed in controlled experimental research. To date, only one published study has examined whether headache onset is a function of exposure to a sustained stressor in a controlled experimental setting (Gannon, Haynes, Cuevas, & Chavez, 1987). Twenty-four subjects (18 to 42 years) were selected for this investigation. Diagnostic headache groups were evenly represented, with eight subjects (3 males and 5 females) in the CMH, CTH, and Non-headache groupings. The investigators attempted to induce and facilitate headache activity by exposing subjects individually to a one-hour cognitive stressor. For this purpose, arithmetic problems were posed every 15 seconds, and a 60 dB buzzer was sounded 22 times on a predetermined schedule. All subjects were informed that the buzzer was a cue that signalled when their performance fell below the group average. Gannon et al. attempted to control for the intrinsic situational demand of eliciting reports of headache onset by informing subjects that the study was assessing physiological responses under various conditions, and that a headache might or might not be experienced in the session. All subjects were required to be headache-free at the beginning of their session.

The stress reactions of subjects were measured by monitoring five physiological variables: heart rate, blood-volume pulse amplitude, frontal EMG, neck EMG, and forearm EMG. Data points comprised one-minute averages that were assessed at the following times: at the end of a 15-minute baseline period; during the final minute of each ten-minute segment in the 60-minute exposure to stress; and during a ten-minute recovery, at the initial minute and subsequently every other minute. Subjects were required to rate their level of headache activity on a subjective rating scale of 0 (no headache) to 10 (extremely painful headache). Headache ratings were taken immediately prior to and following the baseline assessment, at the end of each 10-minute segment during the 60-minute stress exposure, and at the end of the 10-minute recovery period.

Two sets of analysis were of prime theoretical interest in the Gannon et al. study. First, it was important to determine if CMH or CTH subjects exhibited a significant relationship between stress exposure and headache onset. Once subjects were screened for absence of headache activity with the pre-baseline assessment, a 3 (Group: CMH/CTH/Non-Headache) X 8 (Interval: time intervals for stressor) repeated-measures ANOVA was conducted on the headache activity ratings. Nonsignificant findings were reported for the Group X Interval interaction, as well as for the main effect for the Group factor. This indicated that CTH and CMH subjects did not exhibit a level of stress-induced headache activity that was

significantly higher than that reported by the Nonheadache Control group. Interestingly, a significant main effect was noted for the Interval factor. Post hoc comparisons for the time increments on the Interval factor were not given. Nevertheless, a graph of these data indicated that subjects reported a monotonic increase in headache activity over the course of stress exposure. The Gannon et al. study did, therefore, induce headache activity among subjects. CTH and CMH groups were not significantly more reactive in comparison to non-headache controls.

The second analysis of theoretical interest concerned whether there was physiologic evidence to support a diagnosis-specific stress reactivity profile. A 3 (Group: CMH/CTH/Non-Headache) X 2 (Headache State: Headache/Non-headache activity) X 12 (Interval: time intervals for stressor) between-within MANOVA was reported, using the five above-noted physiologic measures as dependent variables. This analysis produced a significant Group X Interval interaction and a significant Group X Headache State interaction. The multivariate Group X Interval interaction was due only to neck EMG-- $F(22,186) = 1.94, p < .01$. The CMH group exhibited a higher level of neck EMG during the six ratings of stress exposure, which runs contrary to the common assumption about a diagnosis-specific (vascular vs. myogenic) stress-reactivity pattern in migraineurs. Subsequent analyses for the multivariate Group X Headache State interaction failed to uncover either statistically significant or clinically meaningful differences on the univariate measures.

The findings reported by Gannon et al. (1987) strongly suggested that the relationship between recurrent headache and experimentally sustained stress is complex, at best. A stress-related response-based etiology for headache activity was disconfirmed by at least two findings: (a) the absence of a significant relationship between stress reactivity and pronounced headache activity among CMH and CTH subjects; and (b) the absence of a diagnosis-specific pattern of physiologic reactivity to stress.

Some research has addressed the third previously noted empirical issue regarding the nature of the relationship between recurrent headache and reactivity to natural stressors. Bakal and associates challenged the stress-response theory by noting that reported headache activity typically occurs at a time of day that precedes meaningful exposure to natural stressors. Bakal, Demjen, and Kaganov (1981) examined pain diaries of 45 subjects with CMH, CTH, or Combined CMH/CTH, and from these data approximately 60 to 75% of the headache activity began between 6:00 a.m. and 10:00 a.m.. Demjen and Bakal (1986) similarly reported that 43% of headaches began prior to 10:00 a.m., among 44 subjects with CTH, CMH, or Combined CMH/CTH diagnoses. Furthermore, Demjen and Bakal (1986), found a significant positive correlation between the proportion of daily headache activity occurring in the early morning, and the overall percentage of distressed thoughts about headaches ($r = 0.31$, $p < .05$). It was reasoned that these findings were

inconsistent with the assumption that headache attacks are a response to situational stressors, that would presumably have elicited typical onset of headache activity in the later hours of the day.

Further to the third noted empirical issue, Holm, Holroyd, Hursey, and Penzien (1986) used numerous operational definitions of perceived stress to study the relationship between recurrent headache activity, stress, and adaptive/maladaptive coping strategies. They administered a questionnaire battery in which inventories of major life events (Cochrane & Robertson, 1973), as well as daily hassles (DeLongis, Coyne, Dakof, Folkman, & Lazarus, 1982) were analyzed for event-related cognitive appraisals (adapted from Hammen & Mayol, 1982) and stress coping strategies (adapted from Lazarus & Folkman, 1984). Ninety-seven CTH subjects (57 females and 40 males) were selected from a pool of 1486 university students. These subjects were matched with 177 headache-free controls, according to age and gender. The mean age was 19 years.

Among the findings noted by Holm et al., univariate tests indicated that CTH subjects reported a greater number of major life events-- $F(1,247) = 4.0, p < .05$ --and daily hassles-- $F(1,247) = 17.6, p < .001$ --as compared to controls. The CTH group also exceeded controls in their mean rating of the undesirability and perceived impact of stressful life events-- $F(1,247) = 10.1$ and 4.3 respectively, $p < .01$ in both cases. Additionally, CTH subjects rated their daily hassles as being more severe, in comparison to

control subjects -- $F(1,247) = 18.7, p < .001$.

Holm et al. also investigated the relative efficacy of coping strategies for self-reported stressful events. The CTH and control group differed significantly on self-reported prevalence with which they used ineffective problem-focused and emotion-focused (or palliative) coping strategies. Univariate analyses on group means for stress coping strategies indicated that CTH subjects made greater use of avoidance and self-criticism, and less use of social contact, in comparison to non-headache controls-- $F(1,254) = 5.55, 5.45, \text{ and } 4.72, \text{ respectively, } p < .05$ in all cases.

The Holm et al. (1986) study presents an important preliminary effort in determining whether recurrent headache subjects exercise maladaptive stress coping responses, in comparison to headache-free controls. These investigators correctly emphasized that their data did not clarify the issue of whether stress-related appraisals and coping behaviors elicited the problem of recurrent headache. These characteristics may have been a consequence of chronic exposure to recurrent pain. This possibility is empirically supported by the finding that social-cognitive functioning is negatively affected by pain chronicity (Philips & Jahanshahi, 1985). Further to this point, negative affect among CMH and CTH subjects is observed as being (a) significantly higher on headache (vs. headache-free) days, and (b) uniquely correlated with "same-day" headache intensity, but uncorrelated with "day-before" or "day-after" intensity ratings (Martin, Nathan, Milech, & van Keppel, 1988).

This suggests that cognitive-affective activities of recurrent headache subjects reflect subjective distress from acute headache episodes, rather than a stable maladaptive coping pattern.

In sum, the empirical evidence strongly contradicts traditional views of CMH and CTH precipitants that have been supported by the Ad Hoc Committee (1962) and IASP (Merskey, 1986). Though it appears illusory to believe that CMH and CTH are etiologically distinct phenomena that are precipitated by stress, these views continue to function as the governance for clinical activities. The stress-response theory of recurrent headache is essentially simplistic, and it is not empirically supported. It overlooks the likelihood that assessments of stress appraisals of daily events are confounded with the stress-inducing impact of pain chronicity among CMH and CTH subjects. Instead, the nature of the relationship between stress-inducing events and recurrent headache activity is likely complex, rather than directly causal.

Treatments for Recurrent Headache

Although it is conceptually arbitrary and empirically unfounded to assume that Migraine, Tension, and Mixed (Migraine/Tension) diagnoses represent qualitatively distinct headaches (Martin, 1985), these classifications remain prevalent in treatment research. The use of these diagnoses continues to be popular largely because CMH, CTH, and Mixed CMH/CTH subjects are purported to respond differently to pharmacologic (Edmeads, 1980; Graham, 1985) and nonpharmacologic (Blanchard & Andrasik, 1985)

interventions. As previously noted, Blanchard and Andrasik (1985) based their support for the traditional diagnostic schema on the observation that relaxation training was effective among twice as many CTH patients as compared to Mixed patients. This argument does not establish the utility of evaluating treatments according to response rates of CMH vs. CTH vs. Mixed subjects. It presupposes rather than demonstrates that treatment outcome is directly attributable to changes in diagnosis-specific pathophysiology. Alternate diagnostic schemas have been proposed in chronic pain treatment research (e.g. Philips, 1978; Takeshima & Takahashi, 1988; Turk & Rudy, 1988). No specific schema has received wide acceptance at this time. This review, therefore, will follow the convention of discussing treatment efficacy as it applies to recurrent headache subjects diagnosed in the traditional categories.

It is beyond the scope of this section to provide an in-depth review of psychophysiologic evidence relating to proposed therapeutic mechanisms in recurrent headache treatments. Instead, the first subsection will examine the effectiveness of pharmacologic treatments for recurrent headache. The subsequent subsection will provide a detailed review of major nonpharmacologic treatments. Cognitive factors that are purported to mediate therapeutic outcome in the nonpharmacologic approach (e.g. efficacy expectations, relaxation ability, hypnotic susceptibility) will be critically examined. The second subsection will also contrast the

cognitive-behavioral and operant paradigms of nonpharmacologic treatments.

Pharmacologic treatment

Pharmacologic treatments for CMH and CTH are designed to manage the pain and anxiety experienced from acute headache episodes, and to prevent the onset of headache activity. Treatment protocols have been developed with a belief in the system specificity of pharmacologic actions (i.e. either vascular or muscular). Nevertheless, previous sections of this dissertation demonstrated that the weight of evidence contradicts the specificity hypothesis of headache etiology. Saper's (1986) review of neurophysiologic mechanisms in headache activity suggested that the variety of headache medications induce unified pharmacologic actions. Saper noted that pharmacologic treatments primarily influence central brain mechanisms, and secondarily produce effects on intra- and extra-cranial vasculature.

CMH or Mixed headaches are treated by a considerable range of pharmacologic strategies (for review see Edmeads, 1980; Saper, 1986; Ziegler, 1984). These include: nonnarcotic analgesics (e.g. acetaminophen); narcotic analgesics (e.g. fiorinal, percodan, or ASA with diazepam or codeine); ergot-type/abortive medications (e.g. ergotamine, caffergot); and prophylactics (e.g. bellergal, sandomigran, sansert). Alternative prophylactic medications include beta blocking agents (e.g. propranolol) and either anxiolytics or antidepressants (e.g. phenobarbital or amitriptyline).

Recent investigations into the utility of migraine-specific vascular agents are represented by three conclusions. First, traditional migraine medications (e.g. ergotamine, amitriptyline, and beta blockers) are equally effective for forms of chronic pain that are not considered to be essentially vascular in nature, such as CTH, low back pain, and multiple sclerosis (Saper, 1986). Second, experiments using adequate controls (e.g. double-blind crossover design) have found that medications with vasoactive properties (e.g. ergotamine) were not more effective than mild analgesics in reducing the CMH symptoms of pain, nausea, and emesis (Bakal, 1982). Third, the empirical evidence shows that migraine medications are indistinguishable from the short-term effects of placebos. Short-term reductions in headache activity are predictably followed by an increase, unless there is some change in the patient's prescription (Bakal, 1982).

Pharmacologic therapies for CTH have been offered in the form of analgesics, anxiolytics, and antidepressants (Bakal, 1982). Prescription analgesics such as acetaminophen and propoxyphene have had limited effectiveness in attenuating CTH pain. Anxiolytic medications (e.g. librium and valium) are prescribed for headache-related distress (anxiety). Muscle-relaxation properties are attributed to anxiolytic medication, and its use is intended to interrupt the severity of CTH activity. Antidepressants (e.g. amitriptyline chloride) are used as a prophylactic for CTH. Interestingly, Bakal (1982) reports that nondepressed CTH subjects

have responded better to this medication than depressed CTH subjects.

Evidence is quite mixed regarding the therapeutic value of pharmacologic treatments for CMH, CTH, and Mixed headaches. Pharmacologic investigations have typically compared one active medication with a placebo, thereby preventing an accurate understanding of the relative efficacy of various pharmacotherapies (Bakal, 1982). Additionally, a minimal amount of research has directly compared pharmacologic with nonpharmacologic treatments (Holroyd, 1986). At the present time there is insufficient evidence to make valid generalizations regarding the efficacy of specific pharmacotherapies, or combined pharmacologic-behavioral interventions.

Nonpharmacologic Treatments

The prevailing question in contemporary behavioral treatments for recurrent headache is not whether the variety of procedures are efficacious, but what specific components elicit the positive outcomes. Treatment success from divergent therapies suggests that there may be some unidentified common therapeutic mechanism (Litt, 1986), or that the positive outcomes are attributable to a complex interaction of bio-psycho-social factors.

The most popular nonpharmacologic treatments for recurrent headache have been thermal or electromyographic biofeedback training (T-BFB, EMG-BFB), as well as some form of relaxation training (for review see Blanchard & Andrasik, 1985; Chapman, 1986;

Holroyd & Penzien, 1986; Turner & Romano, 1984). It has also been common to combine a relaxation protocol with BFB training. Meta-analytic studies and literature reviews have found that relaxation training and/or biofeedback are equally efficacious. On average, these treatments have induced significantly greater reductions in headache activity than no-treatment control procedures and pseudotherapies (pharmacologic and nonpharmacologic placebos). For example, Blanchard, Andrasik, Ahles, Teders, and O'Keefe (1980) derived outcome measures for various CMH therapies, based on a mean percentage of headache reduction per treatment mode. The following reductions were noted in treatments of CMH: T-BFB with autogenic training, 65.1%; T-BFB alone, 51.8%; relaxation training, 52.7%; and medication placebos administered in double blind trials 16.5%. A One-Way ANOVA found significant differences in the efficacy of treatment groups-- $F(3,20) = 5.90, p = .0047$. Post hoc comparisons indicated that while there were no significant differences in headache reduction percentages between the behavioral treatments, each behavioral treatment reduced headache activity significantly more than the placebo or nontreatment control conditions. More recently, Holroyd and Penzien (1986) conducted a meta-analysis on behavioral treatments for CTH. They reported the following levels of mean percentage in headache reduction at post-treatment: EMG-BFB, 46%; relaxation training, 44.6%; combined EMG-BFB and relaxation training, 57.1%; noncontingent BFB, 15.3%; and headache monitoring, -3.9%. A one-way ANOVA demonstrated that there were

significant group differences-- $F(4,61) = 14.5, p < .001$. Post hoc analyses confirmed that percentages of headache reduction for the three behavioral treatments were significantly greater than outcomes for noncontingent BFB, and headache monitoring. No differences were found between the three behavioral treatments. Although Placebo conditions have proven to be less efficacious, on average, in comparison to relaxation and/or BFB training, placebo procedures have been utilized more recently with much success. These data will be discussed later in this section.

Empirical findings indicate that behavioral treatments of recurrent headache activity have long-term effects. A five-year follow-up study was conducted by Blanchard, Appelbaum, Guarnieri, Morrill, & Dentinger (1987). These investigators located 26 of 38 headache patients (CTH, CMH, or Mixed) who had significantly reduced headache activity at post-treatment. Treatment had originally consisted of BFB, relaxation training, or a combination of these two modes. Twenty-one of these patients agreed to participate in the follow-up study by completing daily headache recordings over a 4-week period. Among this sample, 78% of CTH subjects and 91% of CMH or Mixed subjects maintained their initial treatment reductions of 50% of pre-treatment headache activity. Similar findings have been reported for this sample at two-, three-, and four-year follow-up intervals (Blanchard, Andrasik, Guarnieri, Neff, & Rodichok, 1987). In addition, prospective studies have found that post-treatment reduction in headache activity was

maintained at 1- and 2-year follow-up intervals, regardless of whether there had been minimal or regularly scheduled therapist contact (Blanchard, Appelbaum, Guarnieri, Neff, et al., 1988).

The above data only provide qualified support for the long-term efficacy of behavioral treatments. Several issues complicate the interpretation of these findings. For example, it is possible that the results of Blanchard, Appelbaum, et al. (1987) would have been substantially altered if the large proportion of missing patients (44.7%) had been included. The conclusion about long-term effects from behavioral treatment therefore only applies to select subjects who were motivated to participate in follow-up research, and who initially reduced headache activity by 50%. Further, the 4- and 5-year outcome studies did not make appropriate comparisons of headache prevalence rates in the treatment vs. control groups, over the long-term. This fact prevents a valid conclusion from being formulated about long-term treatment efficacy. It is necessary to assess the significance of treatment-induced reductions relative to variations in headache activity that would have otherwise occurred over the 5-year period.

Some investigators suggested that generalized relaxation is a common therapeutic pathway that mediates a positive outcome from behavioral treatments (Litt, 1986; Pikoff, 1984). There is no clear empirical support for this hypothesis. In fact, a major shortcoming of this post hoc theory is that it is premised on treatment outcome studies that have not incorporated control

procedures to assess underlying therapeutic processes. In addition, investigations with prospective research designs have failed to support the hypothesis of a direct covariation between psychophysiologic measures of tension/relaxation and headache activity (e.g. Borgeat, Elie, & Larouche, 1985; Gannon et al., 1987; Haynes, Cuevas, & Gannon, 1982).

An issue of major importance that has been inadequately addressed in the headache treatment literature concerns the relative efficacy of pharmacologic vs. behavioral interventions. Only four studies could be located that evaluated these two treatment modalities (Anderson, Basker, & Dalton, 1975; Holroyd, Holm, et al., 1988; Mathew, 1981; Sovak, Kunzel, Sternbach, & Dalessio, 1981). Adequate experimental controls, particularly concerning compliance to therapeutic regimen, were utilized in only one of these investigations--Holroyd, Holm, et al. (1988).

Holroyd, Holm, et al. (1988) assessed the therapeutic effect of abortive Ergotamine medication (Cafergot or Ergostat) vs. combined Relaxation/T-BFB training, among subjects with either CMH or Mixed CMH/CTH. Treatment efficacy was assessed in a 2 (Group: Pharmacologic/Nonpharmacologic) X 2 (Gender) X 4 (Treatment Period: Pre/1st month/2nd month/Post) Mixed MANOVA. The dependent variables were mean daily headache activity, peak headache intensity, and headache-free days. A significant Group X Treatment Period quadratic interaction indicated that the Ergotamine Group reduced headache activity at an earlier Treatment Period-- $F(9,29)$

= 2.39, $p < .05$. The Ergotamine group reported 30% reduction at the 1-month interval, while the Relaxation/T-BFB group showed no headache reduction during that period. These groups also differed significantly in their use of analgesic medications. Friedman's nonparametric 2-Way ANOVA was utilized in a 2 (Group: Pharmacologic/Nonpharmacologic) X 4 (Treatment Period: Pre-treatment/1-month/2-month/Post-treatment) factorial design, with mean daily analgesic consumption serving as the dependent variable. This analysis demonstrated that only the combined Relaxation/T-BFB group significantly reduced their usage of analgesic medication from pre- to post-treatment-- $\chi^2_{(3)} = 12.58$, $p < .01$. In addition, a significantly greater percentage of subjects in the nonpharmacologic condition meaningfully reduced analgesic consumption. A 50% reduction in daily analgesic use at post-treatment was reported in 78% of the Relaxation/T-BFB subjects, as compared to 40% of the Ergotamine subjects-- $\chi^2_{(1)} = 4.89$, $p < .05$. Finally, different features characterized the subjects who showed marked reductions in headache activity (> 50%) in the different treatments. In the Ergotamine group, a pre-treatment assessment of trait anger was significantly correlated with a lack of post-treatment headache reduction-- $r_{(16)} = -.68$, $p < .001$. The Relaxation/T-BFB group were unique in demonstrating a significant correlation between pre-treatment headache-free days and post-treatment reduction in headache activity-- $r_{(17)} = .67$, $p < .001$.

The Holroyd, Holm, et al. (1988) study did not include a control group, it utilized a small sample ($n = 37$), and replication of its results is necessary before firm conclusions can be drawn about pharmacologic vs. behavioral treatments. Keeping this cautionary note in mind, the Holroyd, Holm, et al. data suggested that there are meaningful differences associated with these two treatment modalities. The therapeutic effect of pharmacotherapy was experienced sooner than the behavioral treatment. It was also more effective among subjects who had less of a propensity to become agitated. On the other hand, the behavioral treatment was equally efficacious in comparison to the Ergotamine treatment, and it was particularly effective among subjects who did not experience a high incidence of daily pre-treatment headache activity. The behavioral approach also was uniquely associated with adaptive changes in analgesic medication consumption.

Cognitive-behavioral vs. operant paradigms.

The previous discussion of research into etiology and precipitants of recurrent headache demonstrated that the assumption of a direct relationship between psychophysiologic activity and headache symptoms is not empirically substantiated. The gradual discovery of this fact gave rise to two competing paradigms that have attempted to explain how behavioral treatments are efficacious--the cognitive-behavioral approach (Meichenbaum, 1976; Turk, Meichenbaum, & Genest, 1983) vs. the operant approach (Budzynski, Stoyva, Adler, & Mullaney, 1973). The fundamental

differences between these two paradigms has been cogently demonstrated over the past decade, particularly in regard to experimental manipulations of proposed therapeutic mechanisms.

The operant model pre-dates the cognitive-behavioral account. Adherents to the operant approach have argued that reductions in recurrent headache activity are attributable to the subject's enhanced awareness of, and control over, suspected physiologic abnormalities underlying headache activity. Therefore, behavioral treatment was interpreted as teaching subjects to reduce sustained contraction in their skeletal muscles (e.g. frontalis, trapezius), or vascular constrictions of their cerebral blood flow (Sargent, Green, & Walters, 1972).

The distinguishing feature of the cognitive-behavioral paradigm is the view that therapeutic outcome from behavioral training is mediated by self-regulatory cognitive processes (Lazarus, 1977; Meichenbaum, 1976; Turk, Meichenbaum, & Berman, 1979). For example, EMG-contingent BFB for CTH is deemed to have the potential to enhance self-efficacy expectations, outcome expectations, as well as coping strategies for dealing with headache-related stressors. The revolutionary significance of the cognitive-behavioral paradigm is the attribution of therapeutic outcome to the perception of self-control over stress-related physiologic activity. In keeping with investigations into psychophysiologic concomitants of headache activity, the cognitive-behavioral model accommodates the absence of a direct relationship

between recurrent headache experience and physiologic arousal. The validity of the operant paradigm has been appropriately challenged due to its failure to account for therapeutic outcomes that are independent of actual control over physiologic processes.

Holroyd et al. (1984) schematically presented the fundamental difference between the cognitive-behavioral and operant paradigms in the case of CTH. As shown in Figure 1, Holroyd et al. attribute the effectiveness of EMG-BFB to enhanced self-efficacy and outcome expectations. These are hypothesized to facilitate the development of adaptive (vs. previously maladaptive) coping strategies for managing CTH-related stressors.

Figure 1

Cognitive-behavioral and Operant Models of Biofeedback
 (Holroyd, K.A., Penzien, D.B., Hursey, K.G., Tobin, D.L., Rogers, L.,
 Holm, J.E., Marcille, P.J., Hall, J.R., & Chila, A.G., 1984)

Empirical support for the cognitive-behavioral paradigm has been demonstrated in related investigations by Andrasik and Holroyd (1980; 1983) and Holroyd et al. (1984). Andrasik and Holroyd (1980; 1983) presented compelling evidence regarding the efficacy of perceived self-control in EMG feedback training for 39 CTH subjects, irrespective of actual EMG activity. Subjects were divided into three EMG feedback groups and one headache monitoring group. EMG feedback groups were informed that they were being trained to relax the frontalis muscle over a four-week treatment period. In fact, subjects in these three groups were successfully taught to either increase, decrease, or maintain frontalis tension. Headache activity scores were reported at intervals of pre-treatment, immediate post-treatment, 6-weeks, and 3-years post-treatment.

The mean level of headache activity for each of the three EMG-BFB groups was significantly less than headache monitoring subjects, over the course of each post-treatment assessment interval. It was theoretically important that the experimental manipulations for EMG frontalis tension were effective. Mean EMG microvolt levels at post-treatment were as follows: Decrease BFB, 1.91; No Change, 3.27; and Increase BFB, 10.12-- $F(2,21) = 9.07$, $p < .02$. Planned orthogonal comparisons revealed that post-treatment outcome measures in headache activity for the three EMG groups did not differ significantly. Between 80 to 100% of subjects in the three BFB groups reduced headache activity by at least 50% at post-

treatment and 6-week follow-up. Moreover, at 3-year follow-up the mean headache activity in each EMG feedback group was still reduced by at least 50%, while the monitoring group increased in mean headache activity by 7%. A Group (Increase/Decrease/No Change/Monitoring) X Trials (Pre-treatment/Post-treatment/6-Week Follow-up/3-Year Follow-up) Mixed ANOVA produced a significant interaction on headache activity reports-- $F(9,72) = 2.41, p < .02$. The only significant planned orthogonal comparison, among the simple main effects, was in headache activity between the recording group and three EMG groups combined-- $F(1,96) = 4.97, p < .05$.

Three conclusions need to be drawn from the investigations of Andrasik and Holroyd (1980; 1983). First, it is evident that a therapeutic reduction in headache activity can be produced by enhancing perceived (vs. actual) self-control over select physiologic processes. Second, the enhancement of perceived self-control can produce a long-term therapeutic outcome. This suggests that behavioral treatments that teach operant control over suspected pathophysiologic processes are not necessary for reducing recurrent headache activity. Long-term therapeutic outcomes for T-BFB, EMG-BFB, or relaxation training (Blanchard, Andrasik, et al., 1987; Blanchard, Appelbaum, et al., 1987) might be mediated by unidentified cognitive factors. Third, shortcomings are evident in previous studies that tested the efficacy of BFB training by manipulating contingent vs. non-contingent feedback. Findings from Andrasik and Holroyd (1980; 1983) clearly indicated that any type of

contingent EMG feedback, whether therapeutic or nontherapeutic in design, was sufficient to reduce recurrent headaches over the long term. It remains to be determined if therapeutic and nontherapeutic contingency of other forms of BFB (thermal, vasomotor, etc.) produces similar effects.

Another major source of empirical support for the cognitive-behavioral approach to treating recurrent headache has been presented by Holroyd et al., (1984). These investigators demonstrated the positive effect of outcome and self-efficacy expectations upon EMG feedback training for CTH sufferers. Under the assumption that EMG training was being undertaken to reduce frontalis tension, a 2 X 2 factorial design manipulated EMG feedback (increase vs. decrease) and performance feedback (high vs. moderate perception of success). A four factor repeated measures ANOVA (EMG Feedback X Performance Feedback X Sessions X Trials) on frontalis EMG activity indicated significant interactions for EMG Feedback X Sessions, and EMG Feedback X Trials-- $F(5,170) = 4.6, p < .001$; $F(5,170), p < .002$, respectively. Subsequent analyses confirmed that EMG feedback groups changed frontalis tension in the manipulated directions across Sessions, and across within-session Trials. As predicted by the cognitive-behavioral paradigm, univariate ANCOVAs on post-treatment headache activity (with pre-treatment headache activity as the covariate) indicated a significant main effect for performance feedback-- $F(1,33) = 5.48, p < .025$. "High-success" subjects reported lower post-treatment headache activity. No other

significant main effects or interaction effects were uncovered.

The demonstrated efficacy of cognitive-behavioral treatments for recurrent headache has drawn attention to several cognitive variables that may mediate therapeutic outcome. The above-noted findings (Andrasik & Holroyd, 1980; 1983; Holroyd et al., 1984) clearly support hypotheses regarding the therapeutic value of enhanced self-efficacy and outcome expectations, as well as perceived self-control and therapist support. Recent literature reviews (Holroyd & Penzien, 1986; Litt, 1986) suggest that there is mixed support for medical and psychological placebo effects, with the later being more effective. Generally, placebo conditions have not been appropriately utilized in a manner that controls for the comparative behavioral procedure. For example, behavioral procedures that are presented as a skills acquisition treatment are often compared to psychological placebos that merely control for the effects of therapist attention--i.e. non-directive discussion sessions about headache activity (Blanchard et al., 1980). Recall that comparable therapeutic outcomes were reported when a placebo procedure was properly matched with the designated behavioral treatment (e.g. Andrasik & Holroyd, 1980; 1983). Future research is necessary to determine if there are distinct cognitive factors that mediate therapeutic outcomes from ("non-specific") placebo vs. ("specific") behavioral treatments.

The efficacy of cognitive coping strategies.

The previous subsection contained evidence to show that cognitive variables comprise the efficacious component in

behavioral treatments for recurrent headache. This point is quite significant for an understanding of cognitive treatment strategies for recurrent headache. It follows from research such as Andrasik and Holroyd (1980; 1983) that the therapeutic elements of behavioral and cognitive pain-attenuating techniques are essentially identical--i.e. behavioral techniques are cognitive in nature. For example, it is unlikely that behavioral relaxation protocols (Progressive Relaxation, BFB, Deep Breathing) can be utilized without inadvertently employing cognitive strategies such as diverting attention from pain experience to the identified treatment procedure, positively re-appraising one's ability to cope with the pain, etc. Therefore, the theoretical distinction between efficacious components of behavioral, cognitive-behavioral, and cognitive pain coping strategies is arbitrary at best (Pearce, 1983; Tan, 1982; Turner & Chapman, 1982).

Cognitive strategies are generally characterized as contributing to pain management by altering the patient's faulty appraisal of situational stressors. Maladaptive stress appraisals in turn exacerbate the perception of pain severity, helplessness, anxiety, and/or depression (Blanchard & Andrasik, 1985; Turner & Chapman, 1982). There has been much uniformity in schematic descriptions of cognitive strategies that are potentially available (e.g. Pearce 1983; Turk, Meichenbaum, & Genest, 1983). Fernandez (1986) recently presented one of the most succinct, and comprehensive of these schemas--see Table 2.

Table 2

Classification of Cognitive Pain Coping Strategies (Fernandez, 1986)

I. Imagery/Fantasy

a) Incompatible imagery

- pertains to events (sensory/situational) incompatible with pain
 - (i) incompatible emotive imagery--elicits emotions of affective pain reactions that are incongruent with pain experience (e.g. imagining a situation that elicits pleasure, anger, or joy, vs. pain-related discomfort)
 - (ii) incompatible sensory imagery--centers on scenes that are either irrelevant to pain stimuli, or that are essentially pleasant in nature (e.g. lying on a beach in the warm sun)

b) Transformative imagery--modifies components of pain experience

- (i) contextual transformation--pain-related situation is re-interpreted in order to alter the subjective impact of pain (e.g. imagining that one's pain has been suffered in the course of struggling to win an Olympic competition)
- (ii) stimulus transformation--pain experience is altered by imagining that particular mechanisms associated with the pain (intensity, duration, etc.) can be altered to reverse the degree of pain severity (e.g. imagining that one is loosening a tight headband that was causing a headache)
- (iii) response transformation--pain responses are relabeled by dissociating select qualities from the pain experience (e.g. during a headache episode, one focuses only on sensations of warmth throughout the shoulders, neck, and head, independent of pain sensations in these areas)

II. Self-Statements

- a) Coping self-statements--self-statements support perceived ability to withstand nociceptive input
- b) Reinterpretive self-statements--avoidance or re-appraisal of harmful or noxious dimensions of pain experience
 - (i) denial-oriented--negates select dimensions of pain
 - (ii) rationalization-oriented--pain experience is re-appraised to identify effects that are positive in nature

III. Attention-Diversion

- Note: this strategy ranges along a continuum from passive distraction to active diversion
- a) Passive distraction--observation of a distractor stimulus during pain experience (e.g. watching a movie to distract one's attention from a pain episode)
- b) Active attention-diversion--subject participates in a distractor task by means of complex interactions that involve a task performance (e.g. memorizing a series of sports statistics in order to divert attention away from a headache)

Empirical support for the efficacy of imagery, self-statement, and attention-diversion strategies in reducing pain has been derived primarily from experimental vs. clinical research (e.g. Spanos, Ollerhead, & Gwynn, 1986; Spanos, Stam, & Brazil, 1981). Comparisons of cognitive strategies in clinical pain research are problematic. Most treatment protocols comprise numerous cognitive strategies that patients utilize across diverse social settings. For example, popular treatments such as relaxation training (Bakal, 1982; Blanchard & Andrasik, 1985) and stress inoculation training (Turk, Meichenbaum, & Genest, 1983) are designed to include a repertoire of imagery, self-statement, and attention-diversion strategies. It is difficult to partial out specific from non-specific effects in assessing whether a desired therapeutic outcome is significantly attributable to select cognitive strategies. Conversely, investigations into the relative efficacy of cognitive coping strategies are more reliable when a time-limited coping method is applied within one laboratory setting to manage experimentally-controlled pain. Given that pain chronicity impacts on behavioral, cognitive/affective, and social dimensions of an individual's life (Philips & Jahanshahi, 1985), it is important to assess whether cognitive strategies that attenuate experimental pain are equally effective in managing various forms of chronic pain.

A recent study by Brown (1984) was unique in assessing the relative efficacy of stimulus-based and response-based imagery

coping strategies for experimental and clinical pain. Stimulus-based imagery was defined in terms of the subject's involvement in sensory dimensions of each imagined scene--e.g. imaginatively focusing on visual, auditory, olfactory, or somatosensory qualities associated with lying on a beach on a warm, sunny day. Response-based imagery highlighted the subject's behavioral response in each scene--e.g. imaginatively focusing on how skeletal muscles were relaxed, and how the rate of breathing reflected a slow, deep pattern, while lying on the sun-filled beach. Thirty-nine migraine subjects were randomly assigned to one of three treatment groups: response-based imagery/stimulus-based imagery/placebo control ("subconscious reconditioning").

In the initial experimental session, subjects were instructed in their respective treatments and then presented with the cold pressor test. The post-test for the cold pressor was administered at the end of four subsequent sessions in which migraineurs were taught to use their various coping strategies in reducing headache activity. Brown (1984) reported that the response-based and stimulus-based imagery groups demonstrated significant pre- to post-test improvement on pain tolerance and pain magnitude. Following a significant group effect in a MANOVA (with composite magnitude and tolerance scores as dependent measures), a significant multivariate contrast was found in a comparison of the imagery groups combined vs. the control group-- $\bar{U}(2,1,36) = 0.727$, $p < .05$. The two imagery groups did not differ significantly on

the pain magnitude and tolerance scores for the cold pressor.

A second aspect of Brown's study examined the relative efficacy of the above treatments in attenuating headache activity. Numerous headache measures (headache index, duration, intensity, etc.) indicated that the three experimental groups did not differ significantly on headache activity at the pre-treatment interval, according to a 4-week baseline period. Each group attended four headache treatment sessions following their training with the cold pressor test. A MANOVA was conducted on group differences for the composite headache scores from baseline to post-treatment. This analysis indicated a significant main effect for treatments-- $U(5,2,36) = 0.513$, $p < .05$. Multivariate contrasts found that the combined headache scores for the imagery groups differed significantly from the placebo control group-- $U(5,1,36) = 0.581$, $p < .05$. Once again, no significant difference was observed in a comparison of the two imagery groups. Subsequent analyses for baseline to 8-week follow-up data indicated that the same group trend occurred for improvement in percentage of headache activity. That is, response- and stimulus-imagery groups were superior to the placebo group, and the two imagery groups did not differ from each other.

The study by Brown (1984) supports the conclusion that compared to a plausible control treatment, pain management training in either response- or stimulus-based imagery is efficacious in reducing migraine headache activity. However, this conclusion

must be qualified by two considerations. First, the mean pre-treatment headache frequency for the migraineurs in Brown's study was one headache per week. Since Brown conducted analyses on the percentage of improvement in headache variables, the reductions in headache activity appear inflated when compared to mean differences in the actual headache activity. A case in point is the fact that baseline to post-treatment improvement on headache frequency only ranged from 12.62% (response-imagery) to 43.69% (stimulus-imagery). This reduction is respectively equivalent to only .5 and 1.7 fewer headaches in mean headache frequency for the imagery groups over the four weeks of treatment. Confirmation of Brown's findings should be attempted by using a headache population with a greater frequency of headache activity--e.g. CTH subjects. The second qualification to the findings of Brown (1984) concerns the methodologic confound of an order effect in administering the cold pressor test in the final treatment session. It is likely that subjects who perceived the headache treatment as being efficacious additionally expected to be able to control cold pressor pain in the post-test. This could account for the identical trends in group data for both the reduction of headache activity and the pain-attenuation of cold pressor pain. Subsequent studies are required that will ensure independence between assessments of experimental pain management and clinical pain management.

Previous literature reviews of psychological treatments for chronic pain support the efficacy of cognitive pain coping

strategies (Blanchard & Andrasik, 1985; Pearce, 1983; Tan, 1982; Turner & Chapman, 1982). However, future research must resolve numerous conceptual and methodologic issues pertaining to the treatment of recurrent headache. These issues include the following: (a) distinguishing the relative contribution of separate cognitive strategies, in treatment protocols for chronic pain (e.g. Rybstein-Blinchik, 1979); (b) determining whether a credible placebo treatment is efficacious, when it is presented as a skill acquisition strategy (e.g. Richter, McGrath, Humphreys, Goodman, Firestone, & Keene, 1986); and (c) confirming whether there is a generalization of reductions in chronic pain activity to pain-related perceptions of stress, anxiety, helplessness, etc. (e.g. Blanchard, Andrasik, Appelbaum, Evans, Myers, & Barron, 1986).

Hypnotic interventions.

The above review has thusfar addressed common nonpharmacologic treatments for recurrent headache. Less common nonpharmacologic interventions emphasize similar pain-attenuating skills, such as increasing relaxation, and decreasing pain-related stress and anxiety (Spinhoven, 1988). These include hypnosis (e.g. Friedman & Taub, 1984; Howard, Reardon, & Tosi, 1982), autogenic training (e.g. Janssen & Neutgens, 1986), and meditation (e.g. Kabat-Zinn, Lipworth, Burney, & Sellers, 1987). Meditation and autogenic training have not received detailed attention in previous literature reviews. This is likely due to the fact that these treatments typically have been applied within a general protocol,

in which treatment components cannot be isolated and evaluated.

There are two competing formulations that account for the efficacy of hypnotic interventions for recurrent headache, as well as for other chronic, acute, and experimental pain phenomena (Nolan & Spanos, 1987). Neodissociation theory contends that hypnotic analgesia is the result of a two-component process (Hilgard, 1977). The subject's deliberate use of relaxation, attention diversion, and anxiety reduction accounts for the first analgesic component. The second component entails a special process that is accessed only when subjects with high hypnotic talent enter into a hypnotic state of consciousness. In this state, the subject's conscious mode of active-volitional cognition allegedly becomes dissociated from his or her autonomous, subconscious mode of receptive-monitoring cognition (Hilgard 1977, 1979, Hilgard & Hilgard, 1975). While in the hypnotic state the subject is aware of pain only through the subconscious mode of receptive-monitoring--i.e. the "hidden observer" (Hilgard, 1977). Subjects with high susceptibility to hypnosis are not cognizant of pain because the hypnotic suggestion purportedly creates an amnesic barrier that separates conscious and subconscious cognitive functioning. Neodissociation theory, therefore, does not attribute hypnotic analgesia to a total absence of pain experience. It instead holds that hypnosis interrupts the conscious awareness of pain sensations.

Social psychological theory presents an alternate formulation of hypnotic analgesia. It holds that hypnosis reduces pain because

hypnotic suggestions encourage subjects to engage in goal-directed strategies that are within their normal coping repertoire (Barber, Spanos, & Chaves, 1974; Chaves & Brown, 1987; Spanos, 1982, 1986; Turk, Meichenbaum, & Genest, 1983). For example, hypnotic suggestions of analgesia aid subjects in focusing attention on images/experiences that promote self-distraction, or that are incongruent with pain experience. Unlike the neodissociation tenet, the social psychological formulation does not attribute pain reduction solely to altered sensory events. It asserts that hypnotic analgesia also takes place through altering cognitive and affective dimensions of pain--e.g. reducing the subject's pain-related distress and negative self-statements (Spanos, 1982, 1986). In social psychological theory, pain analgesia is contingent upon (a) the subject's expectations, motivation, and beliefs about hypnosis; as well as (b) the subject's active interpretation of implicit demands from hypnotic instructions regarding targeted role behavior (Chaves & Barber, 1974; Coe & Sarbin, 1977; Spanos, Hodgins, Stam, & Gwynn, 1984; Spanos, Ollerhead, & Gwynn, 1986). This approach also holds that cognitive coping suggestions can be efficient pain-attenuating techniques for high and low susceptible persons, since responsiveness to suggestions is primarily dependent on contextual factors.

There have been very few investigations into the use of hypnosis as a treatment for recurrent headache. The neodissociation and social psychological formulations of hypnotic analgesia have

not been sufficiently tested in controlled research with recurrent headache subjects. Spinhoven (1988) noted that the available studies support hypnosis as an effective agent in reducing headache activity. Other reviewers uniformly point to methodologic deficiencies in the hypnosis treatment research (Chaves, 1988; Turner & Chapman, 1982; Turner & Romano, 1984). For example, Toomey & Sanders (1983) assessed the pain-attenuating ability of hypnosis on a sample of five subjects. In addition to this insufficient sample size, standardized ratings of daily headache activity were not used. Their treatment protocol was eclectic, and included training in imagery, symptom recognition of headache onset, and distraction from stress-inducing events. These features undermined the ability to make valid inferences about the specific contribution of hypnosis as a treatment for recurrent headache. Similarly, Howard, Reardon, & Tosi (1982) compared baseline headache activity to two successive intervals in which different hypnotic treatments were administered. Headache activity data from only one subject served as the evidence in comparing the successive hypnotic treatments, and no statistical analyses were reported. In spite of the numerous methodological problems in this study, Howard et al. concluded that Rational Stage Directed Hypnotherapy was superior to standard hypnotherapy in treating CMH.

Spinhoven (1988) correctly identified Friedman and Taub (1984) as the only direct comparison of hypnosis to T-BFB. These investigators set out to assess whether brief training in hypnotic

imagery was efficacious in reducing CMH activity over periods of one year (Friedman & Taub, 1984) and three years (Friedman & Taub, 1985). Subjects were exposed to three treatment sessions, and home practice was a major component in this intervention. Friedman and Taub (1984) grouped subjects according to a 2 (Hypnotic Susceptibility--HS: High/Low) X 2 (Hypnotic Imagery: Thermal/Standard) factorial design. Three additional experimental groups were used: T-BFB with Autogenic Training, Passive Relaxation Training, and a Waiting List Control. CMH activity was assessed by measuring peak headache intensity per week, weekly sum of quarterly intervals per day in which headache activity was reported, and units of headache medication consumed per week.

Statistical analyses in these studies were presented in a manner that was, at best, incomplete. Friedman and Taub (1984) initially conducted a 2 (Groups: Waiting List Control/Combined Treatments) X 2 (Interval: Week-1 Baseline/Week-12) Mixed MANOVA. This analysis combined the six treatment conditions without first demonstrating that these groups did not significantly differ on any dependent measure. In addition, headache-related features were assessed for 1-week intervals, which is an inadequate period of measurement by conventional standards. Nevertheless, the MANOVA resulted in a significant multivariate Group X Interval interaction. The univariate simple main effects indicated that the Combined Treatment groups showed significant reductions on all three dependent measures, while the Control group showed no change

over time. The long-term efficacy of the six treatments was next examined by averaging each dependent measure over 3-week intervals during Baseline, Treatment, and 6-month, 9-month, and 1-year Post-treatment. Separate Mixed ANOVAs, using the 6 (Treatment Groups) X 5 (Intervals) factorial design, resulted in nonsignificant interactions, and significant main effects for the Intervals factor. Friedman and Taub stated that these data indicated significant improvements over time intervals. yet post hoc comparisons to baseline CMH activity were not reported. The Mixed ANOVAs also resulted in a significant main effect for the Groups factor on headache medication usage. This finding was obviously of critical importance to their investigation. Group comparisons were not reported, thereby preventing the reader from identifying treatment groups that may have reduced headache activity due to higher overall medication consumption. Finally, planned comparisons demonstrated that HS vs. LS subjects (combined across the hypnosis conditions) continued to show reduced headache activity at 9-month and 1-year intervals. LS subjects only demonstrated reduced CMH activity up to the 6-month interval. This finding undermined the credibility of their initial analysis, which collapsed treatment groups in a treatment vs. control comparison. The lack of a hypnotic treatment effect for LS subjects could have been due to significantly lower levels of outcome expectation and motivation, given that LS subjects were asked to simulate self-hypnosis during regular home practice. These factors were not

assessed in this study.

Friedman & Taub (1985) reported that at a 3-year follow-up, medication usage was significantly reduced from baseline for all treatment groups. These investigators also noted that HS vs. LS subjects continued to report significantly lower headache activity from baseline through to the 3-year interval. This conclusion was advanced in the absence of conducting post hoc or planned comparisons to determine whether the statistically significant 3-year findings were an artifact of differences at the 1-year interval. In sum, the Friedman and Taub (1984, 1985) investigations did not provide empirical evidence for any conclusion about the relative merit of hypnosis in treating CMH. These investigations contained numerous inadequacies. Further to the shortcomings discussed above, additional problems included the following: the index of headache activity that is most clinically meaningful (i.e. sum of headache intensity ratings divided by the number of days monitored) was excluded from their report in favor of less subtle measures; adherence to home practice was not assessed even though it was a critical component of the treatment; the 1-year follow-up data was based on only 68% of the original sample, while the 3-year findings were derived from only 54.5%.

In general, methodological and statistical shortcomings in the available research precludes the ability to form conclusions about the value of hypnosis in treating recurrent headache. Deficiencies in the hypnosis literature include, but are not limited to the

Table 8

Mean Within-Session Ratings of Treatment-Induced Manipulations for
Response-based Imagery Subjects (N=30)

Within-Session Rating	Trials	
	Session 1	Session 2
	M (SD)	M (SD)
Relaxation during each imagined scene:	5.03 (0.78)	5.07 (0.70)
Percentage of time spent imagining each scene:	4.52 (0.95)	4.21 (1.21)
Vividness of each imagined scene:	4.33 (1.01)	4.27 (1.25)
Absorption in each imagined scene:	4.42 (0.94)	4.37 (1.15)
Hypnotic Depth (Hypnotic Imagery Ss only):	3.48 (1.75)	3.98 (1.47)

It is noteworthy that the absence of statistically significant differences on the above self-ratings was due to the fact that subjects reported uniformly elevated levels of treatment-induced experiences. Appendix B indicates that self-ratings were made on a seven-point Likert type scale (from 0 "not at all" to 6 "extremely"). Means for these self-ratings ranged between 3.48 and 5.07, which indicated that the imagery exercises were vividly experienced.

researchers note how the hypnotizability-treatment outcome relationship is mediated by contextual and cognitive factors--e.g., the reduction of catastrophizing, perceived therapist support, positive expectation, and treatment-induced self-efficacy (Chaves, 1988; Spanos, 1982: 1986). Resolution of this issue awaits research that is designed to assess treatment-related processes. It also remains for future headache research to determine the relative efficacy of hypnotic therapy in comparison to other cognitive-behavioral interventions. If hypnosis does prove to be efficacious in treating recurrent headache, the need will remain to identify unique and common therapeutic components, and to specify if these are best accounted for in a neodissociation or social psychological formulation.

Current Investigation

Overview

This dissertation project was undertaken to examine the efficacy of a specific cognitive strategy, response-based imagery, in facilitating the self-management of recurrent headache. Hypnotic and nonhypnotic protocols were utilized to critically evaluate this imagery technique. Two experiments were designed to contribute to the empirical literature in several ways. First, this was the first investigation to appropriately utilize a monitoring control group in assessing the efficacy of hypnotic treatment for recurrent headache. The relationship between hypnotizability and headache reduction was also assessed for subjects in the Hypnotic and Nonhypnotic Imagery conditions. Second, two separate studies were used to replicate and extend the findings of Brown (1984). Subjects who presented either CMH or Mixed CMH/CTH symptoms were recruited for Experiment 1. Experiment 2 included subjects who presented symptoms of CTH. It was previously noted that Brown (1984) assessed the efficacy of nonhypnotic imagery training with only CMH subjects, and that there were methodological problems in her investigation. Third, Experiment 2 in this investigation utilized a credible placebo that was presented as a skill acquisition treatment. There has been limited use of this type of placebo in applied research into recurrent headache. Aside from the above-mentioned effort of Brown (1984), pseudo-skills training placebos have only been used with pediatric migraineurs (e.g.

Richter et al., 1986) and in BFB treatments for adult subjects with CTH (e.g. Andrasik & Holroyd, 1980; 1983). Fourth, this research aimed to determine if treatment-induced reductions in headache activity covaried significantly with the illness attributions of subjects. Each subject's illness schema of recurrent headache (as a health problem per se) was assessed at Pre-treatment and Follow-up. No studies were located that examined the potential relationship between treatment-induced reduction in headache activity and headache-related illness representations.

Dependent Measures: Experiments 1 and 2

Headache rating cards (Appendix A).

Standardized headache rating cards assessed variables associated with headache activity--headache intensity, headache index, and medication usage. Subjects rated headache activity at four intervals each day (Blanchard & Andrasik, 1985).

The headache index was the primary assessment of daily headache activity. It was calculated by summing the four daily headache intensity ratings and then dividing that sum by the number of days that each subject completed rating cards, within each two-week interval of treatment--i.e. Baseline, Post-treatment, and Follow-up. Headache intensity was rated according to the conventional 6-point scale, from no headache, "0", to headache that is extremely intense and incapacitating "5" (Bakal, 1982; Blanchard & Andrasik, 1985). Medication usage was measured according to the 7-point classification scale adapted by Blanchard and Andrasik (1985).

Treatment efficacy ratings (Appendix B).

Within each treatment session, subjects assessed their ability to immediately experience suggested treatment effects, by means of using 7-point likert-type rating scales. The Placebo group assessed how vividly they experienced sensations that were described as being typical of subliminal treatment--from "not at all noticeable" (0) to "extremely intense" (6). This rating referred to the following nontherapeutic sensations in the neck and/or shoulders: warmth, tingling, prickly feelings, and numbness. The Imagery and Hypnosis groups used a similar 7-point scale to rate each imagery scene on the following dimensions: the extent to which they relaxed; the degree to which they were absorbed in the scene; the percentage of time spent imagining the scene; and vividness of the imagined scene. The Hypnosis group additionally rated how deeply hypnotized they felt following each imagined scene.

Physiologic measures (Appendix C).

Cardiovascular functioning was operationally defined in terms of HR, SBP, and DBP. These were utilized as measures of autonomic arousal/relaxation. Resting Frontalis EMG provided a physiologic measure for myogenic tension/relaxation. A detailed description of the method and instruments used in obtaining these variables has been included in the Apparatus section for Experiment 1.

Spielberger State-Trait Anxiety Inventory (Appendix D).

Only the Trait form of the State-Trait Anxiety Scale (Spielberger, Gorsuch, & Lushene, 1970) was used in this investigation. It facilitated the evaluation of pain-related distress among subjects. The A-Trait measure was composed of 20 items, with four-point scales (1 to 4) that yield a score ranging between 20 and 80. The test-retest reliability data are acceptable, ranging from .73 to .86.

Short Form Marlowe-Crowne Social Desirability Scale (Appendix E).

The short form of the Marlowe-Crowne scale (Strahan & Gerbasi, 1972) provided data regarding the extent to which subjects had a propensity to report qualities that are stereotypically socially desirable. These qualities are exemplified by the following: never hesitating to go out of one's way to offer help to another person, never resenting being asked to return a favor, etc. This assessment is generally used as a measure of social defensiveness. It is reasoned that self-reported qualities that are obviously socially desirable reflect the degree to which the subject is socially defensive. Given that the daily ratings of headache activity could be considered personal in nature, this instrument helped to assess the validity of the self-reported headache activity. The K-R 20 reliability coefficient for this abbreviated scale was quite similar to the full Marlowe-Crowne scale--.83 and .87, respectively. Moreover, the correlation of the total scores for these two scales was reported in the range of at least .90.

Implicit Models of Illness Questionnaire (Appendix F).

The Implicit Models of Illness Questionnaire (Turk, Rudy, & Salovey, 1986) is a validated instrument that measures the extent to which individuals conceptualize their illness/"health problem" to be serious, controllable, and changeable. The remaining subscale assesses the degree to which subjects feel personally responsible for the illness. The utilization of this scale enabled the investigator to (a) assess an illness schema that is specific to recurrent headache subjects; and (b) determine the degree to which this multidimensional schema covaried with treatment-induced improvement in headache activity. The coefficient alpha for the four subscales ranges from .68 to .92, which suggests a high reliability for the items composing these scales.

Carleton University Responsiveness to Suggestion Scale (Appendix G).

Hypnotizability was assessed by the Carleton University Responsiveness to Suggestion Scale (CURSS; Spanos, Radtke, Hodgins, Stam, & Bertrand, 1983). It yields three suggestibility scores, each of which are derived from seven items. The Objective scale (CURSS:O) ranges from 0 to 7, and it assesses the subject's self-reported overt response to the seven standardized suggestions. The Subjective scale (CURSS:S) ranges from 0 to 21. It measures how vividly subjects report experiencing the seven suggestions. The Objective-Involuntary scale (CURSS:OI) assesses the incidence of perceived involuntariness that is defined as moderate or strong. This score is derived from a subject's reported response to

suggestions that were passed on the CURSS:O. The CURSS correlates significantly with measures of imaginative activity/absorption (Tellegen & Atkinson, 1974) as well as with other measures of hypnotizability (Shor & Orne, 1962). Self-scored and observer-scored objective responses on the CURSS have been highly correlated in repeated assessments-- $r = .87$, Session 1; $r = .85$, Session 2 (Spanos, Cobb, & Gwynn, 1984). High stability for the three dimensions of the CURSS has also been reported for 2-week and 3-month intervals (Spanos, Radtke, Hodgins, Bertrand, Stam, & Dubreuil, 1983).

Vividness of Mental Imagery Questionnaire (Appendix H).

This 15-item questionnaire (Shor, Orne, & O'Connell, 1966) is a revision of the Betts Questionnaire Upon Mental Imagery (Betts, 1909). It was used to assess the relationship between treatment-induced reductions in recurrent headache activity and the reported propensity to engage in vivid imaginative activity during waking hours. The correlation between the Vividness of Mental Imagery Questionnaire and hypnotizability has been found in the order of .56 (Shor, Orne, & O'Connell, 1966). Shor et al. have also reported that the reliability coefficients for internal consistency of the imagery items were greater than .91 on two administrations.

Perceived Stress Scale (Appendix I)

This scale (Cohen, Kamarck, & Mermelstein, 1983) was included to assess the severity of affective distress prior to exposure to treatment. In addition, this assessment was instrumental in determining whether there was a significant and meaningful relationship between the pre-treatment level of stress and the degree of post-treatment headache improvement. Cohen et al. (1983) report that normative studies for the PSS have been conducted with university student samples. Those data indicate high internal reliability for the PSS--the coefficient alpha is in the order of .85. Test-retest reliability is high when re-administered over a few days (.85), and moderate when administered over six weeks (.55). Cohen et al. (1983) also provide concurrent validity for the PSS. It is significantly correlated with other indices of distress--e.g. physical symptom reporting and exposure to unpleasant life events.

Rosenberg Self-Esteem Scale (Appendix J)

This measure of positive affective functioning is comprised of 10 items that are scored on Guttman scales. Rosenberg (1979) reports test-retest reliability ("coefficient of reproductability") for this assessment in the order of .92, and a coefficient alpha of .72. This measure also demonstrates concurrent validity, insofar as it correlates significantly with assessments of health self-image (.83) and positive self-perception (.67).

Pennebaker Inventory of Limbic Languidness (Appendix K)

This 54-item checklist (PILL) is a trait measure of the

propensity to report physical symptoms. Pennebaker (1982) reports that the PILL has high internal consistency (Cronbach's alpha of .88), as well as high test-retest reliability over a two-month period ($r = .79$). The PILL was administered in the present investigation because of its potential use as a covariate in analyses of self-reported daily headache activity. It was of interest to determine whether the tendency to report physical symptoms was associated with the self-reported severity of daily headache activity.

Method: Experiment 1

Subjects.

Undergraduate university students enrolled in an Introductory Psychology course at Carleton University were recruited for this study. All subjects were offered experimental credit and/or a stipend for their participation. Given that this was a nonclinical sample, the IASP criteria of three months' pain duration was used to distinguish chronic from acute pain (Merskey, 1986).

Subjects were initially screened by means of a self-report questionnaire (Appendix L). Inclusion criteria were set to select those who presented CMH or Mixed CMH/CTH symptoms. The CMH symptoms included the following features: (a) recurrent headaches for at least three months; (b) at least one headache per week, and (c) any three of the next core features--unilateral vs. bilateral headache; throbbing pain; presence of prodrome features; nausea or vomiting accompanying headache; and sensitivity to light with headache onset. Mixed CMH/CTH was defined as the CMH profile with two core features, as well as one or more of the following CTH symptoms: bilateral vs. unilateral headache, pain that is typically focused in the forehead (though it might begin in the occipital or suboccipital region), pain as a dull persistent ache, headache described as a cap or band around the head, and sensations of tightness or pressure accompanying headache. The age range for inclusion into this study was 17 to 50 years of age.

Procedure.

Screening questionnaires were offered to approximately 2000 undergraduate students enrolled in Introductory Psychology. Potential subjects were informed that the investigator was surveying the incidence of chronic pain, particularly recurrent headaches, among undergraduate students. These students were informed that they might be asked to participate (for experimental credit) in an intervention study for chronic pain. Specific details were not released at that time regarding inclusion criteria, or the treatment protocol.

Subjects who met the inclusion criteria from the screening questionnaire were contacted by telephone. All semi-structured telephone interviews (Appendix M) were conducted by the present investigator or a Master's student who was familiar with diagnostic headache criteria. This interview followed a set of questions aimed at confirming/disconfirming whether the inclusion criteria was met. Subjects were not invited into the study if they presented headache-related medical conditions that confounded reported headache activity--e.g. headache-related epileptic seizures, temporomandibular arthritis, etc.

The interviewer invited a subject into a preliminary "assessment phase" of the study once the objective criteria for the CMH or Mixed CMH/CTH profile were met. The preliminary assessment phase entailed the administration of a questionnaire battery, and completion of daily headache monitoring cards over a 2-week

period. All subjects were informed that they would be offered a 2-week "treatment" at some future date to learn a standardized pain management skill. It was also noted that additional monitoring of daily headache activity would be required.

Following the completion of the baseline assessment procedures, subjects were randomly assigned to one of three conditions: Nonhypnotic Imagery, Hypnotic Imagery, or Monitoring Control. The two treatment conditions consisted of two sessions (45 minutes each). Table 3 outlines the general protocol of this investigation.

Table 3

Protocol for Experiment 1

<u>Session</u>	<u>Activity</u>
1	Administration of initial screening questionnaire (Appendix L)
2	Telephone screening interview (Appendix M)
3	Administration of consent form (Appendix N); initiation of daily headache recording over 2-week interval (Appendix A); and random assignment to experimental conditions
4	Administration of questionnaire battery (Appendix D,E,F,I,J,K); treatment session 1 begins (Appendix O); daily headache monitoring is maintained (Appendix A)
5	Treatment session 2 is conducted; initiation of 6-week monitoring period for follow-up assessments of daily headache activity (Appendix A)
6	Administration of IMIQ (Appendix F), Carleton University Responsiveness to Suggestion Scale (Appendix G), the Vividness of Mental Imagery Questionnaire (Appendix H); and debriefing procedure (Appendix P)

The Monitoring group continued to record daily headache activity throughout the treatment and follow-up intervals. They were informed that the investigator required further data in order to establish accurate norms for headache activity among undergraduate students. Monitoring subjects were offered a response-based imagery treatment after the follow-up assessment.

The Nonhypnotic Imagery condition consisted of two 45-minute sessions of response-based imagery training that was guided by tape recorded instructions. Groups of six or fewer subjects met for each session. Subjects sat in semi-reclined lounge chairs through the entire session. SBP, DBP, and HR were assessed at the beginning and end of each session to determine the degree to which this treatment induced physiologic relaxation. Subjects were instructed to imaginatively participate in three 90-second fantasy scenes. Each scene was repeated once, thereby resulting in the administration of six response-based imagery scenes per session. The initial four scenes were guided by tape recorded descriptions that facilitated relaxation responses--e.g. relaxing in the sun while lying on a beach (Brown, 1984). For the fifth and sixth scenes, subjects were instructed to engage in self-guided imagery that was congruent with guidelines for the response-based strategy of the previous scenes. The treatment rationale explained that response-based imagery enhanced self-control over autonomic functioning, and that this in turn facilitated self-control over headache-related pain. Subjects were encouraged to practice

response-based imagery each day, in order to control headache activity as well as headache precipitants.

The Hypnotic Imagery condition differed from the Nonhypnotic Imagery treatment only insofar as subjects were told that hypnosis would facilitate their imaginative involvement in the structured and self-guided scenes. Each treatment session began with a 5-minute hypnotic induction procedure which was modified from Barber (1969). This procedure included inter-related suggestions for relaxation, drowsiness, and entering hypnosis. At the end of each session subjects were "awakened" from hypnosis. The hypnotic imagery sessions lasted approximately 55 minutes each.

Apparatus.

Heart rate and blood pressure ratings (HR, SBP, DBP) were conducted on a Labtron Model 847 sphygmomanometer. Prior to the placement of the cuff microphone on the brachial artery of the subject's left arm, the artery was palpated to increase the reliability of the measurement. The cuff inflated automatically to 30-40 mm Hg above occlusion of the radial pulse. Cuff deflation was automatically controlled at a rate of 3 to 5 mm Hg/sec. SBP and DBP were registered at the beginning and cessation (respectively) of Korotkoff sounds. An audible tone was emitted with each Korotkoff sound. Heart rate was measured during the interval (approximately 30 seconds) of cuff deflation, and assessed according to the inter-beat intervals associated with each pulse (Korotkoff sound). Measurements of SBP, DBP, and HR were

digitally displayed.

Hypotheses.

It was hypothesized that subjects in the Imagery and Hypnosis treatment conditions would significantly reduce mean daily headache activity (headache index) across treatment intervals, while Controls would demonstrate no change. It was further hypothesized that the Hypnotic and Nonhypnotic Imagery groups would not differ significantly on mean ratings of daily headache activity (headache index) across treatment intervals. The preceding hypotheses were made on the basis of the prediction that treatment-induced headache reductions would not be attributable to medication consumption, hypnotizability, or actual changes over trials in cardiovascular functioning (SBP, DBP, HR). A final hypothesis concerned the degree to which (in-session) treatment-induced processes were experienced by Hypnotic and Nonhypnotic Imagery groups. It was predicted that these groups would not differ significantly in mean within-session ratings of the degree to which they experienced experimental effects--i.e. relaxation, imagery vividness, absorption in imagery, and percentage of time spent imagining imagery scenes.

Results Experiment 1

Sample characteristics.

Approximately 2000 students enrolled in Introductory Psychology at Carleton University were informed that an intervention project would be conducted to assess an experimental treatment for chronic pain. A screening questionnaire (Appendix L) was made available to all interested students. Eighty-eight students were considered for entry based upon the congruence of their reported symptom profile with the diagnostic features of CMH or Mixed CMH/CTH (Blanchard & Andrasik, 1985; Merskey, 1986).

Medical confirmation of CMH or Mixed headache profiles was not required for entry into this study. The ethical considerations discussed by Andrasik and Holroyd (1980) were instrumental in this decision. Students were recruited because: (a) one of the three experimental conditions (Monitoring Control) was nontherapeutic by definition; (b) students could be assured of benefitting from their participation, since course credits were guaranteed regardless of the intervention outcome; (c) the use of students increased the likelihood of homogeneity of exposure to life stressors; and (d) previous research (e.g. Antanasio & Andrasik, 1987) found that university students present an elevated level of recurrent headache activity. Although clinically diagnosed subjects were not recruited, all participants were exposed to a series of screening assessments to ensure a meaningful degree of concordance with acceptable CMH or Mixed features.

In the next phase of screening, the 88 potential recruits participated in a semi-structured telephone interview (Appendix M). The purpose of the interview was to confirm each student's CMH or Mixed profile, based on information about headache history and presenting symptomology. Sixty-seven successfully passed the telephone screening interview and volunteered to enroll in this experiment.

The final criteria for selection entailed the demonstration of at least one headache per week, as assessed over a 2-week baseline period. The 67 completed the baseline questionnaire battery and monitored their daily headache activity over the 2-week baseline period by means of four daily ratings of headache intensity (Appendix A).

The subject pool was reduced from 67 to 54 subjects prior to the completion of the baseline assessments (questionnaire battery, and 2-week period of daily headache monitoring). During the interval of Baseline to Follow-up, eight subjects withdrew from this study due to course commitments. These subjects were distributed across treatments in a proportionate manner: Hypnotic Imagery, three subjects; Nonhypnotic Imagery, two subjects; Monitoring Controls, three subjects. One subject withdrew prior to completion of the study due to pregnancy. Three additional subjects were excluded on the basis of missing data. The mean age of the final sample of 42 subjects was 21.41 years ($SD = 6.58$). Participants ranged in age from 17 to 49 years. The proportion of males to females was equivalent across experimental conditions: Hypnotic

Imagery group (4 males, 11 females); Nonhypnotic Imagery group (4 males, 11 females); and Monitoring Control group (1 male, 11 females). This distribution of males-to-females across experimental conditions produced a nonsignificant chi-square-- $\chi^2(2) = 1.71, ns.$

Data analyses were organized sequentially to examine the following issues: (a) treatment efficacy of Hypnotic and Nonhypnotic Imagery training, (b) treatment effects as assessed by the headache index, (c) medication usage across Trials, and (d) the relationship between treatment effects and cognitive and physiologic processes. Results from these data analyses will be presented in this order.

Hypnotic/nonhypnotic treatments.

The initial step in assessing the treatment efficacy of response-based imagery training, in comparison to a Monitoring Control condition, required a comparison of Hypnotic and Nonhypnotic Imagery groups. This issue was examined with a 2 (Condition: Hypnotic/Nonhypnotic Imagery Training) X 5 (Trials: Baseline/Post-treatment/Follow-up 1/2/3) Mixed ANOVA. The headache index (i.e. sum of daily headache intensity ratings / number of days) was selected as the dependent variable for this analysis, since it is conventionally considered to be the most clinically meaningful measure of headache activity (Blanchard & Andrasik, 1985).

It was theoretically important to determine if the Mixed ANOVA produced a significant Condition X Trials interaction, or a

significant main effect for the Condition factor. Nonsignificant findings were evidenced in each case: Condition X Trials-- $F(4,112) = 1.22$, ns; Condition-- $F(1,28) = 2.54$, ns. This indicated that Hypnotic and Nonhypnotic Imagery subjects exhibited equivalent levels of headache activity across Trials. Given these findings, Hypnotic and Nonhypnotic Imagery subjects were combined and classified as the Response-based Imagery group. Headache index means and standard deviations from the above analysis are listed in Table 4.

Table 4

Headache Activity Across Trials for Hypnotic and Nonhypnotic Imagery Subjects

Trials	Response-Based Imagery Groups	
	Nonhypnotic ($N = 15$)	Hypnotic ($N = 15$)
	M (SD)	M (SD)
Baseline	4.42 (2.49)	3.95 (1.56)
Post-treatment	3.83 (2.28)	2.51 (1.58)
Follow-up 1	3.51 (2.52)	2.56 (1.79)
Follow-up 2	3.65 (2.88)	1.93 (1.44)
Follow-up 3	3.29 (2.62)	2.27 (1.41)

Treatment effects on headache index.

A comparison of the Response-based Imagery group to the Monitoring Control group was next examined in a 2 (Conditions:

Response-based Imagery/Monitoring) X 5 (Trials: Baseline/Post-treatment/Follow-ups 1/2/3) Mixed ANOVA. The headache index of mean daily headache activity again served as the dependent variable. This analysis produced a significant Condition X Trials interaction-- $F(4,160) = 2.47, p < .05$. Table 5 illustrates this effect.

Table 5

Headache Activity Across Trials for Response-based Imagery Subjects and Monitoring Controls (N = 42)

Trials	Headache Index Ratings			
	Imagery Group		Control Group	
	M	(SD)	M	(SD)
Baseline	4.18 a	(2.06)	4.04 a	(1.71)
Post-treatment	3.17 b	(2.04)	3.24 a	(2.42)
Follow-up 1	3.03 b	(2.20)	4.17 a	(3.35)
Follow-up 2	2.79 b	(2.40)	4.19 a	(3.11)
Follow-up 3	2.83 b	(2.12)	3.72 a	(2.71)

Note: Within columns, means that share a common subscript fail to differ significantly at $\alpha = .05$.

Analyses of simple main effects indicated that headache activity for the Monitoring Control group did not differ

significantly over Trials-- $F(4,160) = 1.21$, ns. Conversely, the Imagery group demonstrated a significant simple main effect for headache activity across Trials-- $F(4,160) = 6.22$, $p < .001$. Tukey's Least Significant Difference Test (LSD) was utilized in post hoc comparisons. These post hoc tests demonstrated that headache activity at each post-baseline interval was significantly reduced from the Baseline level. Headache activity did not differ significantly in comparisons among post-baseline periods (see Table 5).

The above analyses established that the Response-based Imagery treatment was efficacious in reducing headache activity. Subjects in this condition evidenced significantly lower post-baseline activity in comparison to their mean level at baseline. For Monitoring Control subjects, the index of daily headache activity did not change significantly across trials.

Medication usage.

Medication usage over trials was examined next, to determine if headache reductions were attributable to changes in the quantity or type of medication consumed. Daily medication usage was measured by means of the Menninger categories of analgesic potency (Blanchard & Andraisk, 1985). This assessment is derived from an index of the number of pills consumed, as well as the standardized potency value for each ingested medication. A mean daily index of medication usage was calculated for Baseline, Post-treatment, and the three Follow-up periods. The formula for computing the

medication index was modeled on the previously described index of daily headache activity.

It was theoretically important to determine if the pattern of treatment-induced headache reduction was paralleled by an increase in medication consumption. This finding would have been reflected in a Condition X Trials interaction, or a main effect for Conditions, in a 2 (Condition: Imagery/Monitoring) X 5 (Trials: Baseline/Post-treatment/Follow-up 1 /2 /3) Mixed ANOVA. The dependent variable for this analysis was the index of daily medication usage. The Condition X Trials interaction was nonsignificant-- $F(4,37) = 0.97$, ns. The main effect for the Condition factor was also nonsignificant-- $F(1,40) = 1.64$, ns. A significant main effect was observed for the Trials factor-- $F(4,37) = 2.78$, $p < .05$. Post hoc LSD tests on the marginal means (collapsed across Conditions) indicated that each Post-baseline medication index was significantly lower than the Baseline level. Comparisons among Post-baseline levels of medication usage showed no significant differences. Data for these post hoc comparisons are presented in Table 6.

Table 6

Post hoc Comparisons of Mean Daily Medication Usage Over Trials
(Collapsing Across Conditions--N=42)

Trials	Mean Daily Medication Index	
	M	(SD)
Baseline	0.95 a	(1.04)
Post-treatment	0.58 b	(0.77)
Follow-up 1	0.57 b	(0.88)
Follow-up 2	0.53 b	(0.91)
Follow-up 3	0.63 b	(0.90)

Note: Means sharing a common subscript fail to differ significantly at $\alpha = .05$.		

The preceding analyses regarding treatment efficacy supported the view that reduced headache activity was attributable to the introduction of response-based imagery training. A reduction in headache activity was observed over trials. This occurred in spite of the fact that across these periods, subjects decreased their level of medication usage from Baseline.

Treatment-induced cognitive and physiologic processes.

The previous analyses investigated treatment outcome. It was also of interest to examine if treatment-induced processes (cognitive and physiologic) reflected a pattern over intervals that

was congruent with treatment-induced headache reduction. This objective was met by examining subjects' in-session ratings of the degree to which they experienced the suggested experimental effects. The cognitive variables were mean in-session ratings of the following: relaxation, absorption in imagery, vividness of imagery, percentage of time involved in imagery, and (in the case of Hypnotic Imagery subjects) hypnotic depth (Appendix B).

In-session ratings for relaxation, absorption, vividness, and percentage of time involved in imagery were assessed in separate 2 (Condition: Hypnotic/Nonhypnotic Imagery) X 2 (Trials: Session 1/Session 2) Mixed ANOVAs. Condition X Trials interactions and main effects for Condition and Trials factors were nonsignificant. Depth of hypnosis was assessed for Hypnotic Imagery subjects in an ANOVA, with one within factor for Trials (Sessions 1 and 2). This analysis also yielded a nonsignificant result. The summary table for these analyses is presented in Table 7. Data relevant to these analyses are noted in Table 8.

Table 7

Summary Table for Mixed ANOVAs on Within-Session Ratings of Treatment-Induced Effects

	ANOVA Statistics		
	F	(df)	p
Relaxation during each imagined scene:			
- Condition X Trials	0.36	1,28	ns
- Condition	0.94	1,28	ns
- Trials	0.06	1,28	ns
Percentage of time spent imagining each scene:			
- Condition X Trials	0.21	1,28	ns
- Condition	1.09	1,28	ns
- Trials	2.62	1,28	ns
Vividness of each imagined scene:			
- Condition X Trials	0.87	1,28	ns
- Condition	1.10	1,28	ns
- Trials	0.36	1,28	ns
Absorption in each imagined scene:			
- Condition X Trials	0.15	1,28	ns
- Condition	0.22	1,28	ns
- Trials	0.05	1,28	ns
Hypnotic Depth (Hypnotic Imagery Ss only):			
- Trials	1.55	1,14	ns

Table 8

Mean Within-Session Ratings of Treatment-Induced Manipulations for
Response-based Imagery Subjects (N=30)

Within-Session Rating	Trials	
	Session 1	Session 2
	M (SD)	M (SD)
Relaxation during each imagined scene:	5.03 (0.78)	5.07 (0.70)
Percentage of time spent imagining each scene:	4.52 (0.95)	4.21 (1.21)
Vividness of each imagined scene:	4.33 (1.01)	4.27 (1.25)
Absorption in each imagined scene:	4.42 (0.94)	4.37 (1.15)
Hypnotic Depth (Hypnotic Imagery Ss only):	3.48 (1.73)	3.98 (1.47)

It is noteworthy that the absence of statistically significant differences on the above self-ratings was due to the fact that subjects reported uniformly elevated levels of treatment-induced experiences. Appendix B indicates that self-ratings were made on a seven-point Likert-type scale (from 0 "not at all" to 6 "extremely"). Means for these self-ratings ranged between 3.48 and 5.07, which indicated that the imagery exercises were vividly experienced.

Treatment-induced physiologic processes were also of interest because previous research has sought to identify pathophysiologic mechanisms that mediate CMH and Mixed headache activity. SBP, DBP, and HR were assessed in each session immediately prior to and following exposure to the response-based imagery treatment (Appendix O). The purpose of the analysis on physiologic measures was to determine if response-based imagery training inadvertently taught subjects to relax cardiovascular functioning within and/or across sessions. This question was addressed in a 2 (Condition: Hypnotic/Nonhypnotic Imagery) X 2 (Trials: Session 1/Session 2) X 2 (Intervals: Pre-/Post-session) Mixed MANOVA. SBP, DBP, and HR served as the dependent measures. These data were examined in a multivariate analysis in order to protect against Type 1 error.

The Mixed MANOVA produced only one statistically significant finding--a significant main effect for Intervals. Table 9 summarizes the Multivariate statistics.

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Table 9

Summary Table for Mixed MANOVAs on Physiologic Assessments

	MANOVA Statistics (SBP, DBP, HR)		
	Mult. F	(df)	p
Condition X Trials X Intervals	1.43	3,26	ns
Condition X Trials	0.13	3,26	ns
Condition X Intervals	0.91	3,26	ns
Trials X Intervals	1.49	3,26	ns
Condition	0.68	3,26	ns
Trials	2.31	3,26	ns
Intervals	8.19	3,26	.0005

Two univariate measures accounted for the significant omnibus F ratio in the Intervals factor. These physiologic measures were SBP and HR-- $F(1,28) = 24.80, p < .01$; $F(1,28) = 5.74, p < .05$; respectively. In each case, the Imagery subjects demonstrated a significant pre- to post-session reduction in autonomic functioning. Table 10 presents the marginal means and standard deviations (collapsing across Condition and Trials).

Table 10

Mean Pre- and Post-Session Ratings for SBP and HR (N = 30)

Physiologic Measures	Intervals	
	Pre-session M (SD)	Post-session M (SD)
SBP	116.20 (12.52)	110.20 (12.78)
HR	72.15 (13.23)	69.32 (12.27)

The above analyses indicated that exposure to response-based imagery training was associated with positive cognitive and physiologic processes. On average, subjects reported being imaginatively involved and relaxed during each session. There was also evidence that Imagery subjects exhibited reductions in their autonomic activity over the course of each session. In the absence of similar data from a nontreatment control group, it was not possible to determine whether these cognitive and physiologic processes were a specific effect of exposure to response-based imagery training. Alternatively, these effects could have been due to sitting in a semi-reclined position for the duration of each session.

Discussion: Experiment 1

Overall, findings from Experiment 1 supported the contention that hypnotic and nonhypnotic methods of response-based imagery training were efficacious in reducing headache activity for CMH and Mixed CMH/CTH.

It was hypothesized that Hypnotic and Nonhypnotic Imagery groups would not differ significantly over Trials, on the index of mean daily headache activity. This hypothesis was confirmed. It was also hypothesized that Hypnotic and Nonhypnotic Imagery subjects would demonstrate significantly lower headache activity across treatment intervals, as compared to the Monitoring Control group. This hypothesis was also confirmed. Finally, Imagery subjects did not significantly increase their pattern of medication usage over treatment intervals. Analyses demonstrated that all subjects reduced their Baseline medication consumption over subsequent Trials. This indicated that the observed reduction in mean daily headache activity was attributable to hypnotic and nonhypnotic response-based imagery training, and cannot be "explained away" in terms of medication used by subjects to ameliorate headaches.

The nonsignificant difference in treatment outcome for Hypnotic and Nonhypnotic Imagery groups underscored the importance of investigating whether there was also a similarity in treatment-related processes that mediated headache reduction. This issue was assessed directly in analyses that combined subjects from

Experiments 1 and 2, and it will be reviewed in the General Discussion.

A further concern that was explored in data analyses was whether treatment outcome was attributable to actual changes over Trials in cardiovascular functioning. It was hypothesized, therefore, that subjects would not demonstrate significant changes in cardiovascular functioning (SBP, DBP, HR) over Trials. This hypothesis was formulated on the basis of findings from numerous studies that failed to observe a direct relationship between physiologic changes and treatment-induced reduction in headache activity. The results of analyses supported this prediction. Imagery subjects demonstrated within-session reductions in autonomic functioning (SBP and HR), yet failed to lower physiologic functioning across sessions. Given this finding, treatment efficacy could not be attributed to the inadvertent acquisition of control over relaxation-related physiologic responses. The perceived ability to physically relax was not examined in this investigation. It is possible that Imagery subjects significantly increased their perceived control over headache-related physical functioning, given that they were initially informed that imagery training would facilitate their control over the impact of headache activity on daily functioning. Other investigations have found that perceived vs. actual control over physical functioning is significantly associated with positive treatment outcome (Holroyd et al., 1984).

A supplementary hypothesis predicted that Hypnotic and Nonhypnotic Imagery groups would not differ significantly in mean within-session ratings of treatment-related cognitive processes-- i.e. relaxation, imagery vividness, absorption in imagery, and percentage of time spent imagining each scene. This hypothesis was supported and in addition, the mean ratings on each of these variables indicated that treatment-effects were experienced by Imagery subjects from a moderate to high degree. The absence of differences between Hypnotic/Nonhypnotic conditions on treatment processes was theoretically meaningful. This issue will be fully reviewed in the General Discussion.

Method: Experiment 2

The experimental design, hypotheses, and analyses of the second experiment extended the findings from the previous experiment. The differentiating features were as follows.

Experiment 2: (a) used subjects who presented symptoms of CTH; (b) added a fourth experimental condition, in the form of a Placebo group; (c) expanded the Baseline and Follow-up assessments to include physiologic measures of frontalis EMG and autonomic functioning; and (d) increased the imagery training to four treatment sessions.

Subjects.

Selection procedures for this experiment paralleled those used in the previous study. Inclusion criteria were adjusted to select subjects who presented CTH symptoms. The CTH symptoms included: (a) absence of prodromal symptoms in self-report of "typical" headache; (b) recurrent headaches for at least three months; and, (c) any three of the next features--three or more headaches per week, bilateral vs. unilateral headache, pain that is typically focused in the forehead (though it might begin in the occipital or suboccipital region), pain as a dull persistent ache, headache described as a cap or band around the head, and sensations of tightness or pressure accompanying headache. The age range for this study was 17 to 50 years of age.

Procedure.

Experimental conditions and dependent variables were virtually identical to those described in the previous study. Detailed descriptions will be provided only for the non-redundant features-- i.e., Baseline and Follow-up assessment of frontalis EMG and autonomic activity, and the Placebo condition. Table 11 presents the general protocol for Experiment 2.

Table 11

Protocol for Experiment 2

<u>Sessions</u>	<u>Activity</u>
1	Administration of initial screening questionnaire (Appendix L)
2	Telephone screening interview (Appendix M)
3	Administration of consent form (Appendix N) and questionnaire battery (Appendix D,E,F,I,J,K); Physiologic Assessment (Appendix C); initiation of daily headache monitoring over 2-week baseline interval (Appendix A); and random assignment to experimental conditions
4 - 7	Treatment sessions 1 to 4, conducted over 3-week interval (Appendix O); administration of efficacy ratings within treatment sessions (Appendix B); daily headache monitoring is resumed at post-treatment (Appendix A)
8	Follow-up physiologic assessment conducted at 8 weeks post-treatment (Appendix C); and initiation of 2-week follow-up monitoring of daily headache activity (Appendix A)
9	Administration of IMIQ (Appendix F), CURSS (Appendix G), as well as the Vividness of Mental Imagery Questionnaire (Appendix H); and debriefing procedure (Appendix P)

Baseline and Follow-up physiologic assessment sessions were each approximately 30 minutes in duration. The protocol is noted in Appendix C. Upon arriving for each session, subjects completed a small questionnaire package. They were then instructed to sit in a semi-reclined chair and to relax. Frontalis EMG electrodes and the blood pressure cuff were then placed on the subject. A 10-minute adaptation period followed, in which equipment was calibrated and the subject adjusted to the experimental room. The adaptation period ended without any verbal indication to the subject. SBP, DBP, and HR were then recorded at this interval, and five minutes later. Frontalis EMG recording began next. It was monitored continuously over a five-minute period. Blood pressure and heart rate was then assessed at the completion of the frontalis EMG monitoring. The subject was instructed at this point that the physiologic assessment had been completed.

In regard to the experimental treatments, the Hypnotic and Nonhypnotic Imagery conditions consisted of four sessions of response-based imagery training that were guided by tape recorded instructions. Details of these treatments were previously noted in the Procedure section of Experiment 1. The Placebo treatment ("Subliminal Re-learning") was conducted for six or less subjects who sat in semi-reclined lounge chairs throughout each session. SBP, DBP, HR were assessed immediately prior to and following the slide presentation of the subliminal material. These physiologic measures assessed the degree to which the placebo might

inadvertently facilitate relaxation. However, no indication was given to suggest a potential link between the placebo and relaxation training.

Tape recorded instructions directed subject participation in the placebo condition. Each session presented six series of slides in discrete 1-minute segments. Within each 1-minute segment, four slides were flashed for approximately .5 seconds each, at 15-second intervals. Assessments of the perceived effects of this treatment were administered immediately following each of the one-minute slide presentations (Appendix B).

Subjects in the placebo condition were informed that the slides contained an embedded therapeutic message. The experimenter described a credible rationale as to how the slides would enable subjects to subliminally re-learn automatic maladaptive responses to headaches (i.e. affective, cognitive, physiologic, and behavioral responses). The nonsense syllable, "CUV", was presented simultaneously with each slide. Subjects were instructed to practice this subliminal re-learning task by simply recalling the cue syllable immediately prior to, or during, a headache. This condition therefore was described as a skill acquisition procedure. Subjects were directed to refrain from attempting to detect the embedded pseudotherapeutic message associated with the cue syllable. They were also instructed to avoid "over-attending" to the cue word, when it was momentarily recalled. headache episodes. This cautionary instruction was in fact administered to

prevent subjects from using the nonsense syllable as a distraction from headache-related pain. Appendix O contains a detailed outline of the tape recorded instructions for this treatment.

Apparatus.

Heart rate and blood pressure (HR, SBP, DBP) were assessed by the Labtron Model 847 Sphygmomanometer, using a method identical to that noted for Experiment 1.

Frontalis EMG was assessed by means of a forehead band with pre-set surface electrodes that were placed approximately 2.54 cm above the center of each eyebrow. A ground electrode was located in the center of the forehead. Electrode cups were filled with conductive gel and the forehead area was cleaned with Brasivol and Isopropyl Alcohol prior to EMG assessment. Electrode resistance was maintained at less than 10,000 ohms.

The EMG signal was measured by a Biofeedback Information System Model B-2, with a Model PA-2M Pre-Amplifier. Signals were integrated over 1-minute intervals. The integrated values were digitally displayed and peak to peak mv's were recorded manually.

Hypotheses.

As in the initial experiment, it was theoretically meaningful to determine if hypnotic and nonhypnotic imagery training was equally efficacious in reducing recurrent headache activity. It was hypothesized, therefore, that the Hypnotic and Nonhypnotic Imagery groups would not differ significantly on mean ratings of daily headache activity (i.e. headache index) across Trials. It

was also hypothesized that the combined Imagery groups would significantly reduce mean daily headache activity (headache index) across Trials, and that no reduction would be observed for the Placebo and Monitoring groups. Further, it was hypothesized that these treatment effects would not be attributable to medication usage, actual changes in physiologic functioning (Frontalis EMG, SBP, HR), or hypnotizability. Finally, it was hypothesized that Hypnotic and Nonhypnotic Imagery groups would not differ significantly in mean within-session ratings of the degree to which they experienced experimental effects--i.e. relaxation, imagery vividness, absorption in imagery, and percentage of time spent imagining imagery scenes.

Results: Experiment 2

Sample characteristics.

Approximately 2000 students enrolled in Introductory Psychology at Carleton University were informed that an intervention project would be conducted to assess an experimental treatment for chronic pain. A screening questionnaire (Appendix L) was made available to all interested students. Ninety-two students were considered for entry based upon the congruence of their reported symptom profile with the diagnostic features of CTH (Blanchard & Andrasik, 1985; Merske", 1986).

As previously discussed, medical confirmation of the CTH headache profile was not required for entry into this study. All participants were exposed to a series of screening assessments to ensure a meaningful degree of concordance with acceptable CTH features (Merskey, 1986).

In the next phase of screening, the 92 potential recruits participated in a semi-structured telephone interview (Appendix M). The purpose of the interview was to confirm each student's CTH profile, based upon information about headache history and presenting symptomology. Sixty-three subjects successfully passed the telephone screening interview and volunteered to enroll in this experiment.

The final criteria for selection entailed the use of a minimum rating of average daily headache activity, as assessed over a 2-week baseline period. The 63 recruits completed the baseline

questionnaire battery and monitored their daily headache activity over the 2-week baseline period by means of four daily ratings of headache intensity (Appendix A). The daily headache index was calculated for each participant using the standard formula--sum of headache intensity ratings / number of days (Blanchard & Andrasik, 1985). A daily headache index rating of 1.14 was considered appropriate to ensure sufficient headache activity that was consistent with the conventional CTH profile.

The subject pool was reduced from 63 to 55 subjects prior to the completion of the baseline assessments (questionnaire battery, 2-week period of daily headache monitoring, and frontalis EMG recording). Six subjects dropped out of this study, and two subjects reported a baseline daily headache index that fell below the 1.14 criterion. Through the course of the experiment and summary inspection of data, the sample size was further reduced from 55 to 47 subjects. Two subjects (from separate treatment conditions) withdrew from the experiment due to course commitments. Six other subjects presented incomplete data. The mean age of the final sample of 47 subjects was 20.43 years ($SD = 4.49$). Participants ranged in age from 17 to 40 years. The distribution of males to females was equivalent across experimental conditions: Imagery group (2 males, 10 females); Hypnosis group (4 males, 7 females); Placebo group (5 males, 8 females), and Monitoring group (2 males, 9 females)-- $\chi^2_{(3)} = 2.39$, ns.

In similarity to the first experiment, data analyses were organized to examine the following issues: (a) treatment efficacy of Hypnotic and Nonhypnotic Imagery Training, (b) treatment effects as assessed by the headache index, (c) medication usage across Trials, and (d) the relationship between treatment effects and cognitive and physiologic processes.

Hypnotic/nonhypnotic treatments.

As in experiment 1, the first task was to determine whether the Hypnotic and Nonhypnotic Imagery groups differed from one another in reported headache activity over Trials. This question was examined in a 2 (Condition: Nonhypnotic/Hypnotic Imagery Training) X 3 (Trials: Baseline/Post-treatment/8-week Follow-up) Mixed ANOVA. The headache index (i.e. the average daily headache score for each interval) served as the dependent variable.

The Mixed ANOVA failed to uncover a significant Condition X Trials interaction-- $F(2,42) = 0.05$, ns. The main effect for the Condition factor was also nonsignificant-- $F(1,21) = 0.11$, ns. Therefore, subjects in the hypnotic and nonhypnotic imagery training procedures were grouped within one classification (Response-based Imagery Training) for subsequent analyses. The headache index means and standard deviations are listed in Table 12.

Table 12

Headache Activity Across Trials for Hypnotic and
Nonhypnotic Imagery Subjects

Experimental Condition	Trials		
	Baseline M (SD)	Post-Treatment M (SD)	Follow-up M (SD)
Nonhypnotic Imagery (N=12)	3.35 (1.77)	2.67 (2.09)	2.96 (2.07)
Hypnotic Imagery (N=11)	3.62 (1.76)	2.96 (1.60)	3.09 (1.21)

Treatment effects on headache index.

Treatment efficacy of the response-based imagery training procedure was then examined in a 3 (Conditions: Response-based Imagery/Placebo/Monitoring) X 3 (Trials: Baseline/Post-treatment/Follow-up) Mixed ANOVA. The headache index of mean daily headache activity was once again the dependent variable. This analysis produced a significant Condition X Trials interaction-- $F(4,88) = 4.84, p < .01$. Table 13 illustrates this effect.

Table 13

Headache Activity Across Trials for Response-based Imagery Subjects, Placebo Subjects, and Monitoring Controls (N = 47)

Trials	Headache Index Ratings		
	Imagery Group	Placebo Group	Control Group
	<u>M</u> (<u>SD</u>)	<u>M</u> (<u>SD</u>)	<u>M</u> (<u>SD</u>)
Baseline	3.48 (1.73) a	4.08 (3.25) a	3.37 (1.24) a
Post-treatment	2.81 (1.84) b	3.95 (3.62) a	4.90 (2.61) b
Follow-up	3.02 (1.68) ab	4.21 (3.51) a	4.17 (1.88) ab

Note: Within columns, means sharing the same subscripts fail to differ significantly at $\alpha = .05$.

Simple main effects were calculated for the Condition X Trials data. Headache Index ratings for experimental groups were separately assessed across the three levels of the Trials factor. A trend towards a significant simple main effect was found for the Imagery group-- $F(2,88) = 2.75$, $p < .07$. Tukey's Least Significant Difference test (LSD) was utilized in the post hoc comparisons, as noted in Table 13. These analyses indicated that the Post-treatment headache index of the Imagery group was significantly reduced from their mean Baseline rating-- $t(45) = -2.29$, $p < .05$. This reduction in headache activity did not carry over to the 8-Week

Follow-up assessment. The Baseline/Follow-up comparison was nonsignificant-- $t(45) = -1.56$, ns. The comparison of headache activity at Post-treatment and 8-week Follow-up was also nonsignificant-- $t(45) = 0.73$, ns. The Placebo group demonstrated no significant simple main effect for headache activity across the Trials factor-- $F(2,88) = 0.23$, ns. In contrast, a significant simple main effect was observed across the Trials factor for the Monitoring group-- $F(2,88) = 6.71$, $p < .01$. LSD post hoc comparisons indicated that the Monitoring group significantly increased in headache activity from Baseline to Post-treatment-- $t(21) = 3.66$, $p < .01$. The post hoc comparison of headache activity at Baseline and 8-Week Follow-up was nonsignificant-- $t(21) = 1.92$. A nonsignificant post hoc comparison was also found for Post-treatment and 8-Week Follow-up-- $t(21) = -1.74$, ns.

These data indicated that subjects who received response-based imagery training reduced headache activity at the Post-treatment interval. This therapeutic effect did not carry over to the 8-week Follow-up interval. Conversely, subjects who simply Monitored their headache activity demonstrated a significant increase at the Post-treatment interval, but not at Follow-up. The Placebo manipulation had no significant effect on Baseline levels of headache activity.

Treatment efficacy was next explored by examining the distribution of subjects who did vs. did not reduce headache activity at either Post-treatment or Follow-up, as well as across

Post-treatment and Follow-up. Chi-Square analyses assessed whether there was a significant difference in the distribution of subjects who reduced headache activity within the Imagery, Placebo, and Monitoring Control conditions. Table 14 presents these data.

Table 14

Chi-Square Analysis on Distribution of Subjects Demonstrating Headache Reduction within Treatment Conditions

Experimental Condition	Treatment Status at Post-treatment	
	No Reduction in Headache Activity	Reduction in Headache Activity
Imagery	7	16
Placebo	7	6
Monitoring Control	7	4
$\chi^2_{(2)} = 3.95, ns$		
Experimental Condition	Treatment Status at Follow-up	
	No Reduction in Headache Activity	Reduction in Headache Activity
Imagery	7	16
Placebo	8	5
Monitoring Control	7	4
$\chi^2_{(2)} = 5.07, ns$		
Experimental Condition	Treatment Status Across Post-treatment and Follow-up	
	No Reduction in Headache Activity	Reduction in Headache Activity
Imagery	10	13
Placebo	10	3
Monitoring Control	9	2
$\chi^2_{(2)} = 6.36, p < .05$		
Note: Subjects identified in the Reduction/Nonreduction categories are not necessarily the same individuals at Post-treatment, Follow-up, and across Trials		

The Chi-Square analyses provided mixed support for the efficacy of response-based imagery training. There was a nonsignificant difference in the distribution of Imagery, Placebo, and Monitoring subjects who reduced their baseline headache activity at either Post-treatment or Follow-up. Alternatively, there were significantly more Imagery subjects (vs. Placebo and Monitoring subjects) who reduced headache activity across both Post-treatment and Follow-up.

An analysis related to the Chi-Square data concerned only those subjects who reported headache reduction from Baseline. Among these improved subjects we wished to determine whether the magnitude of improvement in headache activity was significantly greater among those who received Imagery training, as compared to those who received Placebo and Monitoring treatments. Table 14 notes that the number of Placebo and Monitoring subjects who demonstrated headache reduction was minimal. This precluded using the three treatment conditions as the grouping factor for a Mixed ANOVA on the magnitude of headache reduction across Trials. A 2-group schema was therefore adopted for this analysis. Theoretically, a grouping schema of Imagery vs. Nonimagery groups was desirable in order to compare conditions that were a priori defined as treatment vs. nontreatment. From a statistical perspective, the 2-group schema was advantageous in ensuring adequate cell sizes in the Mixed ANOVA. Subjects who demonstrated either Post-treatment or Follow-up reductions in headache activity were included in this analysis.

The magnitude of the percentage of improvement in headache activity was assessed in a 2 (Conditions: Imagery/Nonimagery) X 2 (Trials: Post-treatment/Follow-up) Mixed ANOVA. The percentage of improvement in the Headache Index, relative to the Baseline Interval, was calculated by the standardized formula noted in Blanchard and Andrasik (1985). This Mixed ANOVA produced nonsignificant effects for the Condition X Trials interaction, as well as for the main effect for the Trials factor-- $F(1,31) = 0.15$, ns; $F(1,31) = 2.82$, ns; respectively. The main effect for the Condition factor was significant-- $F(1,31) = 5.20$, $p < .05$. Marginal means for the percentage of improvement in headache activity between conditions are listed in Table 15.

Table 15

Mean Percentage of Improvement in Headache Activity Among Imagery vs. Nonimagery Subjects Who Demonstrated Reductions

Experimental Condition	Mean Percentage of HA Improvement Across Trials		
	N	M	SD
Nonimagery Subjects Who Improved:	14	5.16%	30.44
Imagery Subjects Who Improved:	19	28.56%	28.14

The Mixed ANOVA thereby demonstrated that among participants who reduced headache activity, Imagery vs. Nonimagery subjects demonstrated significantly greater improvement. The large standard

deviations in Table 15 are noteworthy. There was a considerable range in the overall percentage of improvement for the marginal means of both conditions. Nonimagery subjects who reduced headache activity at either Post-treatment or Follow-up demonstrated between -41.87% and 67.12% improvement. Improvement among Imagery subjects ranged between -30.44% and 63.42%.

Medication usage.

Treatment efficacy was next examined in an assessment of medication usage across treatment intervals. The purpose of this analysis was to assess whether reductions in headache activity were attributable to changes in the pattern of medication consumption over Trials. An average daily medication index was calculated for Baseline, Post-treatment, and Follow-up periods. Medication usage was measured in the same way as in Experiment 1.

A 3 (Condition: Imagery/Placebo/Monitoring) X 3 (Trials: Baseline/Post-treatment/Follow-up) Mixed ANOVA was conducted, with the average daily medication index as the dependent variable. The Condition X Trials interaction was nonsignificant-- $F(4,88) = 1.39$, ns. Additionally, the main effect for the Trials factor was less than 1-- $F(2,88) = 0.59$, ns. The main effect for the Condition factor approached significance-- $F(2,44) = 2.96$, $p = .06$. LSD post hoc comparisons of group means (collapsed across Trials) indicated that subjects in the Imagery condition had a significantly lower medication index than subjects in the Monitoring group-- $t(33) = 2.62$, $p < .02$. No other significant

comparisons were found. The trend in medication usage is presented in Table 16.

Table 16

Daily Medication Index Ratings (Averaged Across Trials) Among Treatment Groups

Experimental Condition	Mean Daily Medication Usage			
	N	M	(SD)	Range
Imagery Group	23	0.280	(0.380)	0.000 - 1.714
Placebo Group	13	0.415	(0.399)	0.000 - 1.163
Monitoring Group	11	0.707	(0.704)	0.037 - 2.538

These analyses indicated that response-based imagery training was efficacious, as compared to the Placebo and Monitoring conditions. The daily headache index among Imagery subjects decreased significantly from Baseline to Post-treatment. This index increased significantly among Monitoring subjects, and it remained unchanged for the Placebo group. The number of subjects who reduced headache activity at Post-treatment or 8-Week Follow-up was significantly greater in the Imagery vs. Placebo and Monitoring groups. The magnitude of the percentage of improvement in headache activity over treatment intervals (among subjects who showed reductions in their daily headache index) was significantly greater for Imagery vs. Nonimagery subjects. Finally, the treatment efficacy of Response-based Imagery training could not be validly attributed to

a higher incidence of medication usage among subjects in the Imagery condition. That analysis indicated that the daily index of medication usage was significantly lower (averaged over treatment intervals) for the Imagery group, as compared to the Monitoring group. The next phase of analyses therefore examined treatment effects on cognitive and physiologic processes.

Treatment-induced cognitive and physiologic processes.

Treatment-induced effects were studied by examining within-session self-ratings of subjects. Data for the Hypnotic/Nonhypnotic Imagery group was analysed separately from the Placebo group's data. Recall that in every session, Imagery subjects rated how they had responded to each of the six imagined scenes, using a 7-point unipolar scale. These ratings pertained to the perception of the following: the degree of relaxation, the degree of being absorbed in the imagery, the amount of time spent imagining each scene, and the vividness of each imagined scene. Subjects in the Hypnotic Imagery condition also rated the degree to which they felt deeply hypnotized during each imagined scene. The 7-point unipolar rating scale was used in each of the Placebo sessions to assess subjects' responses to each of the six slide exposures to "subliminal" messages. Self-ratings of the Placebo group referred to the following slide-induced experiences that affected the neck, shoulders, face, or forehead: tingling sensations, prickly feelings, numbness, and warmth.

The mean within-session subjective ratings of Imagery subjects were each examined in separate 2 (Condition: Hypnotic/Nonhypnotic Imagery) by 4 (Trials: Sessions 1/2/3/4) Mixed ANOVAs. These analyses indicated that each of the subjective ratings produced a nonsignificant Condition X Trials interaction, and a nonsignificant main effect for the Condition factor. The main effect for the Trials factor was significant for mean within-session subjective ratings of relaxation, time spent imagining each scene, and vividness of the imagined scenes. The ratings of how absorbed subjects were in the imagined scenes demonstrated a Trials main effect that approached significance. Table 17 presents the summary table for each of the Mixed ANOVAs. The Imagery group's within-session means and standard deviations for the above subjective ratings are noted in Table 18.

Table 17

Summary Table for Mixed ANOVAs on Within-Session Ratings of Treatment-Induced Effects

	ANOVA Statistics		
	F	df	p
Relaxation during each imagined scene:			
- Condition X Trials	0.40	3,63	ns
- Condition	1.04	1,21	ns
- Trials	4.92	3,63	< .01
Percentage of time spent imagining each scene:			
- Condition X Trials	0.46	3,63	ns
- Condition	0.70	1,21	ns
- Trials	5.60	3,63	< .01
Vividness of each imagined scene:			
- Condition X Trials	0.06	3,63	ns
- Condition	1.69	1,21	ns
- Trials	6.19	3,63	< .01
Absorption in each imagined scene:			
- Condition X Trials	0.25	3,63	ns
- Condition	1.09	1,21	ns
- Trials	2.55	3,63	.06
Hypnotic Depth (Hypnotic Imagery Ss only):			
- Trials	1.23	3,30	ns

Table 17 noted that subjects in the Hypnotic Imagery condition ($N = 11$) did not differ significantly over Trials in their within-session ratings of hypnotic depth. The mean level of reported hypnotic depth across Trials was 2.50 ($SD = 1.70$). The treatment-induced response to hypnotic induction was in the moderate range, therefore, for the Hypnotic Imagery group.

As shown by the LSD post hoc comparisons of Table 18, the mean within-session subjective ratings for the Imagery group were significantly lower during session two, and higher during session four. The magnitude of these mean ratings, (from 0 "Not at all" to 6 "Totally") ranged between 3.30 and 5.12. This indicated that Imagery subjects perceived themselves as having effectively experienced each of the treatment-induced effects within sessions.

Table 18

Mean Within-Session Self-Ratings of Treatment-Induced Manipulations for Imagery Subjects (N=23)

Within-Session Rating	Trials			
	Session 1	Session 2	Session 3	Session 4
	M (SD)	M (SD)	M (SD)	M (SD)
Relaxation during each imagined scene:	4.88 (1.0) a	4.47 (1.2) b	4.88 (1.0) a	5.12 (1.0) a
Amount of time spent imagining each scene:	3.63 (1.1) a	3.36 (1.3) a	3.73 (0.9) a	4.23 (1.2) b
Vividness of each imagined scene:	3.50 (1.2) ab	3.30 (1.3) b	3.77 (0.9) ad	4.23 (1.1) cd
Absorption in each imagined scene:	3.75 (1.2) ab	3.51 (1.2) a	3.87 (1.0) ab	4.16 (1.3) b
Note: Within rows, means that share a common subscript fail to differ significantly at $\alpha = .05$.				

Within-session subjective ratings of treatment manipulations among the Placebo group ($N = 13$) were analysed in separate ANOVAs, with one within factor (Trials: Sessions 1/2/3/4). No significant difference across sessions was shown for any of the four subjective ratings--Tingling Sensations: $F(3,36) = 1.17$, ns; Prickly Feelings, Numbness, and Warmth: $F(3,36) < 1$ in all cases.

It was also of theoretical interest to determine whether reductions in CTH activity were associated with a pattern of reduction over Trials in physiologic indices of tension/relaxation. Two sets of analyses were used to study treatment-induced changes in frontalis muscle tension, and cardiovascular arousal. First, resting EMG activity in the frontalis muscle was assessed at Baseline and Follow-up trials. The most meaningful classification schema for this analysis was to group subjects according to (a) whether or not headache activity was reduced over treatment intervals, and (b) whether reduced headache activity was manifest at Post-Treatment and/or Follow-up. The second set of analyses examined changes in cardiovascular functioning (HR, SBP, DBP) within and across Baseline, the four treatment sessions, and the 8-Week Follow-up. Only subjects in the Imagery and Placebo conditions were included in these analyses, since exposure to treatment sessions was exclusive to those groups.

Potential treatment-induced changes in EMG frontalis muscle tension were studied in a 3 (Groups: No CTH Reduction/CTH Reduction at Post-Treatment/CTH Reduction at Follow-up) $\times$ 2

(Trials: Baseline/Follow-up) X 5 (Intervals: 5 successive 1-minute segments within-sessions) Mixed ANOVA. The dependent variable for the repeated measures was the resting frontalis EMG, integrated over five successive 1-minute intervals. It was theoretically important to determine if there was a significant main effect for the Grouping Factor, or if Groups interacted significantly with the Trials or Intervals factor. This finding would have provided evidence of treatment-induced physiologic changes. In fact, the F-ratio was either less than 1 or nonsignificant for all analyses involving the Grouping Factor. Table 19 illustrates the relevant statistics in this analysis.

Table 19

Mixed ANOVA Summary for Frontalis EMG Data

Analysis	ANOVA Summary Table		
	F	(DF)	p
Group (No CTH Reduction, Post-Treatment CTH Reduction, Follow-up CTH Reduction)	0.09	(2,44)	ns
Group X Trials (Baseline, Follow-up)	0.03	(2,44)	ns
Group X Intervals (Minute 1,2,3,4,5)	0.72	(8,176)	ns
Group X Trials X Intervals	1.23	(8,176)	ns
Trials X Intervals	1.52	(4,176)	ns
Trials	5.65	(1,44)	.02
Intervals	0.75	(4,176)	ns

The only significant finding from the Frontalis EMG analysis was the main effect for the Trials Factor. The marginal means indicated that subjects on average demonstrated less Frontalis muscle tension at Baseline vs. Follow-up. Table 20 presents these data.

Table 20

Resting Frontalis EMG Activity for all Subjects (N=47)
at Baseline and Follow-up

Treatment Interval	Frontalis EMG Activity	
	M	(SD)
Baseline	14.09	(7.77)
Follow-up	16.35	(7.31)

The possibility of treatment-induced effects on cardiovascular functioning was assessed in a 2 (Condition: Imagery/Placebo) X 6 (Trials: Baseline/Sessions 1/2/3/4/Follow-up) X 2 (Intervals: Pre-/Post-Session) Mixed MANOVA. SBP, DBP, and HR were the dependent variables. This multivariate analysis was conducted in order to guard against Type 1 error. Once again, the theoretically important aspects of the analysis concerned whether the Condition factor interacted significantly with the within factors, or if it evidenced a significant main effect. Table 21 summarizes the MANOVA statistics.

Table 21

Summary Table of Mixed MANOVA on Cardiovascular Functioning

Analysis	MANOVA Summary Table		
	Mult. F	(DF)	p
Condition (Imagery, Placebo)	0.68	(1, 34)	ns
Condition X Trials (Baseline, Sessions 1-4, Follow-up)	1.07	(15, 20)	ns
Condition X Intervals (Pre-, Post-session)	1.13	(3, 32)	ns
Trials X Intervals	2.84	(15, 20)	.02
Condition X Trials X Intervals	1.00	(15, 20)	ns

The only significant univariate finding that accounted for the multivariate Trials X Intervals interaction was evidenced for Heart rate-- $F(5, 170) = 3.12$, $p < .01$. SBP and DBP had the following nonsignificant interactions-- $F(5, 170)$, $= 1.80$, ns; $F(5, 170) = 1.20$, ns; respectively. Table 22 presents the relevant means and standard deviations for the Heart rate data.

Table 22

Mean Within-Session Measures of HR. Collapsing Across Imagery and Placebo Groups (N=36)

Trials	Session Intervals			
	Pre-Session		Post-Session	
	M	(SD)	M	(SD)
Baseline	70.24 ¹ abc	(8.84)	68.61 ¹ a	(9.93)
Session 1	74.92 ¹ ac	(12.30)	67.94 ² a	(9.12)
Session 2	73.39 ¹ a	(11.07)	68.97 ¹ a	(9.13)
Session 3	72.83 ¹ a	(12.76)	66.94 ² a	(8.99)
Session 4	76.17 ¹ ac	(10.85)	69.81 ² a	(10.45)
Follow-up	67.21 ¹ bc	(8.32)	66.64 ¹ a	(8.63)

Note: Within columns, means sharing a common subscript fail to differ significantly from one another at $\alpha = .05$
 Within rows, means sharing a common superscript fail to differ significantly at $\alpha = .05$.

Simple main effects were initially calculated for pre-/post-session intervals across Trials. Mean Pre-Session HR readings (collapsed across Imagery and Placebo subjects) demonstrated a significant simple main effect over Trials-- $F(5,170) = 3.53, p < .01$. The LSD post hoc comparisons in Table 22 indicated that Pre-session HR readings were significantly lower at Follow-up, in comparison to the Pre-session HR readings for each of the four treatment sessions. Pre-session HR at Baseline was also significantly lower than the pre-session levels recorded for Sessions 1 and 4. It is likely that nonspecific factors associated with the Baseline and Follow-up assessments were responsible for these differences, given the absence of a change in HR across treatment sessions. The Post-session HR readings did not show a significant simple main effect across the Trials Factor-- $F(5,170) = 0.49, ns$.

The data in Table 22 were examined next for simple main effects across pre- and post-session intervals, at each level of the Trials factor. Significant decreases from pre- to post-session HR were observed in the following sessions: Session 1, $F = 8.06, p < .01$; Session 3, $F = 5.75, p < .05$; and Session 4, $F = 6.70, p < .05$ ($df = 1,34$ for each session). These findings indicated that subjects in the Imagery and Placebo conditions generally reduced HR activity during treatment sessions.

The physiologic data was theoretically interesting insofar as it demonstrated that mean reduction in headache activity was not attributable to a pattern of treatment-induced effects across

Trials. Those analyses also demonstrated that the active treatment condition (Imagery training) was not distinct from the Placebo condition in inducing potentially therapeutic reductions in physiologic activity. In sum, the above analyses indicated that the relationship between exposure to treatment conditions and resting Frontalis EMG or cardiovascular functioning was attributable to nonspecific or random effects. In no case was there a pattern of physiologic findings that could be meaningfully related to the treatment-induced reduction in CTH activity.

Discussion: Experiment 2

Data analyses for Experiment 2 replicated the main findings of Experiment 1. Of primary importance was the fact that Hypnotic and Nonhypnotic response-based imagery training were efficacious treatments for CTH. Hypotheses that immediately pertain to this outcome will be discussed in the present section.

Empirical findings confirmed the hypothesis that Hypnotic and Nonhypnotic Imagery groups would not differ significantly on mean ratings of daily headache activity (i.e. headache index) across Trials. The absence of a significant difference in treatment outcome prompted the question of whether the treatment-induced (Hypnotic/Nonhypnotic) processes were essentially similar. This issue will be reviewed in the General Discussion.

There was partial support for the second hypothesis. This hypothesis stated that Imagery subjects would significantly reduce headache activity (headache index) across Trials, while Placebo and Monitoring Control subjects would demonstrate no reduction. In fact, the Hypnotic/Nonhypnotic Imagery group reduced headache activity at post-treatment, while Control subjects exhibited an increase and Placebo subjects showed no change. Nonsignificant differences from the Baseline level of headache activity was observed for all three of these groups by the 8-week follow-up. This suggested that Imagery training was only temporarily successful in reducing CTH activity. Two supplemental analyses were therefore conducted. First, Chi-Square analyses demonstrated

that significantly more subjects in the Imagery condition (vs. Placebo and Control groups) reduced their Baseline level of headache activity across Post-treatment and the 8-week Follow-up. Second, among subjects who reduced headache activity, the percentage of improvement on the mean daily headache index was significantly greater across Trials for the Imagery group (vs. Placebo and Control groups combined). These analyses indicated that response-based imagery training was instrumental in significantly reducing CTH activity at Post-treatment, and it was superior to the combined Placebo and Control conditions in effecting headache reduction over Trials.

It was further hypothesized that the treatment-induced reduction in headache activity would not be attributable to an alteration in pattern of medication consumption over treatment intervals. This change would have confounded the previous findings regarding treatment efficacy. Data analyses supported this hypothesis. The Imagery group also demonstrated significantly less use of medication across Trials, as compared to Monitoring Controls. Given the confirmation of the above hypotheses, it was reasonable to attribute reduction in headache activity to the treatment-induced effects of response-based imagery training.

The next two hypotheses dealt with the relationship between treatment-induced headache improvement and actual changes in physiologic functioning. It was hypothesized that headache reduction would not be associated with a reduction across Trials in

levels of Frontalis EMG. This assertion was supported by the empirical findings. On average, resting Frontalis EMG was significantly lower at Baseline as compared to the 8-week Follow-up. This finding corroborated reports from numerous investigations that have failed to observe a direct relationship between muscle tension and headache activity. Similarly, it was hypothesized that improvement in headache activity would not be attributable to changes in cardiovascular functioning (SBP, DBP, HR). Testing of this hypothesis entailed the assessment of cardiovascular functioning across Trials for subjects in the Imagery and Placebo conditions. Pre-session HR was the only cardiovascular measure to differ significantly over sessions. An interpretable pattern across Trials did not emerge for this variable, thereby providing support for the hypothesis. Pre-session HR was significantly lower at Baseline and Follow-up, as compared to levels recorded for the four treatment sessions. These data suggested that Placebo and Imagery subjects had not inadvertently learned to lower HR as a function of exposure to treatment. Given that all subjects were assessed in a semi-reclined position, following approximately 10 minutes of rest, the significant changes in pre-session HR across Trials was likely attributable to nonspecific factors--e.g. random changes in HR over time, room temperature, time of appointments, etc.

A supplemental hypothesis predicted that Hypnotic and Nonhypnotic Imagery subjects would not differ significantly in mean ratings of the degree to which they experienced within-session

experimental effects--i.e. relaxation, imagery vividness, absorption in imagery, and percentage of time spent imagining imagery scenes. Data analyses provided clear evidence for this hypothesis. This finding was theoretically significant insofar as it suggested that the treatment-induced processes that mediated headache reduction were essentially the same for Hypnotic and Nonhypnotic Imagery subjects. The implications of this finding will be presented in the General Discussion.

Results: Experiments 1 and 2 Combined

The data analyses that remain to be presented, from experiments 1 and 2 combined, concern the relationship between treatment-induced improvement in headache activity and the following measures: hypnotic susceptibility (CURSS: Spanos et al., 1983), the propensity to engage in vivid imaginative activity (VMI: Shor et al., 1966), the predisposition to report physical symptoms (PILL: Pennebaker, 1982), and social desirability/defensiveness (MCSDS: Strahan & Gerbasi, 1972). Subjects scoring high on one or more of these factors might have demonstrated significant differences in how they responded to treatment, or how they reported headache activity (e.g. Brown, 1984). A second area of interest concerned whether treatment-induced reductions in headache activity significantly covaried with pre- and/or post-treatment illness attributions (Turk et al., 1986). The appraisal of recurrent headache as a "health problem" per se had not been directly examined in previous headache research. These analyses were conducted on Hypnotic and Nonhypnotic Imagery subjects from the two experiments, since only these groups demonstrated treatment-induced reductions in headache activity. The difference between mean daily headache index ratings at Post-treatment vs. Baseline served as the measure of headache reduction.

The relationship between headache reduction and the above-noted personality traits was examined by zero-order Pearson Product Moment correlations. The sample size for these correlations ranged

between 48 and 52 subjects. A maximum sample would have been 53 subjects from the Imagery conditions. This number was reduced due to missing data and/or the expressed disinterest in participating in "supplemental" assessments. Recall that in order to guard against biasing subjects' reported levels of headache reduction, hypnotic susceptibility, and involvement in vivid imagination, the later two measures were administered at the end of the last Follow-up session. This was done under two pretexts: (a) that the administration of these measures was part of a separate project that was updating behavioral norms for hypnotic susceptibility among university students; and consequently (b) that assessments of hypnotic susceptibility and imaginative involvement were not related to the headache project, and participation was voluntary. Table 23 summarizes the correlations between headache reduction and the above-noted assessments.

Table 23

Correlations Between Hypnotic Susceptibility and Personality Traits with Headache Reduction--A

Trait Measures	Correlation with Headache Reduction at Post-treatment		
	r	n	p
CURSS-O	.0708	48	ns
CURSS-S1	.0114	48	ns
CURSS-OI	.0455	48	ns
VMI	.0473	52	ns
PILL	-.0016	50	ns
MCSDS	.1171	50	ns

Table 23 illustrates that headache reduction was not significantly correlated with trait measures of vividness of mental imagery (VMI), physical symptom reporting (PILL: Appendix K), and social desirability/defensiveness (MCSDS). Headache reduction also failed to correlate significantly with various dimensions of hypnotic susceptibility. As noted in the Dependent Measures section of this dissertation, the CURSS measured the overt response to hypnotic suggestions (CURSS-O), the vividness of the subjective responses to hypnotic suggestions (CURSS-S1), and the perceived involuntariness of successful responses to hypnotic suggestions (CURSS-OI). The absence of significant correlations between

treatment-induced headache reduction and the PILL or the MCSDS indicated that the experimental effect was not confounded with the degree to which Imagery subjects were socially defensive, or predisposed to report physical symptoms. Appendix Q presents the intercorrelations for the variables utilized in the above analysis. The failure to observe a significant correlation between headache reduction and either hypnotic susceptibility or imagery vividness was not attributable to the fact that Nonhypnotic Imagery subjects were included in this analysis. Table 24 presents the results from separate correlational analyses for Hypnotic and Nonhypnotic Imagery subjects. The results are essentially the same for both groups--i.e. headache reduction was not significantly correlated with hypnotic susceptibility and imagery vividness. The nonsignificant correlations between headache reduction, PILL, and MCSDS are also included.

Table 24

Correlations Between Hypnotic Susceptibility and Personality
Traits with Headache Reduction--B

Trait Measures	Hypnotic Imagery Subjects: Correlation with Headache Reduction at Post-treatment		
	<u>r</u>	<u>n</u>	<u>p</u>
CURSS-O	.0087	23	ns
CURSS-S1	-.1696	23	ns
CURSS-OI	-.1631	23	ns
VMI	-.0539	26	ns
PILL	-.1229	24	ns
MCSDS	.0235	23	ns

Trait Measures	Nonhypnotic Imagery Subjects: Correlation with Headache Reduction at Post-treatment		
	<u>r</u>	<u>n</u>	<u>p</u>
CURSS-O	.1674	25	ns
CURSS-S1	.2883	25	ns
CURSS-OI	.3057	25	ns
VMI	.1716	26	ns
PILL	.1345	26	ns
MCSDS	.1826	27	ns

The association between treatment-induced headache reduction (among Imagery subjects) and illness schema of recurrent headache as a health problem was examined next. It was of particular interest to assess whether headache reduction was predicted by pre-treatment illness beliefs, and also whether post-treatment illness beliefs added a significant increment to the variance explained in treatment outcome. The IMIQ (Turk et al., 1986) operationally defined illness schema in terms of four dimensions--i.e. personal responsibility for the health problem, seriousness of the illness, changeability of the problem, and controllability.

A hierarchical regression analysis was undertaken to examine how headache reduction was associated with Baseline and Follow-up measures of illness schema. The mean percentage of improvement in headache activity over Trials served as the criterion variable (Blanchard & Andrasik, 1985). Two hierarchical levels of predictor variables were utilized. Baseline measures of affective distress and illness beliefs competed for entry into the regression equation at the first hierarchical level. These baseline measures included: perceived stress over the previous month (PSS: Cohen et al., 1983); trait anxiety (Spielberger et al., 1970); self-esteem (Rosenberg, 1979); and the four scales of the IMIQ (Turk et al., 1986). The second level of predictor variables was comprised of the post-treatment IMIQ scales. Complete data on these 11 predictor variables was available for 51 of the 53 Imagery subjects. Since this assessment was exploratory in nature, the minimum case-to-

variable ratio of 4 or 5 cases per predictor variable was considered appropriate (Tabachnik & Fidell, 1983). The intercorrelations among variables used in the hierarchical regression analysis are presented in Appendix R. Table 25 presents the summary data for the regression analysis.

Table 25

Hierarchical Regression of Baseline and Post-treatment Cognitive Measures on Headache Reduction Among Imagery Subjects

Predictor Variables	Mult. R-Sq.	F to Enter	df	Partial Corr.
Step 1 - PSS	.0971	5.27	1,49	.3117
Step 2 - Self-Esteem	.2636	10.85	2,48	.4294
Step 4 - Follow-up IMIQ: Changeability	.3135	3.42	3,47	.2604
Step 5 - Follow-up IMIQ: Seriousness	.3591	3.27	4,46	-.2578
- - - - -	- - - - -	- - - - -	- - - - -	- - - - -

* $p < .05$

** $p < .01$

Only two baseline measures significantly predicted the mean percentage of post-treatment headache reduction. These were the perceived severity of stress over the month prior to treatment, as well as the degree of positive self-esteem. Illness schema dimensions significantly accounted for the variance in headache reduction only when assessed at the Follow-up interval. These measures indicated that following exposure to treatment, the mean percentage of headache reduction was associated with the degree to which headaches were believed to be a health problem that was changeable, and not serious. These cognitive factors accounted for approximately 36% of the variance in treatment-induced headache reduction.

General Discussion

Three issues stand out among the findings of these two experiments. First, the empirical evidence supported the claim that hypnotic and nonhypnotic methods of response-based imagery training were efficacious in enhancing the self-management of recurrent headache. Imagery training successfully induced lower ratings of daily headache activity over Trials.

The findings of Experiments 1 and 2 also indicated that the treatment-induced reduction of headache activity was not attributable to an alteration in the pattern of medication consumption, or to actual changes in physiologic functioning (Frontalis EMG, SBP, DBP, HR). The use of Monitoring Control groups in both studies indicated that the reduction in headache activity over Trials was not likely a random or nonspecific effect. The use of a Placebo group in Experiment 2 further supported the view that headache reduction over Trials was due to exposure to imagery-specific processes. The Placebo condition controlled for the effect of presenting subjects with a treatment that was defined as a means for acquiring "skills" that would control the impact of recurrent headaches on daily activity. Finally, the absence of a significant correlation between headache reduction and social defensiveness or the propensity to report physical symptoms provided evidence that the reported treatment effects were valid.

The second issue of importance concerned the relationship between illness beliefs and the mean percentage of treatment-

induced headache reduction. It was of interest to determine whether treatment-induced headache reduction would covary significantly with Baseline and Follow-up assessments of beliefs about recurrent headache as a health problem per se. The hierarchical regression analysis found that treatment-induced headache reduction was significantly predicted by baseline assessments of positive self-esteem, and perceived stress during the month preceding treatment. These variables may have reflected the degree to which subjects were motivated to reduce their recurrent headache activity. Self-reported perceived stress (at Baseline) indicated that subjects were affectively distressed by their exposure to stressors at the time of enrollment in this study. Positive self-esteem suggested that subjects who reduced headache activity may have been generally optimistic about treatment outcome. Illness beliefs contributed significantly to the variance explained in headache reduction only when assessed at Follow-up. Thirty-six percent of the variance in headache reduction was predicted by Baseline self-esteem and perceived stress, as well as by the Post-treatment beliefs that recurrent headache was a health problem that was changeable, and not serious.

The finding of the regression analysis highlighted the fact that illness beliefs did not mediate treatment outcome. Rather, the fact that only Follow-up IMIQ measures covaried with headache reduction suggest two possible explanations. Actual headache activity may have causally influenced the illness schema that

subjects had about recurrent headache as a health problem. Alternatively, there may have been a reciprocal relationship between illness schema and headache reduction. In both accounts, treatment-induced headache reduction was not simply an outcome variable that was shaped by cognitive factors. This phenomenon runs somewhat contrary to the cognitive-behavioral account of treatment efficacy (e.g. Holroyd et al., 1984). The cognitive-behavioral model has been formulated on the basis of empirical findings that emphasize a unidirectional, causal influence of cognitive factors on headache activity. These cognitive factors include outcome and efficacy expectations, as well as perceived vs. actual success in performing treatment-related skills (Andrasik & Holroyd, 1980; 1983; Holroyd et al., 1984). The current findings suggest the need for future research to specify how illness beliefs and headache activity may be reciprocally and mutually influenced--perhaps in relation to one or more other factors (e.g. social modelling influences, practice effects, other cognitive factors, etc.).

The third and final issue of importance concerned the fact that treatment-induced headache reduction was not significantly correlated with either hypnotizability (Spanos et al., 1983), or the trait disposition to engage in vivid imaginative activity (Shor et al., 1966). This finding confirmed the hypotheses from both experiments. These data replicated and extended the previous findings of Brown (1984), who reported that headache reduction was not associated with two trait measures of imaginative involvement.

The findings from experiments 1 and 2 were more consistent with the social psychological (Spanos, 1982; 1986) formulation of hypnotic analgesia, rather than with the neodissociation account (Hilgard, 1977). As previously outlined, neodissociation theory would have predicted a significant association between hypnotic susceptibility and treatment outcome among Imagery subjects. According to this theory, response-based Imagery training should have enabled high susceptible subjects to slip into a "special state" of hypnotic consciousness, and thereby attain greater reduction in headache activity. The neodissociation position was contradicted, therefore, by the absence of a significant correlation between treatment outcome and hypnotic susceptibility among Hypnotic and Nonhypnotic Imagery groups.

In contrast to neodissociation theory, the absence of a significant correlation between treatment-induced headache reduction and either hypnotic susceptibility or trait imagery was consistent with the social psychological formulation. Spanos (1982, 1986) argued that low susceptible hypnotic subjects are capable of reducing pain to the same degree as high susceptible subjects, providing that both groups are exposed to situational demands that facilitate equivalent use of pain-attenuating strategies. This formulation emphasizes the role of social contingencies that mediate treatment outcome. It also highlights the assertion that personality measures are by themselves unreliable predictors of responsiveness to treatment. The data

from Experiments 1 and 2 suggested that Imagery sessions did in fact induce a uniformly high response pattern among Hypnotic and Nonhypnotic Imagery groups. These groups did not differ in their self-reports of a moderate to high degree of treatment-induced relaxation, imagery absorption, etc. Given that both groups were involved to a similar degree in the treatment sessions, it was consistent with the social psychological formulation of pain control that Hypnotic and Nonhypnotic treatment modes would be equally efficacious. The credibility of this finding was strengthened by the fact that hypnosis and trait imagery were assessed in a manner that was designed to control for a response bias. These assessments were administered at Follow-up, and for the purpose of completing a project that was identified as being unrelated to the treatment process and/or treatment outcome.

In sum, Experiments 1 and 2 replicated and extended the findings reported by Brown (1984). These experiments made novel contributions to the current investigative literature regarding cognitive treatments for recurrent headache in the following ways: (a) Hypnotic and Nonhypnotic response-based imagery training was utilized with subjects who presented with symptoms of CMH, CTH, and Mixed CMH/CTH headache; (b) the conclusion regarding the efficacy of response-based imagery training was supported methodologically by including Placebo and Monitoring Control groups in the experimental design; (c) physiologic, behavioral, and cognitive data were collected simultaneously, to provide a multidimensional

assessment of treatment outcome and treatment-induced processes;
and (d) this was the first research project to examine the
relationship between treatment-induced headache reduction and the
illness schema that subjects had regarding recurrent headache as a
health problem.

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Name: _____ Date: _____
Note: Key, comments, and ratings on back.

[illegible]

Circle your level of pain at breakfast, lunch, supper, and bedtime.
Put an X through the times you take pain medication.

- 5 = Extremely intense headache, incapacitated
4 = Severe headache, difficult to concentrate, can do undemanding tasks
3 = Moderate headache, pain is noticeably present
2 = Mild headache, could be ignored
1 = Very mild headache, aware of it only when attending to it
0 = No headache

Headache:	Time		
	Began	Ended	
#0 _____			
#1 _____	_____	_____	Medication(s): _____
#2 _____	_____	_____	Medication(s): _____
#3 _____	_____	_____	Medication(s): _____

On the 0 to 5 Intensity Scale, your expected score for tomorrow is:

Overall Stress Rating for Today: _____

0 (Not at all Stressful) _____ 10 (Extremely Stressful)

Treatment Efficacy Ratings

NAME: _____

DATE: _____

SESSION #: _____

On the following pages, please place an "X" in the space that corresponds to your actual experience.

(NOTE: EACH QUESTION WAS PRESENTED ON A SEPARATE PAGE)

Imagery and Hypnosis Groups

1. During the previous exercise, I felt relaxed.

Not at all relaxed _ _ _ : _ _ _ : _ _ _ : _ _ _ : _ _ _ : _ _ _ : _ _ _ Totally relaxed

2. During the previous exercise, I was absorbed in the scene.

Not at all absorbed _ _ _ : _ _ _ : _ _ _ : _ _ _ : _ _ _ : _ _ _ : _ _ _ Totally absorbed

3. During the previous exercise, the amount of time in which I was imagining the scene was:

Not at all _ _ _ : _ _ _ : _ _ _ : _ _ _ : _ _ _ : _ _ _ : _ _ _ All of the time

4. During the previous exercise, I experienced the scene as being vivid to the following degree:

Not at all vivid _ _ _ : _ _ _ : _ _ _ : _ _ _ : _ _ _ : _ _ _ : _ _ _ Totally vivid

(HYPNOSIS GROUP ONLY)

5. From the time that the hypnotic induction procedure ended, to the time that you awoke, how deeply hypnotized were you?

Not at all hypnotized _ _ _ : _ _ _ : _ _ _ : _ _ _ : _ _ _ : _ _ _ : _ _ _ Very deeply hypnotized

Placebo Group

1. During the previous exercise, I experienced tingling sensations in my neck, shoulders, face or forehead to the following degree:

Not at all _____ Extremely
noticeable : : : : : : Intense

2. During the previous exercise, I experienced prickly feelings in my neck, shoulders, face or forehead to the following degree:

Not at all _____ Extremely
noticeable : : : : : : Intense

3. During the previous exercise, I experienced numbness in my neck, shoulders, face or forehead to the following degree:

Not at all _____ Extremely
noticeable : : : : : : Intense

4. During the previous exercise, I experienced warmth in my neck, shoulders, face or forehead to the following degree:

Not at all _____ Extremely
noticeable : : : : : : Intense

PHYSIOLOGICAL ASSESSMENT

(Attach EMG Headband and BP Cuff)

1. Adaptation - Subject sits semi-reclined, eyes closed, relaxing
(10 minute interval)
 - 1st SBP, DPB, HR assessment.....minute 10
 - 2nd SBP, DBP, HR assessment.....minute 15
2. Baseline EMG Assessment
 - integrated EMG for 5 measures of
1-minute intervals.....minute 20
3. Final SBP, DBP, HR Assessment.....minute 21

Self-Evaluation Questionnaire: Form 2

DIRECTIONS: A number of statements which people have used to describe themselves are given below. Read each statement and then put a check mark in the appropriate bracket to indicate how you generally feel. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe how you generally feel.

	Almost Never	Sometimes	Often	Almost Always
1. I feel pleasant	()	()	()	()
2. I tire quickly	()	()	()	()
3. I feel like crying	()	()	()	()
4. I wish I could be as happy as others seem to be	()	()	()	()
5. I am losing out on things because I can't make up my mind soon enough	()	()	()	()
6. I feel rested	()	()	()	()
7. I am "calm, cool, and collected"	()	()	()	()
8. I feel that difficulties are piling up so that I cannot overcome them	()	()	()	()
9. I worry too much over something that really doesn't matter	()	()	()	()
10. I am happy	()	()	()	()
11. I am inclined to take things hard	()	()	()	()
12. I lack self-confidence	()	()	()	()
13. I feel secure	()	()	()	()
14. I try to avoid facing a crisis or difficulty	()	()	()	()
15. I feel blue	()	()	()	()
16. I am content	()	()	()	()
17. Some unimportant thought runs through my mind and bothers me	()	()	()	()
18. I take disappointments so keenly that I can't put them out of my mind	()	()	()	()
19. I am a steady person	()	()	()	()
20. I get in a state of tension or turmoil as I think over my recent concerns and interests	()	()	()	()

M-C SDS

Listed below are a number of statements concerning personal attitudes and traits. Read each item and decide whether the statement is true (T) or false (F) as it pertains to you. Check the appropriate bracket. Go with your first judgment and do not spend too much time mulling over any question.

	TRUE	FALSE
1. I never hesitate to go out of my way to help someone in trouble.	()	()
2. I have never intensely disliked anyone.	()	()
3. I sometimes feel resentful when I don't get my way.	()	()
4. I like to gossip at times.	()	()
5. There have been times when I felt like rebelling against people in authority, even though I knew they were right.	()	()
6. I can remember "playing sick" to get out of something.	()	()
7. There have been occasions when I have taken advantage of someone.	()	()
8. I'm always willing to admit it when I make a mistake.	()	()
9. I always try to practice what I preach.	()	()
10. I sometimes try to get even rather than forgive and forget.	()	()
11. When I don't know something I don't at all mind admitting it.	()	()
12. I am always courteous, even to people who are disagreeable.	()	()
13. At times I have really insisted on having things my own way.	()	()
14. There have been occasions when I felt like smashing things.	()	()
15. I would never think of letting someone else be punished for my wrong-doings.	()	()
16. I never resent being asked to return a favor.	()	()
17. I have never been irked when people expressed ideas very different from my own.	()	()
18. There have been times when I was quite jealous of the good fortune of others.	()	()
19. I am sometimes irritated by people who ask favors of me.	()	()
20. I have never deliberately said something that hurt someone's feelings.	()	()

CONSIDER THE FOLLOWING HEALTH PROBLEM: RECURRENT HEADACHES

We would like you to respond to a number of statements about RECURRENT HEADACHES. Please record your responses by placing an "X" in the most appropriate blank on the scale list below each of the statements. This will indicate the level of your agreement or disagreement with each statement about this health problem.

1. This health problem is controllable.

Strongly Disagree ----:----:----:----:----:----:----:----:---- Strongly Agree

2. This health problem requires medical attention.

Strongly Disagree ----:----:----:----:----:----:----:----:---- Strongly Agree

3. This health problem is chronic (long lasting) rather than acute (short-lived).

Strongly Disagree ----:----:----:----:----:----:----:----:---- Strongly Agree

4. This health problem is disabling.

Strongly Disagree ----:----:----:----:----:----:----:----:---- Strongly Agree

5. This health problem is caused by changes in the weather.

Strongly Disagree ----:----:----:----:----:----:----:----:---- Strongly Agree

6. This health problem is painful.

Strongly Disagree ----:----:----:----:----:----:----:----:---- Strongly Agree

7. The symptoms of this health problem are similar to the common cold.

Strongly Disagree ----:----:----:----:----:----:----:----:---- Strongly Agree

8. This health problem is permanent rather than temporary.

Strongly Disagree ----- Strongly Agree

9. This health problem is cured by reduced stress.

Strongly Disagree ----- Strongly Agree

10. This health problem is caused by stress or nerves.

Strongly Disagree ----- Strongly Agree

11. This health problem goes away on its own.

Strongly Disagree ----- Strongly Agree

12. This health problem is caused by one's behavior.

Strongly Disagree ----- Strongly Agree

13. This health problem is cured by proper eating habits.

Strongly Disagree ----- Strongly Agree

14. This health problem is controllable by the individual.

Strongly Disagree ----- Strongly Agree

15. The presence of this health problem relates to something the individual did.

Strongly Disagree ----- Strongly Agree

16. This health problem is contagious.

Strongly Disagree ----- Strongly Agree

17. This health problem is caused by germs or virus.

Strongly Disagree ----:----:----:----:----:----:----:----:---- Strongly Agree

18. This health problem is caused by lack of rest.

Strongly Disagree ----:----:----:----:----:----:----:----:---- Strongly Agree

19. This health problem is serious.

Strongly Disagree ----:----:----:----:----:----:----:----:---- Strongly Agree

20. This health problem often comes back. (That is, even if recurrent headaches go away for a period of time, the headache activity will often return.)

Strongly Disagree ----:----:----:----:----:----:----:----:---- Strongly Agree

21. This health problem is changeable.

Strongly Disagree ----:----:----:----:----:----:----:----:---- Strongly Agree

22. This health problem is caused by poor diet.

Strongly Disagree ----:----:----:----:----:----:----:----:---- Strongly Agree

23. This health problem changes over time.

Strongly Disagree ----:----:----:----:----:----:----:----:---- Strongly Agree

24. This health problem is cured by physical exercise.

Strongly Disagree ----:----:----:----:----:----:----:----:---- Strongly Agree

THE CARLETON UNIVERSITY
RESPONSIVENESS TO SUGGESTION SCALE

Name _____ Date _____

Age _____ Sex _____ Occupation _____

Telephone _____

PLEASE DO NOT OPEN this booklet until specifically instructed
to do so.

Now, in your own words please list all of the suggestions you can remember. You have two minutes to do this.

PLEASE DO NOT TURN THIS PAGE until you are specifically instructed to do so.

PLEASE DO NOT RETURN TO PAGE 2.

PLEASE DO NOT TURN THIS PAGE until you are specifically instructed to do so.

PLEASE DO NOT RETURN TO EARLIER PAGES.

Now, in your own words please list all of the suggestions you can remember. You have two minutes to do this.

PLEASE DO NOT TURN THIS PAGE until you are specifically instructed to do so.

PLEASE DO NOT RETURN TO EARLIER PAGES.

Now, in your own words please list all of the suggestions you can remember. You have two minutes to do this.

PLEASE DO NOT TURN THIS PAGE until you are specifically instructed to do so.

PLEASE DO NOT RETURN TO EARLIER PAGES

SECTION ON OBJECTIVE, OUTWARD RESPONSES

Listed below in chronological order are the specific suggestions you were administered following the standard hypnotic induction procedure. We wish you to estimate whether or not you objectively responded to these suggestions, that is, whether or not an onlooker would have observed that you did or did not make certain definite responses by certain specific, predefined criteria. Thus, in this section we are interested in your estimates of your outward behavior and not in what your inner, subjective experience of it was like. Later on you will be given an opportunity to describe your inner, subjective experience, but in this section refer only to the outward behavioral responses irrespective of what the experience may have been like subjectively.

It is understood that in some cases your estimates may not be as accurate as you might wish them to be and that you might even have to guess. But we want you to make whatever you feel to be your best estimates.

Beneath a description of each of the six suggestions are sets of two responses, labeled A and B. Please circle either A or B for each question, whichever you judge to be the more accurate. Please answer every question. Make sure that you do not skip any questions. Please answer every one.

1. Arm Rising (Right Arm)

You were asked to extend your right arm straight out in front of you, and were told that your arm was rising into the air. Would you estimate that an onlooker would have observed that your arm had risen at least six inches (before the time you were asked to replace the arm in your lap)?

- Circle one:
- A. My arm rose at least six inches
 - B. My arm rose less than six inches

II. Arms Moving Apart

You were asked to extend your arms straight out in front of you with the fingers of one hand touching the fingers of the other. You were then told that your arms were moving apart. Would you estimate that an onlooker would have observed that your hands had moved apart by at least six inches (before the time you were asked to replace them in your lap)?

- Circle one: A. My hands had moved apart at least six inches
 B. My hands moved apart less than six inches

III. Arm Rigidity (Left Arm)

You were asked to hold your left arm straight out in front of you, and were told that the arm was becoming stiff and rigid and that it would not bend. You were then asked to try to bend it. Would you estimate than an onlooker would have observed that there was less than two inches of arm bending (before you were told that your arm was no longer stiff and to replace it in your lap)?

- Circle one: A. My arm was bent less than two inches by then
 B. My arm was bent two or more inches by then

IV. Arm Heaviness (Right Arm)

You were asked to place your right forearm on the table with your hand facing down. You were then told how heavy your arm and hand felt and asked to try to lift them from the table. Would you estimate that an onlooker would have observed that you did not lift your hand and arm at least one inch up from the table (before being told that your arm was no longer heavy)?

- Circle one: A. I did not lift my arm and hand at least one inch by then
 B. I did lift my arm and hand an inch or more by then

V. Experiencing Music

You were told that a record player had been brought into the room, that

you would hear it playing Jingle Belis, and that you would move your head to keep time to the tune. Would you estimate that an onlooker would have observed you to make recognizable movements of the head (before you were told that the record player had been turned off)?

Circle one: A. I did recognizably move my head to the tune

B. I did not recognizably move my head to the tune

VI. Experiencing a Kitten

You were told that a kitten was sitting in your lap. Then you were asked to look at the kitten and to pet it with your hand. Would you estimate that an onlooker would have observed you to make a petting movement with your hand?

Circle one: A. I did make a petting movement wit' my hand

B. I did not make a petting movement with my hand

PLEASE DO NOT RETURN TO EARLIER PAGES

SECTION ON SUBJECTIVE, INNER EXPERIENCES I

In this section we are interested in your inner subjective experiences instead of your outward behavior. We want to find out about what you experienced during each of the suggestions you were given. Please read each question carefully and answer it honestly. The outward response people make to a suggestion may or may not correspond to their inner experience. For example, take a person who's outward behavior is arm rising when given the suggestion that their arm is light and moving upward. In some cases a person may have experienced his or her arm as feeling light. In other cases, however, the person's arm may have moved upward even though it did not feel the least bit light. The important thing to keep in mind is that one type of experience is no better and no worse than the other. This is a scientific study and all we are interested in is getting at the truth of what people experience. So please be honest in answering each of the following questions. Please answer every question. Make sure that you do not skip any questions.

For each question choose the one alternative that best describes your experience.

I. Arm Rising (Right Arm)

You were told that your arm was feeling lighter and lighter and was rising in the air. You were asked to imagine that it was like a balloon and was being filled with air.

During this suggestion my arm felt light:

- (a) Not at all
- (b) To a slight degree
- (c) To a moderate degree
- (d) To a great degree

II. Arms Moving Apart

You were told that your outstretched arms were moving apart, and that they felt like a force was repelling them and pushing them apart.

During this suggestion my arms felt like a force was pushing them apart:

- (a) Not at all
- (b) To a slight degree
- (c) To a moderate degree
- (d) To a great degree

III. Arms Rigidity (Left Arm)

You were told that your outstretched left arm was becoming stiff, rigid, and unable to bend. You were asked to imagine the arm in a splint.

During this suggestion my arm felt stiff and rigid:

- (a) Not at all
- (b) To a slight degree
- (c) To a moderate degree
- (d) To a great degree

IV. Arm Heaviness (Right Arm)

You were told that your arm and hand were very heavy, so heavy that you couldn't lift them from the table. You were asked to imagine heavy weights placed on your hand and arm.

During this suggestion my arm and hand felt heavy:

- (a) Not at all
- (b) To a slight degree
- (c) To a moderate degree
- (d) To a great degree

V. Experiencing Music

You were told that you would hear the song Jingle Bells and that you would move your head in time with the music.

During this suggestion I felt like I was hearing the tune Jingle Bells:

- (a) Not at all
- (b) To a slight degree
- (c) To a moderate degree
- (d) To a great degree

VI. Experiencing a Kitten

You were told that you would see a kitten in your lap and that you would pet the kitten.

During this suggestion I felt like I was seeing a kitten:

- (a) Not at all
- (b) To a slight degree
- (c) To a moderate degree
- (d) To a great degree

VII. Forgetting the Suggestions

You were told that you would be unable to remember any of the suggestions you had been given until you heard a tap and the words "Now you can remember everything". You were told that you would be unable to remember the suggestions even when you were asked to try to remember.

During this suggestion I forgot the suggestions:

- (a) Not at all
- (b) To a slight degree
- (c) To a moderate degree
- (d) To a great degree

PLEASE DO NOT RETURN TO EARLIER PAGES

SECTION ON SUBJECTIVE, INNER EXPERIENCES 11

In this section we are interested in a particular class of subjective, inner experiences. We want to find out about the extent to which you experienced your outward behavior to each suggestion as happening automatically and without a feeling of effort. For example, take a person whose outward behavior is to not bend their arm when they are told that the arm is stiff and unable to bend. Such a person may have felt that his or her arm was unable to bend. For this person it may have felt like the arm became stiff and unable to bend all by itself. In other cases, however, a person may not bend their arm even though they know that they could have bent it if they chose to. This person would have had the feeling of voluntarily choosing not to bend the arm. Remember, one type of experience is no better and no worse than the other. We are equally interested in finding out about experiences that feel automatic and also about those that feel voluntary. All we are interested in is getting at the truth about what people experience. So, please be honest in answering each of the following questions. Please answer every question. Make sure you do not skip any questions. For each question choose the one answer that best describes your experience.

I. Arm Rising (Right Arm)

You were told that your arm was light and rising in the air.

During this suggestion my arm felt like it rose in the air by itself. I experienced this

- (a) Not at all
- (b) To a slight degree
- (c) To a moderate degree
- (d) To a great degree

Remember: if you chose to lift your arm voluntarily, or if your arm did not feel like it rose by itself, you should choose alternative (a)

II. Arms Moving Apart

You were told that your outstretched arms were moving apart.

During this suggestion my arms felt like they were moving apart by themselves.

I experienced this

- (a) Not at all
- (b) To a slight degree
- (c) To a moderate degree
- (d) To a great degree

Remember: If you chose to move your arm voluntarily, or if your arms did not feel like they moved by themselves, you should choose alternative (a).

III. Arm Rigidity (Left Arm)

You were told that your outstretched arm was becoming stiff and unable to bend.

During this suggestion my arm felt like it was unable to bend. I experienced this

- (a) Not at all
- (b) To a slight degree
- (c) To a moderate degree
- (d) To a great degree

Remember: If you chose voluntarily to not bend your arm, or if your arm did not feel unable to bend you should choose alternative (a).

IV. Arm Heaviness (Right Arm)

You were told that your arm and hand were too heavy to lift from the table.

During this suggestion my arm felt unable to be lifted. I experienced this

- (a) Not at all
- (b) To a slight degree
- (c) To a moderate degree
- (d) To a great degree

Remember: If you chose voluntarily to not lift your arm, or if your arm did not feel unable to lift you should choose alternative (a).

V. Experiencing Music

You were told that you would hear the song Jingle Bells.

During this suggestion the tune Jingle Bells seemed to occur automatically, without any effort on my part. I experienced this

- (a) Not at all
- (b) To a slight degree
- (c) To a moderate degree
- (d) To a great degree

Remember: If experiencing the tune seemed to take a good deal of effort on your part, or if you did not experience the tune choose alternative (a).

VI. Experiencing a Kitten

You were told that you would see a kitten in your lap.

During this suggestion the image of a kitten seemed to occur automatically, without any effort on my part. I experienced this

- (a) Not at all
- (b) To a slight degreee
- (c) To a moderate degree
- (d) To a great degree

Remember: If getting an image of a kitten seemed to take a good deal of effort, or if you did not get an image at all choose alternative (a).

VII. Forgetting the Suggestions

You were told that you would be unable to remember the suggestion you had been given.

During this time the suggestions seemed to disappear automatically from my memory, they seemed to just go away by themselves. I experienced this

- (a) Not at all
- (b) To a slight degree
- (c) To a moderate degree
- (d) To a great degree

Remember: If forgetting the suggestions seemed to require effort on your part, if you had to "try and forget", or if you felt that you did not forget anything, you should choose alternative (a).

Vividness of Mental Imagery

This questionnaire involves imagining certain situations. For each of the following questions, please indicate which of the responses in the key below best applies.

- a) perfectly clear, and as vivid as the actual experience
- b) very clear, and comparable in vividness to the actual experience
- c) moderately clear and vivid
- d) not clear or vivid, but recognizable
- e) vague and dim
- f) so vague and dim as to be hardly discernible
- g) no image present at all; you only know that you are thinking of it

Please circle one response for each question.

1. When you close your eyes and try to imagine a specific automobile which you have seen many times, how vividly can you imagine it?

a b c d e f g

2. When you keep your eyes open and try to imagine the automobile, how vividly can you imagine it?

a b c d e f g

3. When you close your eyes and try to imagine a color, such as a bright blue, how vividly can you imagine it?

a b c d e f g

4. When you keep your eyes open and try to imagine a patch of the wall becoming a different color, how vividly can you imagine the changed color?

a b c d e f g

5. When you close your eyes and try to imagine your name and address written on a paper before you, how vividly can you imagine seeing the words?

a b c d e f g

6. When you try to imagin hearing a tune, how vividly can you imagine it?

a b c d e f g

7. When you try to imagin yourself running, how vividly can you imagine yourself actually moving? (This refers to the feeling of your muscles in motion, not to visually seeing yourself running.)

a b c d e f g

8. When you try to imagine your arms as being especially heavy, how vividly can you imagine it?

a b c d e f g

9. When you try to imagine a common odor, such as a fragrant cup of hot coffee, how vividly can you imagine it?

a b c d e f g

10. When you try to imagine a common taste, such as the sweetness of sugar, how vividly can you imagine it?

a b c d e f g

11. When you try to imagine the room as becoming especially warm, how vividly can you imagine it?

a b c d e f g

12. When you try to imagine the room as becoming especially cold, how vividly can you imagine it?

a b c d e f g

13. When you imagine yourself stroking fur, how vividly can you imagine the fur? (This refers to the feeling of touching, not visually trying to see it.)

a b c d e f g

14. When you imagine how it feels when you have a common stomach ache, how vividly can you imagine it.

a b c d e f g

15. When you try to imagine the room spinning, how vividly can you imagine it.

a b c d e f g

PSS

The questions in this scale ask you about your feelings and thoughts during the last month. In each case, you will be asked to indicate how often you felt or thought a certain way. Although some of the questions are similar, there are differences between them and you should treat each one as a separate question. The best approach is to answer each question fairly quickly. That is, don't try to count up the number of times you felt a particular way, but rather indicate the alternative that seems like a reasonable estimate.

For each question choose from the following alternatives by putting a check mark over the appropriate number:

	(0) Never	(1) Almost Never	(2) Sometimes	(3) Fairly Often	(4) Very Often
1. In the last month, how often have you been upset because of something that happened unexpectedly?	0	1	2	3	4
2. In the last month, how often have you felt that you were unable to control the important things in your life?	0	1	2	3	4
3. In the last month, how often have you felt nervous and "stressed"?	0	1	2	3	4
4. In the last month, how often have you dealt successfully with irritating life hassles?	0	1	2	3	4
5. In the last month, how often have you felt that you were effectively coping with important changes that were occurring in your life?	0	1	2	3	4
6. In the last month, how often have you felt confident about your ability to handle your personal problems?	0	1	2	3	4
7. In the last month, how often have you felt that things were going your way?	0	1	2	3	4
8. In the last month, how often have you found that you could not cope with all the things that you had to do?	0	1	2	3	4
9. In the last month, how often have you been angered because of things that happened that were outside of your control?	0	1	2	3	4
10. In the last month, how often have you felt that you were on top of things?	0	1	2	3	4

11. In the last month, how often have you been
angered because of things that happened that
were outside of your control?	0	1	2	3	4
12. In the last month, how often have you found
yourself thinking about things that you have
to accomplish?	0	1	2	3	4
13. In the last month, how often have you been
able to control the way you spend your time?	0	1	2	3	4
14. In the last month, how often have you felt
difficulties were piling up so high that you
could not overcome them?	0	1	2	3	4

RSE Scale

Instructions: Please indicate your degree of agreement or disagreement with each of the following statements by circling the appropriate option for each statement.

SA = strongly agree

A = agree

D = disagree

SD = strongly disagree

1. On the whole I am satisfied with myself.	SA	A	D	SD
2. At times I think I am no good at all.	SA	A	D	SD
3. I feel that I have a number of good qualities.	SA	A	D	SD
4. I am able to do things as well as most other people.	SA	A	D	SD
5. I feel that I do not have much to be proud of.	SA	A	D	SD
6. I certainly feel useless at times.	SA	A	D	SD
7. I feel that I'm a person of worth, at least on an equal plane with others.	SA	A	D	SD
8. I wish I could have more respect for myself.	SA	A	D	SD
9. All in all, I am inclined to feel that I am a failure.	SA	A	D	SD
10. I take a positive attitude toward myself.	SA	A	D	SD

PILL

On the following pages several common symptoms or bodily sensations are listed. Most people have experienced most of them at one time or another. We are currently interested in finding out how prevalent each symptom is among university students. Please respond by checking the appropriate letter for each question.

For example, if your ears tend to ring once every week or so, you would check the brackets for "D" next to item # 3.

Never or almost never experience symptom	Less than 3 or 4 times per year	Every month or so	Every Week or so	More than once every week
(A)	(B)	(C)	(D)	(E)
<hr/>				
	(A)	(B)	(C)	(D) (E)
1. Eyes water	()	()	()	() ()
2. Itching or painful eyes	()	()	()	() ()
3. Ringing in ears	()	()	()	() ()
4. Temporary deafness . . . hard of hearing	()	()	()	() ()
5. Lump in throat	()	()	()	() ()
6. Choking sensations	()	()	()	() ()
7. Sneezing spells	()	()	()	() ()
8. Running nose	()	()	()	() ()
9. Congested nose	()	()	()	() ()
10. Bleeding nose	()	()	()	() ()
11. Asthma or wheezing	()	()	()	() ()
12. Coughing	()	()	()	() ()
13. Out of breath	()	()	()	() ()
14. Swollen ankles	()	()	()	() ()
15. Chest pains	()	()	()	() ()
16. Racing heart	()	()	()	() ()

3 of/de 3

MicroD

Never or almost never experience symptom	Less than 3 or 4 times per year	Every month or so	Every Week or so	More than once every week
(A)	(B)	(C)	(D)	(E)
17. Cold hands or feet even in hot weather			() () () () ()	
18. Leg cramps			() () () () ()	
19. Insomnia			() () () () ()	
20. Toothaches			() () () () ()	
21. Upset stomach			() () () () ()	
22. Indigestion			() () () () ()	
23. Heartburn			() () () () ()	
24. Severe pains or cramps in stomach			() () () () ()	
25. Diarrhea			() () () () ()	
26. Constipation			() () () () ()	
27. Hemorrhoids			() () () () ()	
28. Swollen joints			() () () () ()	
29. Stiff muscles			() () () () ()	
30. Back pains			() () () () ()	
31. Sensitive or tender skin			() () () () ()	
32. Face flushes			() () () () ()	
33. Severe itching			() () () () ()	
34. Skin breaks out in rash			() () () () ()	
35. Acne or pimples on face			() () () () ()	
36. Acne or pimples other than face			() () () () ()	
37. Boils			() () () () ()	
38. Sweat even in cold weather			() () () () ()	

Never or almost never experience symptom	Less than 3 or 4 times per year	Every month or so	Every Week or so	More than once every week
(A)	(B)	(C)	(D)	(E)
39. Strong reactions to insect bites			() () () () ()	
40. Headaches			() () () () ()	
41. Sensation of pressure in head			() () () () ()	
42. Hot flashes			() () () () ()	
43. Chills			() () () () ()	
44. Dizziness			() () () () ()	
45. Feel faint			() () () () ()	
46. Numbness or tingling in any part of body			() () () () ()	
47. Twitching of eyelid			() () () () ()	
48. Twitching other than eyelid			() () () () ()	
49. Hands tremble or shake			() () () () ()	
50. Stiff joints			() () () () ()	
51. Sore muscles			() () () () ()	
52. Sore throat			() () () () ()	
53. Sunburn			() () () () ()	
54. Nausea			() () () () ()	

Headache History and Symptoms

Name: _____ Age: _____

Phone #: _____ Male: () Female: ()

1. How often do you experience headaches?

- () one or less days per month
- () two days per month
- () one day per week
- () two days per week
- () more than two days per week

2. How long have you been experiencing headaches at the rate noted in the previous question?

- () less than three months
- () three to six months
- () six months to one year
- () one to two years
- () two to five years
- () more than five years

3. How would you rate the pain in your typical headache?

- () 5 Excruciating, incapacitating pain--the pain is so intense that you can do almost nothing
- () 4 Horrible, severe pain--the pain makes concentration difficult, but you can do some undemanding tasks
- () 3 Distressing pain--you are able to continue at your job or whatever you were doing
- () 2 Discomforting pain--you are able to ignore the pain at times
- () 1 Mild pain--you are aware of this low level of pain only when your attention is devoted to it
- () 0 No pain

4. What features characterize your typical headache?

- () pain on both sides of head
- () pain in the forehead area, though it may sometimes start at the back of the head or neck
- () pain is a persistent and dull ache
- () pain feels like a cap or band around head
- () sensations of tightness or pressure accompany headache
- () pain on only one side of the head
- () pain is a throbbing sensation
- () one or more of the following signs precede or accompany headache--parts of visual field disappear temporarily, flashing lights, strange odors, tingling sensations
- () nausea or vomiting usually accompanies headache
- () sensitivity to light accompanies headache

Telephone Screening Interview

Name: _____ Student #: _____ Age: _____

Are you?: ☐ Single
☐ Married/living with a common-law spouse

How many years of post-secondary education have you completed?
☐ none; ☐ 1; ☐ 2; ☐ 3; ☐ 4; ☐ 5 or more

1. At what age (approximately) did your headaches begin? _____

At what age did you think of your headaches as a "problem"? _____

2. Was the onset of headaches associated with any particular physical event--e.g. illness, injury, etc.? Y ☐ N ☐

Please specify: _____

3. What is your personal explanation for the cause of your headaches?
 Name as many causal factors that you feel are necessary.

4. How confident are you that you know the real cause of your headaches?

Select a number from 1 to 10: _____

(0)	(10)
Not at all	Totally
confident	Confident

Check here if you do not attribute the cause of your headaches to
any particular factor(s) _____

5. Did the onset of headaches occur at the same time as any particular life event? (e.g. graduating or failing a year at school, etc.)
 Y ☐ N ☐

Please Specify: _____

6. Do any members of your natural family experience recurrent headaches? Check all appropriate brackets.

☐ Father	☐ Mother
☐ Grandfather(s)	☐ Grandmother(s)
☐ Brother(s)	☐ Sister(s)
☐ Uncle(s)	☐ Aunt(s)

7. Have you ever sought medical attention for headaches? Y () N ()

At what age was medical attention first sought? _____

Were you referred to a specialist? Y () N ()

Please specify what kind of specialist(s) you saw:

6. What kind of diagnostic work have you received for headaches?

(Check every item that is applicable)

☐ General physical exam
☐ Neurological exam
☐ EEG--electrodes placed on your head to measure cortical activity
☐ Brain scan
☐ X-rays
☐ Other--please specify: _____

7. Have you ever been given a formal headache diagnosis? Y () N ()

If so, were you diagnosed by your Family Physician? Y () N ()

...if yes, what was the diagnosis? _____

Were you diagnosed by a Headache Specialist? Y () N ()

...if yes, what was the diagnosis? _____

8. How confident are you that your physician(s) has(have) identified the real cause of your headaches?

Select a number from 1 to 10: _____

(0)	(10)
Not at all	Totally
confident	Confident

Check here if your physician(s) did not explain the cause of your headaches: _____

9. What kind of medical treatment have you received for your headaches?
Check all appropriate categories.
- ☐ physiotherapy
 - ☐ psychotherapy (by a Psychiatrist or Psychologist)
 - ☐ relaxation training
 - ☐ biofeedback training
 - ☐ therapy by a Chiropractor
 - ☐ counselling from a Dietician or Nutritionist
 - ☐ prescribed medication
 - ☐ other -- specify _____
10. What medications do you currently take to control your headaches?
Check each applicable category, as well as the approx. dosage.
- ☐ Prescribed medication(s)
List all of these: _____
 - ☐ Non-prescribed drugs
List all of these: _____
 - ☐ Alcohol
Specify: _____
11. Approximately how many times have you sought out a professional in order to have your headaches diagnosed and/or treated? _____
12. What do you presently do in order to manage your headaches?

13. List ALL of the features below that characterize your typical headache.
- ☐ pain on both sides of the head
 - ☐ pain in the forehead area, though it may sometimes start at the back of the head or neck
 - ☐ pain is a persistent and dull ache
 - ☐ pain feels like a cap or band around the head
 - ☐ sensations of tightness or pressure accompany headache
 - ☐ pain on only one side of the head
 - ☐ pain is a throbbing sensation
 - ☐ one or more of the following signs precede or accompany headache--parts of visual field disappear temporarily, flashing lights, strange odors, tingling sensations
 - ☐ nausea or vomiting usually accompanies headache
 - ☐ sensitivity to light accompanies headache
14. Do you have any chronic illness/condition that may or may not be associated with your headaches? Y () N ()
If yes, please specify: _____

INFORMED CONSENT AGREEMENT

This project has been reviewed and approved by the Committee on Research with Human Subjects, in the Department of Psychology at Carleton University. It has been found to preserve the safeguards of subjects' privacy, welfare, and civil liberties.

I hereby agree to cooperate and participate in the research project entitled

The Treatment of Recurrent Headache

conducted by Robert P. Nolan, Amber Hayward, Heather Scott, and supervised by Professor Nicholas P. Spanos of the Psychology Department at Carleton University. I have informed the experimenter that I have no medical condition that would prohibit my participation in this project.

I acknowledge that I have been informed about my participation in this experiment. I understand that data reported from this study will only pertain to group characteristics--thereby protecting my individual privacy.

I understand that I have the freedom and right to withdraw from the experiment at any time, without question or reprimand by either the principal investigators or Carleton University.

Participant's Signature

Date

Principal Investigator

Protocol for Treatment Sessions

Hypnosis and Imagery Groups

Note: Given that the protocol for the Imagery and Hypnosis groups is essentially identical, distinctive features of the Hypnosis protocol will be noted by block letters.

We are going to teach you strategies that you can use to reduce the frequency and intensity of your headaches. Previous research has made it clear that stress is somehow related to headaches, and often a person's pain experience is increased by automatically engaging in a set of physical, emotional, and cognitive reactions to the headache. For example, when some people experience a headache coming on they might think to themselves such things as: "Oh God this is awful...I can't take this anymore...its never going to get better...." Thoughts like this actually increase the impact of the headache. These thoughts are commonly associated with feelings of being overly burdened with, or emotionally defeated by the headache. A cluster of physical symptoms also accompanies this reaction style. The symptoms are expressed in the form of tension in the skeletal muscles, increased heart rate, blood pressure, rate of breathing, and so on. This automatic reaction pattern can interfere with an individual's ability to carry out regular daily tasks. The result is that daily activities are experienced as being stressful, and this experience can further intensify headache activity. As you might expect, automatic reactions to headaches can take on many forms. However, numerous scientific studies have demonstrated that physical, emotional, and cognitive reactions to headaches can be significantly altered through therapies such as the one that you are about to learn.

The (HYPNOTIC) procedure you will be learning today has been used by physicians and psychologists to help people with a variety of pain syndromes, like back pain, arthritis, as well as headache.

PREVIOUS RESEARCH HAS DEMONSTRATED THAT YOUR ABILITY TO BE HYPNOTIZED DEPENDS ENTIRELY ON YOUR WILLINGNESS TO COOPERATE. IT HAS NOTHING TO DO WITH YOUR INTELLIGENCE. AS FOR YOUR WILL POWER--IF YOU WANT TO, YOU CAN PAY NO ATTENTION TO ME AND REMAIN AWAKE ALL THE TIME. ON THE OTHER HAND, IF YOU PAY CLOSE ATTENTION TO WHAT I SAY, AND FOLLOW THESE INSTRUCTIONS, YOU CAN EASILY LEARN TO FALL INTO A HYPNOTIC SLEEP. HYPNOSIS IS NOTHING FEARFUL OR MYSTERIOUS. IT IS MERELY A STATE OF STRONG INTEREST IN SOME PARTICULAR THING. IN A SENSE, YOU ARE HYPNOTIZED WHENEVER YOU SEE A GOOD SHOW AND FORGET YOU ARE PART OF THE AUDIENCE, BUT INSTEAD FEEL PART OF THE STORY. YOUR COOPERATION, YOUR INTEREST, IS WHAT I ASK FOR. YOUR ABILITY TO BE HYPNOTIZED IS A MEASURE OF YOUR WILLINGNESS TO COOPERATE. NOTHING WILL BE DONE THAT WILL IN ANY WAY CAUSE YOU THE LEAST EMBARRASSMENT.

Visualization--vividly imagining scenes and events--are part of this treatment. I will soon ask you to practice visualizing some commonplace scenes (WITH THE AID OF HYPNOSIS). Remember, this procedure is like daydreaming, but I'd like you to bring it more under your control (WITH THE AID OF HYPNOSIS), so that you can imagine specific events for a

given period of time. First, find a position that is comfortable for you. As you are sitting there, I will ask you to visualize some scenes. Imagine these situations as vividly as you can. Involve yourself fully in the image. I want you to imaginatively participate in each scene that is presented. For example, in the first scene I will ask you to imagine lying on a beach. I want you to imagine how you really feel as you are lying on the beach in this scene. The idea of a vivid image is that you get the feeling of a real, actual experience. You will hear the image described on tape. As you listen to the scene, create the image in your mind, doing exactly what you would do and feeling as you would feel in the real situation. When the description is finished, keep imagining the scene until I ask you to stop.

Baseline Physiologic Assessment (SBP, HR, RR)

Please close your eyes.

NOW I'M GOING TO BEGIN THE HYPNOTIC PROCEDURE--BRIEF PAUSE. RELAX COMPLETELY. RELAX EVERY MUSCLE OF YOUR BODY. RELAX THE MUSCLES OF YOUR LEGS. RELAX THE MUSCLES IN YOUR ARMS. MAKE YOURSELF PERFECTLY COMFORTABLE. LET YOURSELF BE LIMP. RELAX MORE AND MORE; MORE AND MORE. RELAX COMPLETELY. RELAX COMPLETELY. RELAX COMPLETELY.

YOUR LEGS FEEL HEAVY AND LIMP, HEAVY AND LIMP. YOUR ARMS ARE HEAVY, HEAVY, HEAVY AS LEAD. YOUR WHOLE BODY FEELS HEAVY, HEAVIER, AND HEAVIER. YOU FEEL TIRED AND SLEEPY, TIRED AND SLEEPY. YOU FEEL DROWSY AND SLEEPY, DROWSY AND SLEEPY. YOUR BREATHING IS SLOW AND REGULAR, SLOW AND REGULAR.

YOU FEEL PLEASANTLY DROWSY AND SLEEPY AS YOU CONTINUE TO LISTEN TO MY VOICE. JUST KEEP YOUR THOUGHTS ON WHAT I AM SAYING. YOU ARE GOING TO GET MUCH MORE DROWSY AND SLEEPY. SOON YOU WILL BE DEEPLY ASLEEP BUT YOU WILL HAVE NO TROUBLE HEARING ME. YOU WILL NOT WAKE UP UNTIL I TELL YOU TO. AT EACH COUNT YOU WILL FEEL YOURSELF GOING DOWN, DOWN, DOWN INTO A DEEP, COMFORTABLE, A DEEP RESTFUL SLEEP. A SLEEP IN WHICH YOU WILL BE ABLE TO DO ALL SORTS OF THINGS I ASK YOU TO DO... (EXPERIMENTER COUNTS SLOWLY TO TEN).

YOU ARE FEELING COMFORTABLE AND RELAXED, COMFORTABLE AND RELAXED, AND YOU ARE IN A DEEP, SOUND SLEEP--A DEEP, SOUND SLEEP--FULLY PREPARED TO RESPOND TO AND EXPERIENCE WHAT I WILL ASK YOU TO DO; ...FULLY PREPARED TO RESPOND TO EACH OF THE SUGGESTIONS WHICH I WILL NOW GIVE YOU.

Make sure that you are seated comfortably. Relax. Let your breathing take on a slow, deep, easy rhythm. Let any problems, worries, or concerns fade into the background. Let these fade away, more and more, every time you easily let out each breath.

I will now introduce the first imagined scene. Let the scene become as vivid as possible, and allow yourself to imaginatively participate in this scene. (Pause 10 seconds)

Response-Based Imagery Scene #1 (90 seconds)

You are lying on a beach near the ocean. You feel the hot sun on your skin. As you are lying in the sun you hear the ocean waves lapping onto the beach. Their rhythmic sound lulls you to a very quiet, almost drowsy state. You feel the tension draining away from your body. The muscles of your arms and hands feel so relaxed. Concentrate on the relaxation in your feet, legs, back, your neck and head. The muscles are completely relaxed... tension draining away... gone. The muscles in your face, around your eyes, mouth, the back of your head... so relaxed. Think about this feeling of relaxation. Any tension in your body is draining away. Your breathing becomes quiet and slow. You are totally calm... relaxed. You feel wonderful. (Brief pause)

Now stop imagining the scene. Please open your eyes (BUT REMAIN HYPNOTIZED). Try to not disrupt your general feeling of relaxation. We now want you to report on how vividly you experienced this scene. Please pick up the questionnaire and pen that are at your side. Rate your experience according to the few questions in front of you. Do not talk during this segment.

1st Treatment Efficacy Rating (Appendix B)

Please close your eyes once more. Let your body assume a comfortable position. Allow yourself to (DRIFT EVEN DEEPER INTO HYPNOSIS AND) become even more relaxed. Relax all the muscles in your body. Let each muscle become heavier and heavier... any tightness will fade away as your breathing becomes quiet and slow. You are relaxing deeper, and deeper.

We will do another scene in a moment. Remember, I want you to learn how to experience vivid imagery, through your participation in what you (HYPNOTICALLY) imagine. Just as in the previous scene, I want you to include your own actual responses in the imagery that is about to be presented. This will make the image more vivid, that is, more like the real experience of the scene I present. Don't worry if the previous scene wasn't very vivid for you. Some people are initially better than others at this, but repetition and practice has been shown in research to help everyone gradually imagine events as if they were really happening.

Please prepare yourself to imagine the scene now. Allow the (HYPNOTIC SUGGESTIONS) instructions to enable you to create the imagined scene in your mind... doing exactly what you would do in the real situation. When I finish the description, keep imagining the scene until I tell you to stop. Let's do the scene now.

Repeat Response-Based Imagery Scene #1

Now stop imagining the scene. Please open your eyes (BUT REMAIN HYPNOTIZED). Try to not disrupt your general feeling of relaxation. We now want you to report on how vividly you experienced this scene. Please pick up the questionnaire and pen that are at your side. Rate your experience according to the few questions in front of you. Do not talk during this segment.

2nd Treatment Efficacy Rating (Appendix B)

Please close your eyes once more. Let your body assume a comfortable position. Allow yourself to (DRIFT EVEN DEEPER INTO HYPNOSIS AND) become even more relaxed. Relax all the muscles in your body. Let each muscle become heavier and heavier...any tightness will fade away as your breathing becomes quiet and slow. You are relaxing deeper, and deeper.

Prepare yourself to imagine the scene now. Allow the (HYPNOTIC SUGGESTIONS) instructions to enable you to create the imagined scene in your mind...doing exactly what you would do in the real situation. When I finish the description, keep imagining the scene until I tell you to stop. Let's do the scene now.

Response-Based Imagery Scene #2 (90 seconds)

You are sitting at home in a comfortable chair. You are sitting near a sunny window. It feels so good to be sitting with nothing to do, no need to rush anywhere.... You are feeling so relaxed. You feel a bit warm and perhaps a bit drowsy. Your body sinks back into the chair and you feel as though the chair enfolds you. Your head falls back comfortably against the head rest. You notice that all the muscles in your neck, head, and face feel quite relaxed. Your shoulders, back, legs...no tension at all. They feel quite limp and relaxed. Concentrate on this feeling...so warm, drowsy, relaxed. Your breathing is quiet and deep. Your mind is completely calm as well. Calm...quiet...relaxed. (Brief pause)

Now stop imagining the scene. Please open your eyes (BUT REMAIN HYPNOTIZED). Try to not disrupt your general feeling of relaxation. We now want you to report on how vividly you experienced this scene. Please pick up the questionnaire and pen that are at your side. Rate your experience according to the few questions in front of you. Do not talk during this segment.

3rd Treatment Efficacy Rating (Appendix B)

Please close your eyes once more. Let your body assume a comfortable position. Allow yourself to (DRIFT EVEN DEEPER INTO HYPNOSIS AND) become even more relaxed. Relax all the muscles in your body. Let each muscle become heavier and heavier...any tightness will fade away as your breathing becomes quiet and slow. You are relaxing deeper, and deeper.

Please prepare yourself to imagine the scene now. Allow the (HYPNOTIC SUGGESTIONS) instructions to enable you to create the imagined scene in your mind...doing exactly what you would do in the real situation. When I finish the description, keep imagining the scene until I tell you to stop. Let's do the scene now.

Repeat Response-Based Imagery Scene #2

Now stop imagining the scene. Please open your eyes (BUT REMAIN HYPNOTIZED). Try to not disrupt your general feeling of relaxation. We now want you to report on how vividly you experienced this scene. Please pick up the questionnaire and pen that are at your side. Rate

your experience according to the few questions in front of you. Do not talk during this segment.

4th Treatment Efficacy Rating (Appendix B)

Please close your eyes once more. Let your body assume a comfortable position. Allow yourself to (DRIFT EVEN DEEPER INTO HYPNOSIS AND) become even more relaxed. Relax all the muscles in your body. Let each muscle become heavier and heavier...any tightness will fade away as your breathing becomes quiet and slow. You are relaxing deeper, and deeper.

Now your task is to (HYPNOTICALLY) imagine a scene of your own choice. The only stipulation is that your scene should be similar in design to the previous scenes. That is, it should help you to actually become relaxed. Most of us have experienced certain moments in which we felt especially calm, relaxed, or peaceful...such as lying on a special couch while reading a favorite book; or strolling down a country road on a pleasant day; or enjoying the solitude of sitting in front of a warm fireplace on a cool evening. You can either imagine a scene that you have previously experienced, or you can imagine an entire new scene that would help you to feel relaxed. You also may choose to return to one of the scenes that you imagined earlier in this session. Remember, let yourself feel your body actually relaxing during this imagined scene. Enjoy how your breathing will become slow, deep, and easy...and how any tightness in your muscles will drain out of them throughout the exercise. Close your eyes, and begin imagining your selected scene now.

Selected Scene Visualization (90 seconds)

Now stop imagining the scene. Please open your eyes (BUT REMAIN HYPNOTIZED). Try to not disrupt your general feeling of (HYPNOTIC) relaxation. We now want you to report on how vividly you experienced this scene. Please pick up the questionnaire and pen that are at your side. Rate your experience according to the few questions in front of you. Do not talk during this segment.

5th Treatment Efficacy Rating (Appendix B)

Please close your eyes once more. Let your body assume a comfortable position. Allow yourself to (DRIFT EVEN DEEPER INTO HYPNOSIS AND) become even more relaxed. Relax all the muscles in your body. Let each muscle become heavier and heavier...any tightness will fade away as your breathing becomes quiet and slow. You are relaxing deeper, and deeper.

Now your task is to (HYPNOTICALLY) imagine your selected scene once more. Remember, your scene should be similar in design to the previous scenes. That is, it should help you to actually become relaxed. Let yourself feel your body actually relaxing during this imagined scene. Enjoy how your breathing will become slow, deep, and easy...and how any tightness in your muscles will drain out of them throughout the exercise. Close your eyes, and begin imagining your selected scene now.

Repeat Selected Scene (90 seconds)

Now stop imagining the scene. Please open your eyes (BUT REMAIN HYPNOTIZED). Try to not disrupt your general feeling of relaxation. We now want you to report on how vividly you experienced this scene. Please pick up the questionnaire and pen that are at your side. Rate your experience according to the few questions in front of you. Do not talk during this segment.

Post-treatment Physiologic Assessment (SBP, DBP, HF)

Please close your eyes once more. Now I am going to help you to return to your normal state of alertness. As you follow my instructions you will gradually become more and more alert.

YOU'RE GOING TO WAKE UP IN A FEW MINUTES. YOU WILL FEEL REFRESHED, WIDE AWAKE, AND IN A GOOD MOOD. I WILL COUNT FROM ONE TO TEN, AND WITH EACH COUNT YOU WILL FEEL YOURSELF BECOMING MORE AND MORE AWAKE. READY? NOW; 1...BEGINNING TO AWAKE...2...3...MORE AND MORE AWAKE...4...5...STILL MORE AWAKE...6...7...GRADUALLY BECOMING AWARE OF THE SOUNDS IN THE ROOM, AND THE PRESENCE OF OTHERS...8...ALMOST COMPLETELY AWAKE...9...10...WIDE AWAKE AND REFRESHED...OPEN YOUR EYES...WIDE AWAKE. PLEASE REMAIN WHERE YOU ARE FOR THE MOMENT, AND SIT BACK WITH EYES OPEN. (BRIEF PAUSE)

(IMAGERY GROUP ONLY) Begin to notice how refreshed you generally feel. Take note of your relaxed muscles, your rate of breathing, and the relaxed flow of your thoughts. Gradually become aware of the sounds in this room, as well as the presence of others around you. Slowly begin to move your muscles. You may wish to stretch the muscles in your arms or legs. Let your eyes open gradually. Please remain where you are for the moment, and sit back with your eyes open.

Today I asked you to practice achieving vivid images by becoming (HYPNOTICALLY) involved in each scene--that is, by actually imagining how your muscles feel, your breathing, etc. We will continue with the same kind of practice next session.

It's important use these imagery/HYPNOTIC skills when you have a headache, or when you feel a headache coming on. No matter what you are doing or thinking about just before or during a headache, take a few minutes out and imagine one of these scenes. Get yourself into a comfortable position, in a quiet place if possible, and imagine a scene or two. IT WILL BE POSSIBLE FOR YOU TO UTILIZE SELF-HYPNOSIS BY SIMPLY ALLOWING YOURSELF TO BECOME RELAXED AND IMAGINATIVELY FOCUSED--JUST AS YOU DID IN THIS SESSION. Remember what you've learned about vivid imagery. You must really put your own responses and feelings into the imagery. Practice it for 5 or 10 minutes every day. You may find that it takes you a while to get into it, but that's okay. Just keep going. Some people find it more difficult to do than others. It just takes practice. You'll get better and better at it the more you do it. Try to practice the imagery/SELF HYPNOSIS technique when you have time, in order to become more efficient at using it.

Placebo Group

We are going to teach you strategies that you can use to reduce the

frequency and intensity of your headaches. Previous research has made it clear that stress is somehow related to headaches, and often a person's pain experience is increased by automatically engaging in a set of physical, emotional, and cognitive reactions to the headache. For example, when some people experience a headache coming on they might think to themselves such things as: "Oh God this is awful...I can't take this anymore...its never going to get better...." Thoughts like this actually increase the impact of the headache. These thoughts are commonly associated with feelings of being overly burdened with, or emotionally defeated by the headache. A cluster of physical symptoms also accompanies this reaction style. The symptoms are expressed in the form of tension in the skeletal muscles, increased heart rate, blood pressure, rate of breathing, and so on. This automatic reaction pattern can interfere with an individual's ability to carry out regular daily tasks. The result is that daily activities are experienced as being stressful, and this experience can further intensify headache activity. As you might expect, automatic reactions to headaches can take on many forms. However, numerous scientific studies have demonstrated that physical, emotional, and cognitive reactions to headaches can be significantly altered through therapies such as the one that you are about to learn.

"Subconscious reconditioning" will be part of this treatment. This technique has been used by physicians and psychologists to help people with a variety of pain syndromes, like back pain, arthritis, as well as headache. When a person has been exposed to regular headache activity for a long period of time, reactions like those described earlier become so automatic that the individual no longer pays attention to this activity. Over time, it becomes extremely difficult to gain an awareness that these reactions are happening at all. Psychologists call these reactions "subconsciously controlled reactions". The treatment that you will be receiving will recondition the stress-related subconscious thoughts and feelings that are at the root to many of your headache episodes. We will present slides to you with embedded (or hidden) therapeutic messages. These messages are designed to enable you to subconsciously re-learn your automatic headache reactions. This technique is called "subliminal re-learning". The messages have been therapeutically designed so that the subconscious thoughts and feelings, related to your headaches, can be re-learned.

During the subliminal re-learning treatment, it is important that you do not attempt to think about what the specific message might be. Such thinking will interfere with the regular procedure of allowing the therapeutic message to penetrate your subconscious mind. Normally when people are given a message, they analyse it critically. It is important to not engage in critically thinking about the subliminal, therapeutic message. You will best enable these messages to have their therapeutic effect by simply allowing yourself to uncritically view the slides.

Baseline Physiologic Assessment (SBP, DBP, HR)

To engage in subliminal re-learning to the fullest extent, please listen to the following set of instructions. Remember, you don't have to be concerned with deliberately trying to make this treatment work effectively. Its effectiveness only depends on your ability to

uncritically view the incoming therapeutic information.

With each subliminal message, you will hear the cue syllable, "CUV". You may notice a slight tingling sensation in your neck, shoulders, or forehead and face. This is a normal response in subliminal re-learning for the message set, and you need not be concerned about it.

Please find a position that is comfortable for you. Okay, now I want you to adopt as open and uncritical an attitude as possible. Your openness will allow the scenes to penetrate the barriers that are normally set up, so that the subliminal re-learning process can occur. Check that you are comfortably seated. Allow yourself to sit back and let your attention focus on the following messages. The subliminal information will be present now.

1st Slide Presentation

(ASSESSMENT INSTRUCTIONS) Do not disrupt your attentional focus and the openness of your attitude. We want you to report on how intense the cue syllable is in your memory. You will also be asked to indicate the degree to which you feel various sensations in your muscles. The experimenter will hand you a questionnaire and pen. Please rate your experience according to the few questions in front of you. Do not talk during this segment.

1st Treatment Efficacy Rating (Appendix B)

(SLIDE PREPARATION INSTRUCTIONS) Let your body assume a comfortable position. Allow yourself to focus even more uncritically on the next series of slides. Sit back, and permit the subliminal messages to penetrate deeper and deeper into your subconscious. Cue syllables will once again be presented as conscious markers for each subliminal message set. The subliminal information will be presented now.

2nd Slide Presentation

Do not disrupt your attentional focus and the openness of your attitude. We want you to report on how intense the cue syllable is in your memory. You will also be asked to indicate the degree to which you feel various sensations in your muscles. The experimenter will hand you a questionnaire and pen. Please rate your experience according to the few questions in front of you. Do not talk during this segment.

2nd Treatment Efficacy Rating (Appendix B)

REPEAT THE CYCLE OF SLIDE PREPARATION AND ASSESSMENT INSTRUCTIONS UNTIL COMPLETION OF THE 6TH SLIDE PRESENTATION AND ASSESSMENT

Post-Session Physiologic Assessment (SEP, DEP, HR)

Sit back once more. Begin to notice how refreshed you generally feel. Take note of the sensations in your muscles, and gradually become more aware of your rate of breathing and the flow of your conscious thoughts. You will notice the sounds in this room, as well as the

presence of others around you. Slowly begin to move your muscles. You may wish to stretch the muscles in your arms or legs. Remain where you are for the moment, and sit back with your eyes open. Please do not talk. (Brief pause)

It is important to try using the cue syllable ("CUV") when you have a headache, or when you feel a headache coming on. No matter where you are, intensely recall the cue syllable. Do not attempt to consciously uncover any associations in your subconscious. Rather, once you have recalled the cue syllable, just go about your regular activities. The subliminal effect will follow naturally.

Debriefing Summary

The purpose of this experiment was to determine the efficacy of response-based imagery training for the reduction of headache activity. This cognitive coping technique was taught in the form of hypnotic suggestions and imaginative activity. For participation in this experiment it was necessary to assess each person's average headache activity prior to, during, and following the experiment.

The hypnosis and imagery procedures used in this experiment enabled individuals to acquire a pain management skill that can facilitate self-control over headaches. This treatment was designed to induce a sense of relaxation and control over the pattern of headache recurrence. For this reason, it was necessary to compare levels of average headache activity by means of an experimental condition that controlled for various dimensions of cognitive pain control. One experimental group in Experiment 1 ("Subliminal Re-learning") was in fact only encouraged to feel more in control over their headaches. This was the placebo condition. Another group, in Experiment 1 and 2, was only asked to monitor their headache activity throughout the time that the experiment was conducted. The monitoring condition enabled us to determine if individuals would have undergone changes in headache activity for reasons that we did not anticipate and control for in this experiment.

Thank you for participating in this study. The data that we have collected, as a result of your cooperation, will be compared to investigative findings from other research laboratories in Canada, the United States, and Europe.

Appendix Q

Correlation Matrix for Hypnotic Susceptibility and Personality Traits

	CURSS-O	CURSS-S1	CURSS-OI	VMI	PILL
CURSS-O					
CURSS-S1	^{**} .7631 (N=48)				
CURSS-OI	^{**} .8016 (N=48)	^{**} .7831 (N=48)			
VMI	[*] .3252 (N=47)	.2475 (N=47)	.2739 (N=47)		
PILL	.0055 (N=47)	.0627 (N=47)	.0129 (N=47)	.0691 (N=49)	
MCSDS	-.1748 (N=46)	-.0204 (N=46)	.0287 (N=46)	-.1204 (N=49)	-.1034 (N=48)

Superscript ^{**} signifies $p < .05$

Superscript ^{***} signifies $p < .01$

Correlation Matrix for Variables Utilized in Hierarchical Regression Analysis: N=51

	IMI-Q-S	IMI-Q-PR	IMI-Q-CN	IMI-Q-CH	FIMI-Q-S	FIMI-Q-PR	FIMI-Q-CN	FIMI-Q-CH	SLF-EST	TR-ANX	PSS
IMI-Q-S											
IMI-Q-PR	-.1044										
IMI-Q-CN	-.6269**	.1500									
IMI-Q-CH	.0270	.4714**	.0168								
F-IMI-Q-S	.7207**	.0553	-.3788**	.2317							
F-IMI-Q-PR	-.1277	.6680**	.0050	.3695**	.0044						
F-IMI-Q-CN	-.4740**	.0804	.6280**	.0370	-.4782**	.2246					
F-IMI-Q-CH	-.0548	.2768*	.0516	.5394**	.0751	.4870**	.1274				
SLF-EST	.1508	-.3580**	-.0559	-.1215	.1938	-.2436	.1129	-.0375			
TR-ANX	-.0153	.4295**	-.1119	.3970**	-.0834	.3065	-.1910	.1891	-.7506**		
PSS	-.0341	.2872*	-.0919	.3163*	-.0710	.1901	-.1303	.1543	-.6820**	.7257**	
HA-REDUCED	-.0294	-.0938	-.0828	.1183	-.1339	.1148	.1251	.3057*	.0858	-.0305	.3117*

* - $p < .05$
 ** - $p < .01$

Appendix R (cont'd)

Abbreviation Schema for Variables in Correlation Matrix:

- IMIQ-S - Baseline administration of IMIQ, Seriousness subscale
 (Turk et al., 1986)
- IMIQ-PR - Baseline administration of IMIQ, Personal
 Responsibility subscale (Turk et al., 1986)
- IMIQ-CN - Baseline administration of IMIQ, Controllability
 subscale (Turk et al., 1986)
- IMIQ-CH - Baseline administration of IMIQ, Changeability
 subscale (Turk et al., 1986)
- FIMIQ-S - 8-Week Follow-up administration of IMIQ, Seriousness
 subscale (Turk et al., 1986)
- FIMIQ-PR - 8-Week Follow-up administration of IMIQ, Personal
 Responsibility subscale (Turk et al., 1986)
- FIMIQ-CN - 8-Week Follow-up administration of IMIQ,
 Controllability subscale (Turk et al., 1986)
- FIMIQ-CH - 8-Week Follow-up administration of IMIQ,
 Changeability subscale (Turk et al., 1986)
- SLF-EST - Baseline administration of Self-esteem (Rosenberg, 1979)
- TR-ANX - Baseline administration of STAI-Trait subscale,
 (Spielberger et al., 1970)
- PSS - Baseline administration of Perceived Stress Scale
 (Cohen et al., 1983)
- HA-%REDUCED - Mean percentage of reduction in headache activity
 (headache index) across Trials (Blanchard & Andrasik,
 1985)

END

1|6·0|2·90

FIN

Never or almost never experience symptom	Less than 3 or 4 times per year	Every month or so	Every Week or so	More than once every week
(A)	(B)	(C)	(D)	(E)
39. Strong reactions to insect bites			() () () () ()	
40. Headaches			() () () () ()	
41. Sensation of pressure in head			() () () () ()	
42. Hot flashes			() () () () ()	
43. Chills			() () () () ()	
44. Dizziness			() () () () ()	
45. Feel faint			() () () () ()	
46. Numbness or tingling in any part of body			() () () () ()	
47. Twitching of eyelid			() () () () ()	
48. Twitching other than eyelid			() () () () ()	
49. Hands tremble or shake			() () () () ()	
50. Stiff joints			() () () () ()	
51. Sore muscles			() () () () ()	
52. Sore throat			() () () () ()	
53. Sunburn			() () () () ()	
54. Nausea			() () () () ()	