COGNITIVE DECLINE IN CHRONIC PAIN PATIENTS: A NEUROPSYCHOLOGICAL EVALUATION

DISSERTATION

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Leesa C. Nite, B.A.
Denton, Texas

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The purpose of the present study was to investigate cognitive functioning in a group of 30 chronic pain patients (CPG) as compared to a group of 39 acute pain patients (APG). In order to assess cognitive performance, certain subtests were selected from the McCarron-Dial System (MDS) of Neuropsychological Evaluation. Specifically, a measure of haptic discrimination was used along with the Bender Visual Motor Gestalt Test. As such, completion of these subtests required a cortically mediated, central nervous system processing of sensory information. This particular method of assessment was chosen because it provided a nonverbal measure of higher-order cognitive performance. Additionally, the haptic measure provided separate scores for right and left hemispheric functioning. Data analysis revealed significantly poorer Bender performance among CPG members ($t(69) = -5.09$, $p = .0004$, two tailed). Further data analysis revealed that the CPG performed significantly poorer on certain of the haptic discrimination subtests. Specifically, both texture and configuration scores for the

right hemisphere were significantly lower among CPG members (texture, $p = .042$ and configuration, $p = .002$). Subsequent analyses were conducted to determine predictive relationships between important variables. These data are discussed in terms of their clinical significance and importance for future research.

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CHAPTER I

INTRODUCTION

Pain resulting from acute disease or traumatic injury is termed acute pain. Typically, acute pain diminishes as healing progresses, and eventually, once healing is complete, the associated pain remits. In some cases, however, pain persists beyond the expected healing time of the original injury or continues due to some ongoing degenerative process. This type of pain is termed chronic pain. For purposes of research, chronic pain has typically been defined as any pain lasting six months or more (Sternbach, 1974).

In the recent past, the medical and legal communities have focused increased attention on the clinical aspects of chronic pain. This focus was in response to the extreme cost impacting the nation's economy, both in terms of dollars and in loss of skilled labor. In a 1976 report, it was noted that approximately 14 billion dollars were spent that year in treatment of low back pain and compensation for lost labor alone (Akeson & Murphy, 1977). Moreover, it is estimated that there is an additional expense of one billion dollars accruing each year associated with chronic pain.

The logical response of the contemporary medical community toward the alleviation of pain typically consists of efforts to identify the organic cause and follow with treatment of the traumatized site. Unfortunately, when the pain has become chronic in nature, efforts of this kind commonly fail. Evidencing this are results from surgical outcome studies demonstrating that procedures such as laminectomy and spinal fusion often fail to render long-term relief (Akeson & Murphy, 1977; Bonica, 1974). In general, it is becoming increasingly evident that the typical chronic pain patient represents an inadequate understanding of the relationship between pain perception and the pain eliciting stimulus (Urban, 1982). Because traditional medicine has been repeatedly frustrated in its efforts to treat chronic pain, much recent research has been devoted to explicating the etiology of chronic pain (Fordyce & Steager, 1979).

An understanding of chronic pain is clouded by documentation that, in most cases, pain lingers beyond healing of the originally traumatized tissue (Beals & Hickman, 1972; Fordyce, Lensky, Calsyn, Shelton, Stolov, & Rock, 1984). Akeson et al. (1977) hypothesized that this results from inflammatory neural changes that potentiate chronic neuritis and neurofibrosis. While this theory has some heuristic appeal, there are inherent problems which limit its utility. First, these inflammatory processes

involve soft-tissue changes and, as such, elude visualization and documentation using traditional medical technology. Moreover, given the same presenting diagnosis, it is unclear why these inflammatory neural changes would develop in one individual and not in another. Because of the difficulty associated with the pathophysiologic objectivication of chronic pain, the question of a functional etiology is continually raised.

Research designed to uncover personality differences between chronic pain patients and organic pain patients has, overall, found no substantive distinctions (Lawlis & McCoy, 1983). When looking at the responses of chronic pain patients on standard measures of personality (e.g., MMPI), considerable heterogeneity is revealed (Lawlis & McCoy, 1983). Therefore, it is unlikely that a specific personality type is somehow predisposed to developing chronic pain. However, the stress associated with persistent pain certainly effects change in psychological functioning. One of these changes is addressed in research which documents a decline in cognitive functioning among chronic pain patients (Lefebrre, 1981; Smith, Aberger, Follick, & Ahern, 1986). These authors explain the observed decline in cognitive functioning as a by-product of depression. For this explanation they rely on previous research looking at the information processing

of depressed, nonchronic pain subjects (for discussion of this research, see Beck, 1967).

The research tradition begun by Beck (1967) views depression as the outcome of inaccurate information processing. As such, this perspective is exclusively psychological/cognitive in its consideration of etiology. Any neurobehavioral link between depression and cognitive performance is overlooked. Moreover, this author found no case where the neurobehavior of depression, chronic pain, and cognition was addressed. Speaking from a more general perspective, some authors presume an interaction between psychologic and neurologic factors which becomes increasingly complex as time passes (Sternbach, 1968; Swanson, 1984; Swerdlow, 1981). Viewing the chronic pain patient from this perspective, it becomes clear that any research inquiry must encompass the interaction between psychology and physiology in order to be maximally productive. This viewpoint opposes research having a unidimensional goal of uncovering a functional etiology as overly simplistic in its conceptualization of chronic pain, regardless of the research question. In the study of declining cognitive functioning, it is advisable to consider the interaction between psychologic and neurologic factors in hopes of more thoroughly addressing the issue.

Theories of Pain Perception

To begin an investigation of the neuropsychology of chronic pain, an understanding of pain sensation, or more accurately, pain perception is warranted. Classical medical theories of pain characterize it as a primary sensation referred to as nociception involving a relatively simple physiologic system (Crue, Kenton, Carregal, 1978). This conception of pain has been repeatedly challenged by recent studies documenting that the experience of pain is a kind of summary event arising from a complex interaction between several components. Included in this interactional model are pathophysiologic changes in surrounding tissue, sensory perception (primarily mediated by spinal pathways and thalamic structures) and cognitive emotional sequelae (primarily mediated by cortical and limbic structures linked to thalamic structures via ascending and descending neural tracts). Development of this interactional model reflects a revolutionary change in the classical medical conception of pain perception in that substantial emphasis is placed on the cognitive and emotional aspects of pain.

Beecher, as early as 1956, emphasized the importance of the meaning patients attached to their pain. In his now classic study, Beecher compared the frequency of requests for narcotics by individuals suffering combat wounds with those of hospital patients having comparable surgical wounds. His results showed that only 25% of the soldiers

requested narcotic pain medication while 80% of the hospital patients requested narcotics. Beecher reasoned that a wounded soldier was often allowed to return home because of his injury while hospital inpatients were frequently separated from loved ones and confined to a seemingly overwhelming and frightening place. Thus, Beecher accounted for the discrepant use of narcotics among the war wounded and surgical inpatients by emphasizing the different meaning pain had for each. More recently, Melzak and Casey (1968) have emphasized the motivational and emotional aspects of pain as synergistically related to the neurology and physiology of pain. These more contemporary models of pain perception are essentially describing it as a neuropsychologic phenomenon. This applies whether or not the pain is acute or chronic. Most authors agree that cognitive and emotional factors become increasingly involved in pain perception as the duration of pain lengthens (Chapman & Bonica, 1985).

In a 1974 publication entitled Pain Patients: Traits and Treatment, C. P. Sternbach states that though the transition from acute to chronic pain is little understood, one thing seems clear; the character of the pain changes as it becomes increasingly chronic until it eventually bears little similarity to the pain of the initial injury. He goes on to add that in a significant number of chronic pain patients, both general physical health and psychological

health deteriorate as pain continues, often leaving the patient somatically preoccupied, depressed and irritable. Wittig (1982) adds to this list of symptoms the prevalence of sleep disturbance among chronic pain patients.

In a 1977 article, Sternbach outlines a distinction between acute and chronic pain modeled after Selye's formulation of the general adaptation syndrome (Selye, 1976). Sternbach notes that the onset of acute pain elicits an alarm reaction synonymous with discharge of the autonomic nervous system. In an effort to supply vital tissues with increased energy needs, the heart rate, blood pressure, and respiration increase. This is accompanied by the shunting of blood to vital organs, release of stored glycogen from the liver, pupil dilation, and diaphoresis (Guyton, 1976). If the sensation of pain continues past this initial stage of alarm, then the body begins to adapt, homeostatic conditions are restored, and the autonomic responses return to normal. However, in the case of a substantial passage of time where pain does not relent, there is corresponding taxing of homeostatic mechanisms leading to a state of adrenal exhaustion and general physical decline. Sternbach notes parallels between this state of exhaustion and the onset of clinical depression. This is in agreement with the repeated documentation of depression in chronic pain patients (Atkinson, Kremer, & Risch, 1986; Blumer & Heilbronn, 1982; Donlon & Bittle,

1970; Freeman, Calsyn, & Louks, 1976; Krishner, France, & Houpt, 1985; Maruta, Swanson, & Swenson, 1976; McCreary, Turner, & Dawson, 1974; Skevington, 1983; Schaffer, Donlon, & Bittle, 1980).

Other authors emphasize the prevalence of anxiety in chronic pain patients (Lawlis & McCoy, 1983; Swanson, 1984). In keeping with Sternbach's (1977) discussion of chronic pain, anxiety could be viewed as the psychological expression of the body's efforts to restore and maintain physiological homeostasis following the initial stage of alarm in Selye's (1976) model. As such, anxiety may represent a relatively early affective response to chronic pain which eventually gives way to depression once the individual's adaptive resources are exhausted. This is entirely speculative and its value is heuristic. In general, a neuropsychological understanding of chronic pain seems necessary to best address its affective and cognitive sequelae. Because other authors have noted a decline in cognitive performance among chronic pain patients as well as of prevalence of depression among chronic pain patients, the following section is devoted to a discussion of depression and related phenomena. Particular attention is paid to their manifestation in chronic pain patients.

Models of Depression and Anxiety in Chronic Pain Patients

At a general level, depression is conceptualized as either endogenous or nonendogenous in type. Specifically,

nonendogenous or reactive depression is considered to be a normal occurrence among all people. It usually involves a reaction toward some disappointing event in a person's life, such as loss of a job, break-up of a relationship, or death of a loved one. Though it may be quite severe in intensity, it usually resolves within a matter of weeks at the most. Endogenous depression is thought by most researchers to be the result of a disturbance in biochemistry with at least a partial genetic etiology (Rush, Weissenburger, Vinson, & Giles, 1983). When authors discuss the topic of depression, it is not always clear whether they are referring to a reactive depression or an endogenous depression. This probably stems from the fact that a phenomenological difference between the two is not always clear. It seems most logical to assume that there is probably always some interaction between psychology and physiology in any type of depression. Also, depending on a given author's background and training, they typically take one or two major perspectives in their discussion of the etiology and treatment of depression; specifically, either a cognitive or biologic perspective is used. However, a thorough understanding of the cognitive and biologic perspectives reveals that they are not mutually exclusive, despite the fact that they are rarely jointly addressed.

Cognitively based research models of depression tend to focus on information processing capacity and coping

styles of depressed subjects. A common finding is marked tendency among depressed subjects to make errors of logic owing to faulty information processing (Beck, 1967; Hammen, 1978; Krantz & Hammen, 1979). Aaron Beck (1967) attributes these cognitive errors to systematic misinterpretations of events using a pervasively negative set. For example, he has repeatedly found that depressed subjects consistently interpret their life experience in a negative way so that both their sense of esteem and personal efficacy suffer. Much debate has pivoted on the question of which comes first, the distortions or the depression; however, there is no clear answer to this question to date. Whatever the case, cognitively based researchers have documented a common tendency in depressed to focus on the negative aspects of their experiences with an intensity that leads to a distortion of reality.

More biologically oriented researchers explain the faulty information processing of depressed subjects as arising from the motivational and attentional deficits which accompany endogenous depression. According to this model, depressed individuals frequently overlook essential details, leaving them prone to making both errors of logic and memory encoding. This explanation of faulty information processing differs from that of Beck (1967) in a lack of emphasis on the subject's thought content. Unlike purely cognitive models of depression, this model

does not attribute the prevalence of cognitive distortion to a negative interpretive set. Rather, errors in information processing are seen as one competent in a syndrome of generalized apathy and physical lethargy. This model of depression is much more biologic in tone and is therefore consistent with Sternbach's description of depression in chronic pain patients as an effect of unrelenting stress (Sternbach, 1977).

Recall that Sternbach describes depression as paralleling the onset of the last stage in Selye's general adaptation syndrome termed exhaustion (Selye, 1976). Akiskal and McKinney (1975) aptly describe this position by defining depression as a psychobiologic final common pathway with multiple interacting determinants. In addition to the attentional deficits often found in depressed subjects, appetite and sleep are typically disturbed (Akiskal & McKinney, 1975). Clearly, depression represents a generalized state of psychophysiological dysfunction.

Biologically based research models of depression focus primarily on one of two major areas. The most extensively researched of these two areas deals with the relationship between the biogenic amines norepinephrine and serotonin, cortisol secretion, and endogenous opioid peptides commonly referred to as endorphins. The second less extensively researched area deals with altered hemispheric

lateralization in depressed subjects. An overview of each of these two areas follows with particular attention paid to the relevance of each as they relate to the study of chronic pain.

When the body is stressed, one of its many responses is an increased secretion of cortisol, an adrenal steroid hormone similar to the pharmacologic agent hydrocortisone. Cortisol has stress buffering effects which are mediated through altered metabolic processes and anti-inflammatory mechanisms. The secretion of cortisol is initiated by a discharge of hypothalamic corticotropin releasing factor (CRF) by the hypothalamus. This results in relay of a message from the pituitary gland to the adrenal cortex which then releases cortisol into the bloodstream of the stressed individual. In addition to influencing the increase of serum cortisol, CRF secretion also results in increased levels of serum endorphins (Guillemin, Vargo, Rossier, Minick, Ling, Rivier, Vale, & Bloom, 1977). These substances have an opiate-like effect on the body inhibiting the perception of pain and elevating mood (Mueller & Genazzanni, 1984).

Previous research has documented unusually high levels of cortisol in depressed subjects (Kaplan & Sadock, 1985). Under normal conditions, noradrenergic mechanisms inhibit CRF release. This restraint on the hypothalamic discharge of CRF is accomplished via a negative feedback loop based

on serum cortisol levels. This inhibitory mechanism services the body's need for homeostatic regulation. Apparently, there is a disturbance in noradrenergically mediated neurotransmission in depressed subjects resulting in a disinhibited release of CRF and consequent high levels of serum cortisol.

Information Processing and Depression in Chronic Pain Patients

Much research has been devoted to the information processing capacity of depressed chronic pain patients. This research has documented that just as depressed subjects without chronic pain are prone to making cognitive errors, so are depressed chronic pain patients (Lefebrre, 1981; Smith, Aberger, Follick, & Ahern, 1986). In their discussion of depression in chronic pain patients, these authors drive an explanation of the observed faulty information processing from a cognitive model of depression. While they do not deny the physical substrates of cognition, they simply do not address them. Therefore, these authors focus solely on the cognitive corollaries and do not attempt to elucidate the neuropsychology of chronic pain.

Conversely, other authors have looked exclusively at the biology of depression in chronic pain patients. In a 1986 study, Atkinson and others compared serum cortisol levels in a group of 36 chronic pain patients, 24 of whom

met diagnostic criteria for depression, to serum cortisol levels in a group of 28 depressed pain-free subjects (Atkinson, Kremer, Risch, & Dana, 1986). Both the depressed chronic pain group and the depressed pain-free group showed significant elevations of serum cortisol when compared to the nondepressed chronic pain group. This finding strongly suggests the presence of disturbed serum cortisol regulation in chronic pain patients arising not from the pain itself but from parallel disturbances in affect. This calls to mind Sternbach's (1977) discussion of depression in chronic pain patients as related to the stress-induced adrenal dysfunction described first by Selye (176). There are many psychological variables such as financial status, level of education and degree of family support which collectively intervene in the chronic pain patient's course. Moreover, these psychosocial variables interact with variables of genetic endowment, general physical health, age and gender in creating a combination of stressors and resources for dealing with stress. The effect of these multiple interactions is, by definition, highly individualized. Therefore, it seems illogical to assume that all chronic pain patients are equally likely to become depressed; though it is clear than many, perhaps most, eventually do.

Moreover, because there is an absence of research documenting a clear relationship between cognitive

performance and the neurobiology of depression, this link remains a speculative one. Still, an understanding of the human body's response to stress coupled with an understanding of the neurobiology of depression points to such a link.

Because the body's release of endorphin is controlled by the same mechanisms regulating the release of cortisol, it is not surprising to find documented cases of simultaneous elevations of both cortisol and endorphin. In a 1983 study of 24 chronic pain patients, serum cortisol levels and serum endorphin levels were both abnormally elevated (Atkinson, Kremer, Risch, & Bloom, 1983). Recalling previously cited research correlating depression with faulty information processing, it is interesting to discover a documented correlation between sensory information processing deficits and abnormal endorphin regulation (Almay, Johanssen, Von Knorring, 1978). It is important to note that the information processing typically measured in depressed subjects is of a verbal, analytic type (Beck, 1967). These abilities are believed to be primarily mediated by the left hemisphere of the cerebral cortex (D'Elia & Perris, 1973). Conversely, sensory information processing is largely a nonverbal task. Therefore, a direct comparison between the two is problematic, especially as hemispheric lateralization may be involved.

There is a growing number of researchers investigating the presence of right hemispheric dysfunction in depressed subjects (Davidson & Fox, 1982; Davison, Schwartz, & Saron, 1978; D'Elia & Perris, 1973; Swartzburg, 1983). Most of these studies have relied on analyses of EEG data showing alpha dysynchrony in the right hemispheres of depressed subjects. Other researchers have looked at subjects with damage to the right hemisphere. These researchers report that these patients often show pronounced disturbances in affective modulation (Flor-Henry, 1969a, 1969b; Goldstein, 1962; Hacaen, 1962). The research literature review revealed only one study using neuropsychological assessment methods to explore the effects of depression (Newman & Sweet, 1986). In this study, global deficits were observed on verbal analytic subtests requiring focused concentration and attention. No lateralized findings were reported. This seems to contradict research documenting right hemispheric dysfunction. However, the fact that the neuropsychological testing involved a heavy verbal component may have masked the presence of right hemispheric dysfunction.

More importantly, the literature review revealed no studies using standardized neuropsychological assessment of chronic pain patients. This despite the fact that the instrumentation needed to measure neuropsychological functioning is readily available. Most neuropsychologic assessment, even when it proposes to measure primarily

sensory information processing, heavily taps the subjects' capacity for centrally mediated, higher order cognitive processing (see later discussion of these devices). This would seem to underscore its utility in investigating the observed decline in cognitive skills among chronic pain patients. To some degree, testing with instruments such as the WAIS-R could provide this information. However, such instruments rely heavily on both receptive and expressive verbal skills, as well as providing very little information regarding hemispheric lateralization. The present research circumvented both of these problems by using a standardized neuropsychologic measure of sensory information processing.

Research Problem

As noted previously, other investigations of cognitive functioning in chronic pain patients have framed it in terms of inaccurate information processing associated with an underlying depression. While this approach has provided some useful insights it sheds little light on the underlying neuropsychology of cognition and depression. The present research represented an effort to explicate the neuropsychological sequelae of chronic pain especially as they impact cognitive functioning. It is reasonable to hypothesize that these changes are associated with an underlying depression; however, the neuropsychological perspective on information processing was chosen by the present author as the primary focus for exploration. As

stated previously, other researchers have already investigated the psychological link between depression and information processing. This author could not find a single study investigating the neuropsychology of chronic pain patients as related to their information processing capacity.

The present research attempted to address the neuropsychological nature of chronic pain through a comparison of standardized neuropsychological measures taken from an acute pain group (APG) and a chronic pain group (CPG). The specific measures were defined by their originators as measures of sensory information processing (McCarron & Dial, 1979).

It is important to remember that these instruments tap more than peripherally mediated sensory processing. Rather, they tap the subjects' ability to analyze and synthesize complex sensory input--a task which requires centrally mediated, higher-order cortical processing of sensory input. The advantage of these sensory neuropsychological measures is that they provide information regarding hemispheric lateralization, but do not require extensive verbal processing to complete. An overview of neuropsychologic assessment follows.

Models of Neuropsychological Assessment

Two major models of neuropsychological functioning have become predominant. The more clinical of the two was

developed by A. R. Luria (1966, 1970, 1973). There was no attempt on Luria's part to quantify or standardize his assessment techniques. As a consequence, Luria's model is more clinical than psychometric in design. It has been labeled "Functional Systems Theory" because it emphasizes a complex, dynamic cooperation between various regions of the brain that are subsumed into functional systems. Different regions of the cerebral cortex are thought to be responsible for certain information processing activities with communication between these regions being evidenced in complex behavioral integrations. As such, this represents a meshing of a strict localization approach and a mass action approach to the study of brain behavior relationships. Moreover, this is why this procedure is said to assess higher-order cognitive functioning despite its use of sensory information as the primary stimulus to which the subject responds. As such, this procedure does more than simply assess peripheral neurologic functioning as done in a standard neuro-exam with procedures such as soft-tough, pin-prick, and two-point discrimination.

The second of the two major models was developed by Halstead and Reitan (Halstead, Reitan, & Davison, 1974). The theoretical underpinnings of this model are not substantially different from Luria's model. However, it does radically differ in its psychometric properties. Specifically, the Halstead-Reitan neuropsychologic battery

is a combination of several standardized subtests all with documented reliability and validity.

The McCarron-Dial System (MDS)

L. McCarron and J. G. Dial (1986) have developed a battery of standardized neurometric and behavioral measures patterned after the traditional views of brain function discussed above. The MDS is a state of the art evaluation system which uses a computerized format of data analysis. Five neuropsychological factors are assessed: a verbal-cognitive factor, a sensory factor, a motor factor, an emotional factor and an integration coping factor. Data for all five factors have been collected on both normal subjects and neuropsychologically impaired subjects. Resultant statistical norms are used as a descriptive information base from which individual comparisons can be made.

The present study investigates data from only one of the five factors, namely the sensory factor. The sensory factor is assessed using a combination of the Bender Visual Motor Gestalt Test (BVMGT) (Bender, 1938) and the Haptic Visual Discrimination Test (HVDT) (McCarron & Dial, 1979). A more detailed description of these two instruments is included in the methodology section of this paper.

The sensory factor of the MDS assesses brain-behavior relationships arising from associations between temporal,

parietal and occipital regions of the brain. These various regions, while representing differentiated structures of the cerebral cortex, form a dynamic, interacting neurological system. Since a bimodal sensorimotor integration of visual and tactile stimuli involves a complex array of cortical mechanisms, a generalized disturbance in the dynamics of the central nervous system--such as that presumed to underlie endogenous depression--should be detected. Additionally, as the HVDT renders scores for both right and left hemispheric parietal-occipital functioning, any lateralized sensory deficits should be detected.

A good performance on the BVMGT requires a more generalized capacity to focus and sustain attention. As a neuropsychological measure, it is capable of identifying organic pathology only when it is frank. Such pathology is expressed in gross motor and fine motor deficits. However, even when these motor systems are intact, disturbances in attentional capacity are readily revealed when the Bender is scored using a conservative system such as the Koppitz system (Koppitz, 1975).

Rationale and Statement of Hypotheses to be Tested

The present study differs importantly from previous research looking at cognitive decline associated with chronic pain in its use of nonverbal tasks (BVMGT and HVDT) to assess information processing capacity. The

present study's use of nonverbal assessment techniques is designed to aid in clarifying the specific nature of the loss in cognitive abilities seen in chronic pain patients. Traditionally, such cognitive deficits have been assessed exclusively through verbal tasks and researchers have explained their findings through a reliance on Beck's theories of depression (Beck, 1967). Because the nonverbally executed, visual-spatial tasks used in the present study require no verbally reported interpretation of social context, these tasks are insensitive to the kind of errors in cognitive processing Beck describes. Nevertheless, performance on both the BVMGT and HVDT can be expected to be adversely affected by anxiety and/or depression interfering with attentional capacity. It should be noted that the present study included no measure of depression. Therefore, while it is reasonable to consider a neuropsychologic relationship between depression and cognitive performance the present study provides no direct evidence of this relationship. Rather, data from the present study provides a heuristic and supportive base upon which to build future research regarding the neuropsychology of depression and cognitive functioning in chronic pain patients.

In the present study it was hypothesized that when compared to the acute pain group, members of the chronic pain group would do more poorly on both the Bender and the

HVDT. This seemed a reasonable prediction given previous documentation in chronic pain patients (Atkinson et al., 1986; Freeman et al., 1976; Lefebrre, 1981; Smith et al., 1986).

Because the HVDT assesses separate left and right hemispheric functioning, it provides more precise data regarding neurologic functioning than that which can be gleaned from BVMGT data alone. Should lateralized deficits emerge on the HVDT a neurologic basis for losses in information processing capacity is implied. That is, lateralized findings could not be explained solely on the basis of global attentional deficits; rather, presence of more circumscribed neurologic dysfunction is suggested. Such findings would not be surprising given the literature documenting disturbances in right hemispheric functioning among depressed subjects (Davidson et al., 1978; D'Elia & Perris, 1973; Swartzburg, 1983).

Because it was assumed that pain chronicity in some way leads to a decline in cognitive performance, it was hypothesized that BVMGT and HVDT scores would correlate with pain chronicity and that a predictive relationship between the two would emerge. Should a predictive relationship between scores on these neuropsychologic measures and duration of pain be found, particularly if lateralized changes demonstrate predictive power, alterations in neurochemistry can be assumed to underlie

the development of these changes. As previously noted, depression is a common corollary of chronic pain (Atkinson et al., 1986), and depression has been associated both with neuroendocrine and endorphin deregulation (Atkinson et al., 1983) as well as changed in hemispheric lateralization (Davidson et al., 1978). Because sleep patterns are intricately related to brain chemistry (Kaplan & Sadock, 1985) it was expected that the chronic pain group would report a relatively greater frequency of disturbed sleep, in addition to the predicted neuropsychological changes.

Hypotheses

To summarize, the following hypotheses were tested in the present study.

1. The chronic pain group would demonstrate poorer performance on the BVMGT when compared to the acute pain group at a level significantly above chance.

2. The chronic pain group would demonstrate poorer performance on the HVDT when compared to the acute pain group at a level significantly above chance.

3. The chronic pain group would demonstrate greater sleep disturbance when compared to the acute pain group at a level significantly above chance.

4. Negative correlations would emerge between research subject's BVMGT and HVDT scores and the length of time they had experienced pain, that is, the longer the

duration of pain the lower the BVMGT and HVDT scores would be.

5. A predictive relationship would emerge between sensory factor scores and chronicity of pain.

CHAPTER II

METHOD

Subjects

A group of 39 chronic pain patients was compared to a group of 30 acute pain patients on several measures. Subjects in the acute pain group (APG) had all gone to the private practice office of a board certified neurologist located in a large midwestern city. The names, addresses, and phone numbers of patients with an initial presentation of either lumbar or cervical pain were provided to the author of the present study. Following this, a form letter containing a statement of written consent was sent to each potential subject (see Appendix A). Individuals who returned the letter to their physician's office were contacted by phone and scheduled for the initial testing session. In the interim before testing, their charts were reviewed with answers to certain demographic questions recorded on a data sheet also detailed in Appendix A. This procedure resulted in a sample of 30 APG subjects, one of which had presented for treatment more than 3 months prior to testing.

The 39 subjects comprising the chronic pain group (CPG) had all experienced pain for a minimum of 6 months

prior to their evaluation. They were each evaluated by one of two registered Occupational Therapists employed in a large midwestern city. All 39 of the CPG members had been referred for evaluation to the private practice office of the two evaluating Occupational Therapists for forensic purposes. The two therapists who tested the CPG had completed formal instruction in MDS administration techniques. Once testing was completed, all of the resultant data was recorded in a patient file which the present author was permitted to review for purposes of extracting data. Demographic data was extracted including age, gender, type of complaint (lumbar or cervical), date of injury and date of testing. Additionally, HVDT and BVMGT data were collected as well as the patient's response to a question regarding the presence of a sleep disturbance.

Instruments

Two tests comprising the MDS sensory factor were used; these were the Bender Visual Motor Gestalt Test (BVMGT) and the Haptic Visual Discrimination Test (HVDT). A demographic questionnaire was also used to gather information regarding age, gender, type of complaint and duration of pain. This questionnaire included an item asking subjects whether or not they generally slept well. A dichotomous response of either yes or no was recorded.

Bender Visual Motor Gestalt Test (BVMGT). The BVMGT (Bender, 1938) is one of the most frequently used neuropsychological screening devices. It was designed to identify problems in perceptual motor integration. The BVMGT consists of a series of nine geometric designs which are reproduced by the subject using a simple paper and pencil format. The BVMGT was scored using the Koppitz scoring system (Koppitz, 1975). The Koppitz system identifies four types of errors: distortions of the figures; failure to integrate elements into a unified whole; rotation of the figure; and perseveration in drawing the figure or any of its parts. The number of errors can range from 0 to 30. In addition to being sensitive to frank brain dysfunction, the Koppitz scoring system is also sensitive to a poorly concentrated effort like that seen in anxious or depressed states.

For purposes of the present study, BVMGT scores were converted into T scores using conversion tables supplied by McCarron and Dial (1979). The mean of this T distribution was 70 with a standard deviation of 10.

Haptic Visual Discrimination Test (HVDT). The HVDT (McCarron & Dial, 1979) is a measure of tactile-visual discrimination. As such, it is primarily a measure of parietal-occipital brain function. Both right and left parietal-occipital functioning is assessed through a presentation of all test items to both the subject's right

and left hand. First, all items are presented to the right hand, followed by presentation to the left with at least a two hour interval in between each presentation. This method renders a total right HVDT score and a total left HVDT score. Because of the contralateral crossing of spinal tracts, HVDT scores from the right hand are indicative of left parietal-occipital function and vice versa.

During administration of the HVDT, objects of differing shape, size, texture and spatial configuration are sequentially presented to the subject's hand with the subject's vision of the object obscured by a cloth screen. The subject is asked to point with the other hand to one of five photographs which the subject believes visually matches the object being felt. A total of 48 objects are presented first to the right hand and then to the left hand. This method renders four separate scores for discrimination of shape, size, texture and configuration. These four scores are then summed to render a total HVDT score for both the right hand and the left hand.

The HVDT has an established reliability of .90 with a standard error of measurement of 2.17 (McCarron & Dial, 1979). Total raw scores can be converted into scaled scores using normative tables supplied in the HVDT manual (McCarron & Dial, 1979). For purposes of the present study, total raw scores for the right and left hands were

converted into T scores with a mean of 70 and a standard deviation of 10.

Procedure

Data for the CPG were collected from an archival data base with care taken to select only files of patients having experienced pain for 6 months or longer at the time of testing. The data were collected during two separate three-day visits to the office of the private practice occupational therapy group. This group of chronic pain patients was characterized by stress arising via their involvement in litigation. However, as litigation is not an uncommon corollary of chronic pain, this does not significantly limit the generalizability of the resultant findings. Prior permission was obtained from the attorneys of each patient by the occupational therapist in charge of the group.

Returned letters from potential APG members were forwarded to the present author by the neurologist treating these patients. Each patient was then contacted by phone to schedule the first of two testing sessions.

After scheduling an initial one and one-half hour testing session with each member of the APG, his or her file was reviewed for collection of demographic data. Each then presented for testing and was given the BVMGT to complete, followed by administration of the HVDT to the right hand. A second appointment was made to complete the

HVDT; this consisted of an administration to the left hand of each subject. In no case did the interval between the first and second session exceed one week.

Data analysis began with a conversion of APG HVDT data to T scores according to MDS instructions. Data for CPG had already been converted to T scores using the same formula. This T score conversion had the effect of controlling age related contaminants and facilitating a direct comparison of CPG and APG HVDT data.

CHAPTER III

RESULTS

To check for possible sources of contamination, frequency data for demographic variables were entered into analyses of central tendency and a series of chi-square analyses. The analysis revealed a mean age for the APG of 28.5 years (*SD* = 9.8) with a range of 19 to 56 years. The mean age for the CPG was 38.5 years (*SD* = 12.6) with a range of 18 to 83 years. This resulted in a significant age difference between the two groups, $t(69) = 2.54$, $p = .001$. However, this did not present problems in most of the subsequent data analysis as the *t* score conversions controlled for secondary variance arising as a function of age.

Gender frequency data were entered into chi-square analyses and no significant differences between the number of males and females in the two groups emerged. Of the 30 APG members, 16 were male and 14 were female. Of the 39 CPG members, 22 were male and 17 were female.

A chi-square analysis of the frequency of lumbar and cervical complaints in the two groups revealed no significant differences. In the APG, 13 subjects presented with initial complaints of lumbar pain and 17 subjects

presented initial complaints of cervical pain. In the CPG, 17 subjects presented with initial complaints of lumbar pain and 22 complained of cervical pain. As previously mentioned, all of the APG had experienced pain for three months or less as compared to the CPG members who had experienced pain for an average of 23.7 months (SD) with a range of 6 to 84 months in duration.

One additional chi-square analysis was performed on the frequency of yes and no responses to the question of whether or not the subject generally slept well. Results of this analysis indicated a significant difference between the two groups with subjects in the CPG reporting more sleep disturbance than subjects in the APG (phi = -.45, p = .0002). This chi-square analysis was used to test the study's third hypothesis stating that the CPG would report a greater frequency of sleep disturbance when compared to the APG.

Hypothesis one, stating that the CPG would perform more poorly on the BVMGT when compared to APG, was tested by a comparison between BVMGT group means. A significant difference between BVMGT group means did emerge, $t(69)$ = -5.09, p = .0004, two tailed, with the CPG demonstrating an overall poorer performance than the APG.

To test hypothesis two, which predicted a relative poorer HVDT performance among CPG members when compared to APG members, group data was entered into a two tailed t

test. No significant difference emerged for total HVDT $\underline{t}$ scores either for the right or left hand. However, significant differences did emerge between the two groups on left configuration scores, $\underline{t}(69) = -2.08$, $\underline{p} = .042$, and left texture scores, $\underline{t}(69) = -3.18$, $\underline{p} = .002$. Results of this analysis revealed that the CPG did significantly worse on the left hand configuration and texture subtests than did the APG.

To test hypothesis four, predicting a negative correlation between HVDT and BVMGT scores and pain duration, a set of correlations were performed using the CPG data only. Because the APG had, in each case, experienced pain for three months or less at the time of testing, data from this group provided little heterogeneity. For this reason, it was decided to delete APG data from this univariate analysis. Results of this analysis indicated that as the length of time a subject experienced pain increased, his or her performance on the HVDT generally tended to worsen. This trend was particularly strong for left HVDT scores. Table B-1 summarizes these results. BVMGT scores were not significantly correlated with duration of pain.

Hypothesis five was tested using a series of multiple regressions with BVMGT and HVDT scores as criterion variables. Both CPG and APG data were included in this analysis. Table B-2 summarizes the prediction of BVMGT and

HVDT scores. It can be seen from Table B-2 that only the criterion BVMGT and the independent variable of groups membership demonstrated any predictive relationship. Moreover, only 34% of the variance in BVMGT scores was accounted for by group membership.

Another series of multiple regressions were conducted with BVMGT and HVDT scores as criterion variables. This time, APG data was deleted so that duration of pain could be entered as a predictor. (Recall the small amount of variance in pain duration for the APG.) Table B-3 summarizes these data. It can be seen from Table B-3 that with respect to BVMGT performance, none of the variance was significantly accounted for by any of the chosen predictors. This was true despite the fact that group membership was a significant predictor of BVMGT performance. Therefore, it can be concluded that some variable(s) inherent in CPG group membership, other than those measured, accounted for the variance in BVMGT scores. It can also be seen from Table B-3 that while duration of pain was not significantly predictive of BVMGT scores, it was predictive of both right and left HVDT scores. Moreover, it appears that gender may have had an additional influence on left HVDT scores as its predictive power approached significance. To further explicate the role of pain duration and gender on left HVDT performance, a composite score consisting of left configuration and

texture scores was created. This composite score was included in further regression analyses as a criterion variable. Entering the same set of predictors into a step-wise multiple regression with the new left configuration/ texture composite score as the criterion variable, gender was selected first (p = -.02) accounting for 15% of the variance. Duration of Pain was selected next (p = .03) accounting for 11% of the variance. Neither type of Complaint nor Age were selected as significant predictors. In order to identify which gender had done most poorly on the left configuration and texture subtests, group means for males and females were compared using the composite score. Results of this analysis revealed the CPG females had performed significantly poorer than the CPG males, $t(39) = 2.54$, $p = .02$, two-tailed. To double check for significant gender differences on this composite score emerging independent of group membership, combined group data were entered into a t test. No significant difference between males and females was found using both APG and CPG data. This suggests that the relatively lower left HVDT scores among the CPG females was not due to the effects of gender alone.

CHAPTER IV

DISCUSSION

Hypothesis one, stating that the CPG would demonstrate lower BVMGT scores than the APG, was clearly supported. Interestingly, this difference could not be explained on the basis of pain chronicity as no predictive relationship emerged between CPG members' pain duration and their BVMGT performance. Because a good performance on the BVMGT requires considerable attention to detail, especially when scored using the Koppitz (1975) system, the relative poor performance of the CPG might have resulted from poorly focused and sustained attention. If so, these attentional deficits occur early in the course of chronic pain (within the first six months) and precede any lateralized neurologic dysfunction.

Poorly focused attention has been previously associated with high levels of anxiety (Kaplan & Sadock, 1985) and anxiety is commonly documented in chronic pain patients (Lawlis & McCoy, 1983; Swanson, 1984). The relative poor performance of the CPG might have resulted from attentional deficits secondary to anxiety. Again, if this is so, this anxiety related loss of attentional capacity occurs early in the course of chronic pain.

However, as no measure of anxiety was recorded, such an explanation must be considered speculative. The observed difference between the APG and CPG Bender scores could have occurred as a function of chance or as a result of some other, unidentified, group characteristic having to do with chronic pain.

An approach which combines the theoretic constructs of Selye (1976) and Swanson (1984) best explains the possible onset of anxiety occurring in chronic pain patients. Swanson uses the metaphor of an information processing system to discuss the body's response to pain. As he describes it, under normal conditions when some noxious stimulus acts upon peripheral pain receptors, this event is relayed in the form of information to the central nervous system via an intricate set of networks. This information is reviewed by the Central Nervous System which then outputs a set of feelings and behaviors designed to protect the individual from further injury. However, when perception of pain lingers substantially beyond removal of the injurious stimulus--as in the case of chronic pain--this information processing system may become ineffective.

Swanson (1984) hypothesizes that the chronicity of this process results in the patient becoming anxious. He explains this by drawing the following analogy: acute pain is to chronic pain as fear is to anxiety. In this paradigm, fear is the emotional response to the sudden

appearance of an identified external threat, namely, the cause of the pain with anxiety being the emotional outcome of ongoing pain experienced in the absence of a known cause.

When Swanson's (1984) theory is viewed from the perspective of Selye's (1976) model, we can see that fear is the emotional component of the first stage of alarm. Selye focuses on the physiological component of sympathetic discharge fueling the fight or flight response. It is easy to see fright as the emotional elaboration of this physiological response to stress. However, when the stress continues though no external threat is identified--as in the case where no organic cause for pain is found--fear dissolves into anxiety. From Selye's perspective, when stress is unrelenting, the body attempts to adapt to being continually taxed. For awhile, adaptation to a new level of stress may result. Borrowing from Swanson (1984), anxiety may be the emotional elaboration of a strained adaptation which is accomplished in the face of persistent pain of unknown origin. Seen in this way, it is not surprising that pronounced anxiety could develop in early months following injury, especially when the individual is continually frustrated by an unproductive search for the cause of his or her pain. This pronounced anxiety could explain the cognitive deficits observed early in the cause of chronic pain.

Hypotheses two, three, and five addressed the HVDT performance of CPG members as compared to APG members. When compared to the APG, the CPG did demonstrate a significantly poorer performance on the HVDT, primarily on the subtests measuring right hemispheric functioning. This trend was especially evident on the texture and configuration subtests. Because discrimination of texture and configuration involved attention to very fine detail (relative to shape and size) it may be that these subtests required more sustained attention for completion. As a result, a wide range of higher cortical functions are required to complete these subtests. That is, temporal-frontal associations may be involved in addition to the parietal-occipital associations that are localized in the task of visual-motor integration. Therefore, the increased attentional demands results in a corresponding spread of neurologic involvement in much of the cerebral cortex. This may explain the generally high correlations between HVDT scores and measures of intelligence (McCarron & Dial, 1979).

Furthermore, it seems logical that structural damage to the right parietal-occipital region would result in a lowering of all the left HVDT subtests. This was not the case in the present study, as only the left texture and configuration scores were significantly lower in the CPG than the APG. It is unlikely that this resulted from some

localized disturbance indicative of right parietal-occipital structural damage (e.g., a lesion or infarct). It seems more logical to conclude that these findings resulted from some reversible neurochemical disturbance affecting right hemispheric functioning. Abnormal cortisol and endorphin regulation in chronic pain patients (Almay et al., 1978; Atkinson et al., 1983; Atkinson et al., 1986) may somehow be involved in the onset of depression and in consequent altered lateralization (see Swartzburg, 1983 for a summary of the lateralization research). Indirect support for a hypothesized relationship between lowered left HVDT scores and a disturbance in neurochemistry is provided by research documenting a correlation between endorphin levels and sensory processing capacity (Von Knorring et al., 1979) and affective disturbance in chronic pain patients (Johansson, Almay, & Von Knorring, 1979).

An unexpected finding in the present study was the especially poor performance on the left HVDT subtests among CPG females. It is generally accepted that cross-culturally, females evidence a higher incidence of depression (Kaplan & Sadock, 1985); the reasons for this are presently unclear. Nevertheless, if the relatively lower left configuration/texture scores were some kind of depressive equivalent, it is not surprising to find this trend especially prevalent among the CPG females. It may be that female chronic pain patients are somehow more

vulnerable to development of depressive sequelae. This is a highly speculative thought requiring empirical substantiation. Because of a small sample size in the present study, specifically 17 female chronic pain patients, variance in their scores could have resulted from sampling bias. Although there are limitations to the present study, further investigation of the interaction between gender and chronic pain does seem warranted.

Hypothesis three predicted that CPG members would demonstrate disturbed sleep relative to APG members. This hypothesis was supported by the results of the present study. It should be noted that only very limited information was gathered regarding quality of sleep. Specifically, subjects simply reported yes or no to a question of whether or not they slept well. No information was gathered regarding the nature of their sleep patterns. It could not be determined whether CPG members suffered from sleep onset insomnia, early morning awakening, or some mixture of both. Early morning awakening has been closely linked to depression while sleep onset insomnia has been linked to anxiety (Kaplan & Sadock, 1985). Whatever the nature of the CPG's sleep disturbance, it cannot be solely attributed to being in pain. Presumably, many members of the APG were still experiencing pain, yet their sleep was not significantly disturbed.

Many researchers have documented parallel diurnal variations in peripheral endorphin levels and cortisol levels. Apparently these two systems (i.e., the endorphin and neuroendocrine systems) work closely in the regulation of circadian rhythms and in the regulation of sleep, specifically (Dent, Guilleminault, Albert, Polsner, Cox, & Goldstein, 1981). This is not a surprising finding given the growing body of research which points to an intimate anatomical and physiological relationship between these two systems (see Zis, 1988 for a review of this literature). It is also not surprising to find literature correlating these two systems with sleep disturbance in depressed subjects (Dent et al., 1981). It may be that the disturbed sleep of subjects in the CPG resulted from a deregulation in neuroendocrine and endorphin systems. This conclusion must be considered speculative due to the study's limited sample size, the poor psychometric properties of the measure (a single dichotomous question), and an absence of any correlative neuroendocrine measures. Nevertheless, the hypothesized relationship between disturbed sleep, depression, chronic pain, and endorphin/neuroendocrine regulation has precedence and is therefore tenable.

In a 1988 article published in Psychoneuroendocrinology, A. P. Zis discusses the relationship between endorphin and corticotropin regulation. This author bases his discussion on research suggesting an endorphin mediated inhibition of

the human hypothalamic-pituitary-adrenal axis (HPA) (Morley, 1981; Pfeiffer & Herz, 1984). Putting it simply, it is believed that in some way, endorphins are crucial in the homeostatic regulation of corticotropin release. The exact sequence of biochemical events is difficult to tease out and certainly beyond the scope of this study. Nevertheless, findings in the present study are peripherally supportive of the hypothesized regulatory function of endorphin on HPA function. Moreover, the stress-related changes in physiology described by Selye (1976) help to explain the deregulation of these systems. Much of Selye's work focused on the pathophysiology of Cushing's disease. Cushing's disease is characterized by pathophysiologic findings in pituitary tissue (e.g., adenoma) (Buch, 1985) and hyperplastic changes in the corticotropin secreting cells of the pituitary (Zis, 1988). Approximately 30 years after Selye's seminal work, the scientific community is beginning to document the existence of a pathophysiologic continuum containing both depression and Cushing's disease (Gold, Extein, Pickar, Rebar, Ross, & Goodwin, 1980); Gold & Chrousos, 1985; Pepper & Krieger, 1984). It has even been suggested that the pituitary pathology of Cushing's disease results from hyperstimulation of the pituitary arising from higher-order cortical structures (Burch, 1985; Pepper & Krieger, 1984). This provides a link between the psychology of a given

person (i.e., his or her thoughts and feelings) and the metabolic and endocrine derangement of the endorphin/ corticotropin regulatory systems. With this, the major anatomical and physiological building blocks needed to substantiate Selye's work have largely fallen into place. It now becomes reasonable to abandon more simplistic models of chronic pain which portray it as a form of malingering or hypochondriasis in favor of more complex multifactoral models. Moreover, unmanaged stress can be seen as a potentially causative factor and, as such, a proper focus for treatment.

Clinical and Research Implications

Results from the present study provide some substantiation for the use of neuropsychologic assessment techniques in the investigation of chronic pain and depression. It appears that there are measurable neuropsychologic changes that occur over time when a patient is chronically stressed by the experience of pain. Future research investigating neuroendocrine and endorphin correlates of neuropsychological data taken from chronic pain patients would provide interesting insights into the etiology of these neurologic changes, particularly those of a lateralized nature.

From a clinical perspective, the results of the present study substantiate the early use of stress reducing techniques in the treatment of chronic pain. Subjects in

the present study evidenced some loss of attentional capacity before the sixth month of pain duration. With increased pain duration, actual decline in neurologic functioning was measured. These results provide a factual base for arguing that, especially in back-related injury, the acute phases of medical treatment should include a component of stress-management. This is contradictory to the typical medical management of acute injury which often involves palliative measures intended to facilitate symptomatic relief. In a certain number of cases, following a protracted period of symptomatic treatment, the patient is pronounced as having "chronic pain" and referred for psychological treatment. By this point, much decline has already transpired leaving the patient with a compromised set of neuropsychological resources with which to participate in their treatment. Early intervention intended to "inoculate" the patient against the effects of some commonly occurring psychosocial stressors would perhaps prevent a degeneration in neuropsychologic faculties such as those observed in the present study. Moreover, as it appears that females may be particularly vulnerable to a decline in neuropsychologic functioning secondary to unrelenting pain, it would seem advisable to provide supportive intervention as early as possible in their treatment. One method of providing this support, as well as stress-management evaluation, would be to enlist

these patients into group psychotherapy early in the course of their treatment. This would ensure continued contact with qualified psychotherapists as well as cultivation of a much needed system of social support.

Conclusions

The results of the present research suggest that certain neuropsychological changes are correlated with pain chronicity. Moreover, an absence of these neuropsychologic findings in acute pain patients suggests that they occur as a function of pain chronicity. These changes are reflected in sensory processing deficits and most likely parallel a drop in attentional capacity. Additionally, there is some evidence suggesting a relationship between right hemispheric dysfunction and pain lasting well beyond the sixth month following the patient's initial presentation.

An integration of these findings with relevant research points to a relationship between chronic pain and a neuropsychologically mediated disturbance in endorphin regulation. It is suggested that the relentless and pervasive force of stress inherent in the experience of chronic pain may intervene with constitutional factors in developing sensory deficits of the kind observed in the present study. Future research is needed to substantiate these findings. It is recommended that larger sample sizes be included with equal representation of patients having had a few months of pain, and those suffering for a number

of years. Alternatively, a longitudinal study investigating the neuropsychological status of a group of subjects from the time of original presentation through a year or more of pain duration would provide much insight. Finally, it is recommended that future investigations of chronic pain use standardized neuropsychologic assessment methods in service of addressing the multidimensional aspects of chronic pain. Because of the potential links between unrelenting stress, chronic pain and neuropsychologic decline, it is suggested that early stress-reducing clinical intervention would perhaps abort the development of depression and the associated loss in neuropsychological function. Moreover, results of the present study suggest that early clinical intervention may be particularly important with female patients in order to prevent the development of depression and its neuropsychologic sequelae.

APPENDIX A

INFORMED CONSENT AND DEMOGRAPHIC DATA SHEET

Informed Consent

Dear Patient,

Your neurologist is supporting my dissertation research by allowing me to contact certain of her patients with an offer to participate as a subject in my research. I am researching the effect pain has on an individual's ability to think and concentrate on a series of tasks requiring eye-hand coordination. In order to complete these tasks, two one hour testing sessions will need to be scheduled. Should you decide to participate, these sessions will be scheduled at your convenience. You may choose to participate on the basis of your interest alone. There will be no penalty should you choose not to participate. After reading the following statement, should you choose to participate, please sign your name below indicating your understanding of your rights as a participant.

I understand that my participation in this study is entirely voluntary. I also understand that I release my physician of any medical/legal responsibilities for my involvement in the present study. Finally, I understand that my medical records will be reviewed as part of the data base for the present study.

_____

Signature

Acute Pain Patient Demographic Data

Name:_____ Gender: Male___ Female___

DOB:_____

Date of initial diagnosis:_____

Type of Complaint: Lumbar_____ Cervical_____

Physician's name:_____

Patient's Pnone: Home_____ Work_____

Patient's Address_____

_____

Does the patient report (on average) difficulty sleeping?

Yes_____ No_____

Times and dates available for scheduling participation:

Monday:_____ Thursday:_____

Tuesday:_____ Friday:_____

Wednesday:_____ Saturday:_____

APPENDIX B

TABLES

Table B-1

Correlations Between Dependent Variables and Pain Duration

Dependent Variable	*r*	*p*
HVDT Right	-.36	.012
HVDT Left	-.51	.0001
Texture Right	NS	NS
Configuration Right	-.25	.05
Texture Left	-.27	.04
Configuration Left	-.37	.009

Note. NS = Nonsignificant.

Table B-2

Prediction of BVMGT and HVDT Scores, APG and CPG Data Combined

Criterion	Predictors	Significance Level	% Variance Accounted for
BVGMT	Sex	NS	
	Age	NS	
	Type of Complaint	NS	
	Group Membership	.0001	34%
HVDT Right	Sex	NS	
	Age	NS	
	Type of Complaint	NS	
	Group Membership	NS	
HVDT Left	Sex	NS	
	Age	NS	
	Type of Complaint	NS	
	Group Membership	NS	

Note. NS = Nonsignificant.

Table B-3

Prediction of Chronic Pain Group, BVMGT and HVDT Scores

Criterion	Predictors	Significance Level	% Variance Accounted for
BVGMT	Sex	NS	
	Age	NS	
	Type of Complaint	NS	
	Pain Duration	NS	
HVDT Right	Sex	NS	
	Age	NS	
	Type of Complaint	NS	
	Pain Duration	.02	21%
HVDT Left	Sex	.06	not reported
	Age	NS	
	Type of Complaint	NS	
	Pain Duration	.0008	34%

Note. NS = Nonsignificant.

REFERENCES

Akeson, W. H., & Murphy, R. W. (1977). Editorial Comment. Clinical Orthopedics and Related Research, 129, 1-3.

Akiskal, H. S., & McKinney, W. T. (1975). Overview of recent research in depression. Archives of General Psychiatry, 32, 285-305.

Almay, B. G. L., Johansson, F., & Von Knorring (1978). Endorphins in chronic pain: Differences in CFS endorphin levels between organic and psychogenic pain syndromes. Pain, 15, 153-162.

Atkinson, H. H., Kremer, E. F., Risch, S. C., & Bloom, F. E. (1963). Neuroendocrine function and endogenous opioid peptide systems in chronic pain. Psychosomatics, 2, 899-901.

Atkinson, J. H., Kremer, E. F., Risch, C., & Dana, R. (1986). Neuroendocrine responses in psychiatric and pain patients with major depression. Biological Psychiatry, 21, 612-620.

Beals, R. K., & Hickman, N. W. (1972). Industrial injuries of the back and extremeties. Journal of Bone and Joint Surgery, 51, 1593-1611.

Beck, A. T. (1967). Depression: Clinical, experimental and theoretical aspects. New York: University of Pennsylvania Press.

Beecher, H. K. (1956). Measurement of subjective responses: Quantitative effects of drugs. New York: Oxford University Press.

Bender, L. (1938). A visual motor gestalt test and its clinical use. New York: American Orthopsychiatric Association.

Bettencourt, C. M., Calstrorn, P., Hargreaves-Brown, S., Lindan, K., & Long, C. M. (1986). Using work simulation to treat adults with back injuries. American Journal of Occupational Therapy, 40, 12-18.

Blumer, D., & Heilbronn, M. (1982). Chronic pain as a variant of depressive disease: The pain prone disorder. Journal of Nervous and Mental Disease, 170, 381-406.

Bonica, J. J. (Ed.), (1974). Advances in neurology: International symposium on pain, New York: Rven Press.

Bwich, W. M., (1985). Cushing's disease: A review. Archives of Internal Medicine, 145, 1106-1111.

Chapman, R. C., & Bonica, J. J. (1985). Current concepts: Chronic pain. Kalamazoo, Michigan: The Upjohn Company.

Crue, B. L., Kenton, B., & Carregal, E. J. (1978). The continuing crisis in pain research. In G. L. Smith, H. Merskey, & S. C. Gross (Eds.), Pain meaning and management (pp. 3-12). New York: Spectrum Publications, Inc.

Davidson, R. J., & Fox, A. F. (1982). Asymmetrical brain activity discriminates between positive and negative affective stimuli in human infants. Science, 218, 1235-1236.

Davidson, R. J., Schwartz, G. E., & Saron, C. (1978). Frontal vs. parietal EEG asymmetry during positive and negative affect. Presented at the Annual Meeting of the Society for Psychophysiologic Research meeting, Madison, WI.

E'Elia, G., & Perris, C. (1973). Cerebral functional dominance and depression. Acta Psychiatry of Scandanavia, 38, 8-96.

Denny-Brown, D., Meyer, J. S., & Horenstein, S. (1952). The significance of perceptual rivalry resulting from parietal lesion. Brain, 15, 433-471.

Dent, R. M., Guilleminault, C., Albert, L. H., Polsner, D., Cox, H., & Goldstein, R. (1981). Diurnal rhythm of plasma immunoreactive beta-endorphin and its relationship to sleep stages and plasma rhythms of cortisol and prolactin. Journal of Clinical Endocrinology and Metabolism, 52, 942-947.

Flor-Henry, P. (1969a). Psychosis and temporal lobe epilepsy: A controlled investigation. Epilepsia, 10, 363-395.

Flor-Henry, P. (1969b). Schizophrenic-like reactions and affective psychoses associated with temporal lobe epilepsy. American Journal of Psychiatry, 126, 777-779.

Fordyce, W. E., Lensky, D., Calsyn, D. A., Shelton, J. L., Stolov, W. C., & Rock, D. L. (1984). Pain measurement and pain behavior. Pain, 18, 53-69.

Fordyce, W. E., & Steger, J. C. (1979). Chronic pain. In O. F. Pomerlan & J. Q. Brady (Eds.), Behavioral medicine theory and practice (pp. 125-153). Baltimore, MD: Williams and Williams Co.

Freeman, C., Calsyn, D., & Lauks, J. (1976). The use of MMPI with low back pain patients. Journal of Clinical Psychology, 32, 294-298.

Gold, P. W., & Chrousos, G. P. (1985). Clinical studies with corticogropin releasing factor: Implications for the diagnosis and pathophysiology of depression, Cushing's disease and adrenal insufficiency. Psychoneuroendocrinology, 10, 401-419.

Gold, P. W., & Extern, I., Pickar, D., Rebar, R., Ross, R., & Goodwin, F. K. (1980). Suppression of plasma cortisol in depressed patients by acute intravenous methadone infusion. American Journal of Psychiatry, 37, 862-863.

Goldstein, K. (1962). The effect of brain damage on personality. Psychiatry, 15, 245-260.

Gullemin, R., Vargo, T., Rossier, J., Minick, S., Ling, N., River, C., Vale, W., & Bloom, F. (1977). Beta-endorphin and adrenocorticotropic are secreted concomitantly by the pituitary gland. Science, 197, 1367-1369.

Guyton, A. C. (1976). Textbook of medical physiology. Philadelphia: W. B. Saunders Co.

Hecaen, H. (1962). Clinical symptomology in right and left hemispheric lesions. In V. B. Mountcastle (Ed.), Interhemispheric relations in cerebral dominance ((pp. 215-243). Baltimore: The Johns Hopkins Press.

Hammer, C. L. (1978). Depression distortion and life-stress in college students. Cognitive Therapy and Research, 2, 189-192.

Kaplan, H. I., & Sadock, B. J. (1985). Synopsis of psychiatry, behavioral sciences and clinical psychiatry (4th ed.). Baltimore, MD: Williams and Wilkins.

Koppitz, E. M. (1975). The Bender Gestalt test for young children (Vol. 2). New York: Grune & Stratton.

Krantz, S., & Hammen, C. L. (1979). Assessment of cognitive bias in depression. Journal of Abnormal Psychology, 88, 611-619.

Krishnan, K. R., France, R. D., & Houpt, J. L. (1985). Chronic low back pain and depression. Psychosomatic, 26, 299-302.

Lawlis, G. F., & McCoy, C. E. (1983). Psychological evaluation: Patients with chronic pain, symposium on evaluation and care of lumbar spine problems. Orthopedic Clinics of North America, 14.

Lefebrre, M. F. (1981). Cognitive distortion and cognitive errors in depressed psychiatric and low back pain patients. Journal of Consulting and Clinical Psychology, 49, 517-525.

Luria, A. R. (1966). Higher cortical functions in man. New York: Basic Books.

Luria, A. R. (1970). The functional organization of the brain. Scientific American, 222(3), 66-78.

Luria, A. R. (1973). The working brain. New York: Basic Books.

Maruta, T., Swanson, D. W., & Swenson, W. M. (1976a). Low back pain patients in a psychiatric population. Mayo Clinic Proceedings, 51, 57-61.

McCarron, L, & Dial, J. G. (1979). Sensory integration: The haptic visual processes. Dallas, Texas: McCarron-Dial Systems.

McCarron, L., & Dial, J. G. (1986). McCarron-Dial evaluation system manual. Dallas, Texas: McCarron-Dial Systems.

McCreary, C., Turner, J., & Dawson, E. (1974). Differences between fictional vs. organic low back pain patients. Pain, 4, 73-78.

Melzack, R., & Casey, K. L. (1968). Sensory, motivational and central control determinants of pain: A new conceptual model. In D. R. Kenshalo (Ed.), The skin senses (pp. 423-443). Springfield, IL: Charles C. Thomas.

Morley, J. E. (1981). The endocrinology of the opiates and opioid peptides. Metabolism, 30, 195-209.

Mueller, E. E., Genazzanni, A. R. (Eds.) (1984). Central and peripheral endorphins: Basic and clinical aspects. New York: Raven Press.

Newman, P. J., & Sweet, J. J. (1986). The effects of clinincal depression on the Luria-Nebraska neuropsychological battery. International Journal of Clinical Neuropsychology, 18(3), 109-114.

Pepper, G. M., & Kreiger, D. T. (1984). Hypothalamic-pituitary-adrenal abnormalities in depression: Their possible relation to central mechanisms regulating ACTH release. In R. M. Post & J. C. Ballinger (Eds.), Neurobiology of mood disorders (pp. 245-270). Baltimore: Williams & Williams.

Pfeiffer, A., & Hers, A. (1984). Endocrine actions of opioids. Hormonal Metabolism Research, 16, 386-397.

Reitan, R. M., & Davison, L. A. (1984). Clinical neuropsychology: Current status and applications. Washington, DC: Winston & Sons.

Rush, A. J., Weissenburger, J., Vinsom, D. B., & Giles, D. E. (1983). Neuropsychological dysfunctions in unipolar nonpsychotic major depression. Journal of Affective Disorders, 5(4), 281-287.

Schaffer, C. B., Donlon, P. T., & Bittle, R. M. (1980). Chronic pain and depression: A clinical and family history. American Journal of Psychiatry, 137(1), 118-120.

Selye, H. (1976). The stress of life - revised edition. New York: McGraw-Hill.

Skevington, S. M. (1983). Chronic pain and depression: Universal or personal helplessness. Pain, 15, 309-317.

Smith, T. W., Follick, M. J., & Ahern, D. K. (1986). Cognitive distortion and psychological distress in chronic low back pain. Journal of Consulting and Clinical Psychology, 54, 573-583.

Sternbach, R. A. (1968). Pain: A psychophysiological analysis. New York: Academic Press.

Sternbach, R. A. (1974). Pain patients: Traits and treatment. New York: Academic Press.

Sternbach, R. A. (1977). Psychological aspects of chronic pain. Clinical Orthopedics and Related Research, 129, 150-155.

Sternbach, R. A., Wolf, S. R., Murphy, R. W., & Akeson, M. D. (1972). Traits of pain patients: The low back "loser." Psychosomatics, 14, 226-229.

Swanson, D. W. (1984). Chronic pain as a third pathologic emotion. American Journal of Psychiatry, 141, 2-12.

Swartzburg, M. (1983). Hemispheric laterality and EEG correlates of depression. Research Communications in Psychology, Psychiatry, and Behavior, 8(2), 187-205.

Swerdlow, M. (Ed.) (1981). The therapy of pain. Philadelphia: J.B. Lippencott, Co.

Urban, B. J. (1982). Therapeutic aspects in chronic pain: Modulation of noliception, alleviation of suffering, and behavioral analysis. Behavioral Therapy, 13, 430-437.

Wexler, B. E. (1980). Cerebral laterality and psychiatry: A review of the literature. American Journal of Psychiatry, 137, 279-291.

Wittig, R. M., Zorick, F. J., & Blumer, D. (1982). DIsturbed sleep in patients complaining of chronic pain. Journal of Nervous and Mental Disorders, 429-431.

Zis, A. P. (1988). Opioidergic regulation of hypothalamo-pituitary adrenal function in depression and Cushing's disease: An interim report. Psychoneuroendocrinology, 13,(5), 419-430.